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AGAMMAGLOBULINEMIA

Report of Two Cases and Review of Literature*

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The early symptoms and signs of many diseases are referable to the upper respiratory tract. A recently discovered abnormality of the plasma proteins, agammaglobulinemia† or hypogammaglobulinemia, is no exception.

In this syndrome there is a low or absent Gamma globulin, and as a result individuals suffering from this disorder are unusually susceptible to repeated and severe bacterial infections. Infections of the nose, sinuses, ears, throat, lungs, and meninges are quite common in the majority of these patients, and for this reason the otolaryngologist should be thoroughly familiar with agammaglobulinemia; therefore, the purpose of this paper is to review comprehensively the literature on this subject and to present two cases illustrating the characteristic features of this disease.

DEFINITION

Agammaglobulinemia is a congenital or acquired syndrome, the cardinal features being *1.* increased susceptibility to bacterial disease, with a history sometimes dating to childhood, *2.* marked deficiency of circulating plasma Gamma globulin, *3.* absence of circulating antibody in the blood tissues, and

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†The term agammaglobulinemia, which has been established by precedent, will be used in this paper although there is seldom a complete absence of Gamma globulin in the serum of patients with this condition.

4 failure of antibody production in response to antigenic stimulation [1, 2]

The normal range of serum Gamma globulin is about 600 to 1200 mg per 100 cc of serum [1] Although the term agammaglobulinemia implies an absence of Gamma globulin in the plasma, the deficiency is usually not complete The level of Gamma globulin is usually less than 30 mg per 100 cc of serum [1] Clinical experience has indicated that a circulating concentration of Gamma globulin of more than 100 to 150 mg per 100 cc is necessary to provide protection against bacterial infections,[1] therefore, to make the diagnosis of agammaglobulinemia the serum Gamma globulin should not be greater than 150 mg per 100 cc of serum (about 2 per cent of the total protein per 100 cc of serum)

CLASSIFICATION

Individuals having very low levels of circulating Gamma globulin may be divided into four groups *1* congenital agammaglobulinemia, *2* acquired or adult agammaglobulinemia, *3* secondary hypogammaglobulinemia, and *4* transient or physiologic hypogammaglobulinemia Patients in the first three groups have a permanent defect of their antibody-producing tissue Newborn infants comprise the fourth group and have only a temporary lowering of the Gamma globulin level

Congenital Agammaglobulinemia This is a syndrome observed in children and is characterized by recurrent bacterial infections beginning in infancy or very early childhood where there is an inability to synthesize Gamma globulin from birth [1, 3, 4] The incidence strongly suggests that this defect is inherited as a sex-linked recessive characteristic like that of hemophilia [1, 4] Thus, it is transmitted by women, who are otherwise normal, to one-half of their male offspring A few cases of congenital agammaglobulinemia have been reported in females [5, 6] Gitlin and Janeway[1] have not substantiated a diagnosis of congenital agammaglobulinemia in any of the female children which they have studied, however, they point out that this type of inheritance pattern statistically *does* permit the rare possibility of this disease occurring in a female child. In these cases there is often a suggestive familial history. There may be a history of one or more male relatives having died from an overwhelming infection early in childhood. Symptoms usually appear at about six months when the infant has used up the Gamma globulin which he received from his mother. Many of the children handle themselves well until the age of three or four years. Before the days of antibiotics, many of these children apparently succumbed to their first or second major infection.

Acquired or Adult Agammaglobulinemia. This syndrome is so named because of its apparent onset later in life, although it has been observed in older children. It differs in several respects from the congenital form. It may occur in males or females. There is lack of a familial tendency. It is manifested by recurring bacterial infections, which begin at any time from childhood to old age, and which are associated with a severe hypogammaglobulinemia.[1] These patients apparently handle infection well until suddenly they lose the ability to form antibodies. Consequently, they suffer many bacterial infections.

Secondary Hypogammaglobulinemia. Low or measurably absent serum Gamma globulin is frequently observed as a manifestation of many systemic diseases.[2] Apparently, these patients develop hypogammaglobulinemia as a result of some diffuse disease of the reticuloendothelial system which interferes with the production of antibodies by normal plasma cells.[4] This finding is noted frequently in pediatrics in association with nephrosis.[2] Leukemia, lymphoma and myeloma may cause hypogammaglobulinemia.[4-7] Porges[8] has described a method for demonstrating secondary agammaglobulinemia in the presence of abnormal myeloma protein. His results suggest that an absence of normal Gamma globulin can occur in multiple myeloma even when electrophoresis and other tests indicate abnormal amounts of Gamma globulin. He reported absence of normal Gamma globulin in two patients with myeloma. The first patient had episodes of pneumonia, otitis media and sinusitis. The second patient had recurrent attacks of pneumonia. This finding would account for the increased susceptibility to infection which is seen in some patients with multiple myeloma Other causes of secondary hypogammaglobulinemia include total body irradiation and treatment with mechlorethamine hydrochlorid (nitrogen mustard)

Transient or Physiologic Hypogammaglobulinemia This constitutes a state of temporary deficiency in circulating plasma Gamma globulin resulting from an accentuation of the normal inadequacy of Gamma globulin synthesis in the first few months after birth [1] Serum Gamma globulin levels are normal in the newborn infant, but drop by about one-third in the first month of life [10] They remain at these levels for about three months and then gradually begin to rise Normal adult values are reached at the age of two years

The newborn infant acquires the major portion of his Gamma globulin from the maternal circulation The fact that he does not synthesize Gamma globulin in adequate amounts until one to three months after birth accounts for the physiologic and transient fall in Gamma globulin the early months of life The normal infant is born with 1,000 mg per 100 cc of serum Gamma globulin, and most infants produce Gamma globulin to keep their total serum concentration from falling below 300 mg per 100 cc [9] Gitlin and Janeway[1] have observed two infants with serum levels of 75 to 150 mg per cent of Gamma globulin who had severe infections Thus, this period of transient hypogammaglobulinemia in an infant's life may be a hazardous one, particularly in the premature infant Because of this physiological lowering of the Gamma globulin in infants, one must be extremely cautious in establishing a diagnosis of congenital agammaglobulinemia in an infant below six or eight months of age, especially if the infant is a female

ETIOLOGY

The symptoms of agammaglobulinemia are directly related to the deficiency or absence of circulating Gamma globulin [1] In congenital agammaglobulinemia Gamma globulin is also absent in the tissues, thus, the circulating deficit is part of a total body deficit [1] The biologic defect in this disease is a failure of synthesis of Gamma globulin [1] The fact that administered Gamma globulin circulates in patients with agammaglobulinemia for as long as it does in normal persons indicates that the basic defect is failure to synthesize Gamma globulin rather than abnormally rapid destruction or loss[9]

A profound disturbance of the hematopoietic system accompanies agammaglobulinemia Recent evidence points toward plasma cells or lymphocytes as the principal sites of antibody and Gamma globulin formation[3,11,12] Recent studies based on the assumption that antibodies are produced by immature plasma cells and lymphoblasts have shown that the lymph nodes of patients with idiopathic low and absent Gamma globulin do not respond with plasmocytosis to antigenic stimulation[13,14,15] In a few cases changes have been observed in bone marrow, namely, the marked reduction of plasma cells[16] That this finding may be a clue to the etiology is the fact that occasionally there is noted an absence of Gamma globulin in multiple myeloma,[17] a disease characterized by derangement of plasma cells Possibly more than one cell is concerned, as opinions vary as to the development and morphology of cells To support this theory, Good and Campbell,[18] using quantitative bone marrow counts of plasma cells, have been able to correlate plasmacytosis with elevation of serum Gamma globulin in patients with rheumatic fever

The plasma cell (Plasmacyte) was discovered by Cajal in 1890[19] A preponderance of evidence favors the reticuloendothelial cell as the probable forerunner of the plasma cell[12,20-24] In regard to function, Bing and Plum[25] in 1937, were the first workers to call attention to the relationship of plasmocytosis and hyperglobulinemia Now it is fairly well established that antibodies are produced, for the most part, by the precursor of the plasma cell[25] Paris, et al ,[25] reported a patient with agranulocytosis of the aplastic type, with an unusually extensive reactive plasmocytosis of bone marrow, *i e* , 60 2 per cent, and a hyperglobulinemia of 3 08 gm per 100 ml In this patient there was hyperplasia of the reticulum cells within bone marrow, lymph nodes, and spleen, in addition, in the bone marrow, transitional forms were observed which were morphologically intermediate between reticulum cells and plasma cells These findings support the concept of the reticuloendothelial origin of the plasma cell Good's findings support the concept that plasma cells are directly involved in the sequence of events resulting in the formation of antibodies and Gamma globulin [24] Evidence in the literature demonstrates conclusively that plasma cells form antibodies in experimental animals [26]

Most of the pathological changes reported in agammaglobulinemia have been those secondary to repeated or long-standing infections [27] Janeway, et al,[28] reported that an autopsy on a patient with agammaglobulinemia revealed only a strikingly diminished Gamma globulin content of the lymphoid tissue, and that a connective tissue biopsy on a patient with this disorder showed a low Gamma globulin content Function tests, biopsies, and postmortem studies show that hepatic dysfunction is not responsible for failure of Gamma globulin synthesis [29] The clotting mechanism is normal in patients with this disorder [29] Lymph nodes of patients with agammaglobulinemia tend to be small, poorly developed, and have a relatively thin cortex with few germinal centers [26] As has been mentioned previously, lymph nodes and bone marrow of these patients are characterized by failure to develop plasma cells and secondary follicles in response to both primary and secondary antigenic stimulation [24]

The patients originally described by Bruton and associates were boys, and it was assumed for a time that the disease occurred only in males and was genetically determined, being transmitted as a sex-linked recessive trait While it is true that most of the patients are boys who have a congenital absence of Gamma globulin, this syndrome has been reported in two females [5,6] An acquired form of this disease is now recognized which affects both males and females of all ages [1,17,30,31] Two possible mechanisms in the pathogenesis of agammaglobulinemia have been suggested, one being a sex-linked hereditary process as seen in young males, and the other in adults of both sexes being acquired from some sort of assault on the Gamma globulin producing function [17] Seltzer, et al,[1] reported a case of agammaglobulinemia in an adult male who had had histoplasmosis They raised the question as to whether histoplasmosis and the resulting infection of the antibody-forming reticuloendothelial system may have resulted in depression of Gamma globulin, however, there is reason to believe that both congenital and acquired agammaglobulinemia are hereditary, for as Brem and Morton[27] point out, it is difficult to envision a noxious agent which can pick out one specific function and eliminate it entirely without damaging related functions They state that the elimination of a single protein fraction is much more characteristic of a hereditary process in which the gene for that particular function is defective, afibrinogenemia and hemophilia being notable examples of this process In support of the hypothesis that the acquired type may also be congenital is the fact that many inherited diseases make their appearances at widely varying ages There is a distinct possibility of an inherited basic biologic defect manifesting itself in delayed failure of Gamma globulin production[27] That failure in Gamma globulin synthesis is responsible for the deficiency in adult agammaglobulinemia has been indicated by the demonstration of a normal rate of degradation of administered radioiodinated Gamma globulin[37,38]

Brem and Morton[27] remind us that the Gamma globulin fraction is composed of a heterogenous group of globulins with different physical and chemical properties, and that one of the perplexing aspects is that the whole Gamma globulin fraction can be virtually eliminated at once They deduce that since there is a simultaneous elimination of the whole fraction, all of the Gamma globulin is elaborated by a highly specialized tissue, and that the function of this tissue can be deranged in varying degrees

Of particular interest is the fact that cases of acquired agammaglobulinemia have been reported in patients with multiple congenital deformities,[21,27,29] moreover, this disorder has been described in siblings[27] Various authors have reported hypergammaglobulinemia in parents of patients having acquired agammaglobulinemia,[37,40,41] thus, the association of this syndrome with congenital deformities, its occurrence in siblings, and the existence of hypergammaglobulinemia in parents of some cases, strongly suggest a hereditary basis in the etiology of acquired agammaglobulinemia

Varco, et al.,[12] describe the homotransplantation of skin in two children suffering from congenital agammaglobulinemia. This is in sharp contrast to the observation that normal persons always reject grafts from unrelated donors. In these patients immunological studies indicated a complete immunologic paralysis. In an adult with acquired agammaglobulinemia they reported the delayed rejection of a skin homotransplant. They describe this patient as having an incomplete immunological paralysis. These experiments demonstrate dramatically the inability of patients with this condition to produce antibodies, and support the theory that rejection of homografts has an immunologic basis.

CLINICAL MANIFESTATIONS

The symptoms of agammaglobulinemia are primarily those of infection. Since the manifestations of infection are quite varied, each patient presents an individual problem. These patients are unusually susceptible to bacterial infections, and many of them have repeated severe crippling diseases. Sinusitis, otitis media, pneumonia, meningitis and bacteremia follow each other with extreme rapidity. The individual pattern *of infection and the severity of any one episode are highly* variable. Gitlin and Janeway[1] point out, that while meningitis has occurred in a single child on two or even three separate occasions, in the same child attacks of pansinusitis and otitis media were far more frequent.

Some patients develop chronic bacterial infections, such as chronic otitis media, chronic pansinusitis, and bronchiectasis. There is no specific characteristic of any of the infections that would implicate the diagnosis of agammaglobulinemia, thus, the presenting symptoms of patients suffering from this disorder are quite protean, some having bronchiectasis and pneumonia and others arthritis, pyoderma with septicemia, and even severe gastro-intestinal conditions. Gitlin and Janeway[1] surveyed a great many children who were subject only to repeated attacks of tonsillitis and upper respiratory infections, and did not find any with congenital agammaglobulinemia.

The role of antibiotics in the defense against bacterial infections is well demonstrated in patients with agammaglobulinemia. Although these patients have an unbelievable number and variety of illnesses, the infections may be no more severe than those observed in normal individuals. The bacterial infections in these individuals respond to treatment with chemotherapeutic and antibiotic agents.[4] This fact may explain why this condition was not recognized clinically until after the antibiotic era, for prior to that time there is a good possibility that patients with absent Gamma globulin did not survive long enough to establish a history of recurrent infections.

One of the unusual features of this condition is the apparent ability to deal with virus infections.[47] The observation has been made that patients with this anomally have had measles, mumps, chickenpox and poliomyelitis with no greater frequency or severity than normal individuals.[4] This is true of both children and adults.[43] Thus one would conclude that immunity to virus infections is not wholly dependent on Gamma globulin, or that smaller amounts of the substance are needed to protect against viruses than against bacteria. One case has been reported to have had three attacks of parotitis, but the diagnosis of mumps as against recurrent parotitis could not be confirmed.[44] One exception to the general rule is apparent susceptibility to vaccinia virus. Carson and Connell[45] reported a fatal case of vaccinia gangrenosa in a nine-month-old white infant with hypogammaglobulinemia. Four similar cases have been reported in the literature.[5,46-48] A few children with low Gamma globulin, without pre-existing skin involvement, have apparent inability to produce vaccinia virus neutralizing antibodies either in sufficient quantity or quality.[45] In such children generalized vaccinia with severe necrotizing lesions may develop, on the other hand, many patients with agammaglobulinemia have been vaccinated without complications.[28,44,47]

Patients with agammaglobulinemia have a severe immunologic handicap.[4] Immunologic reactivity is paralyzed or extremely slight. Dick and Schick tests are uniformly positive. As antibody responses cannot be provoked by antigenic stimulation, immunization against disease by usual means is impossible.[2,1] Gitlin and Janeway[1] have been unable to demonstrate reagins in many of these children tested with the pollens of numerous grasses and trees, and with antigens prepared from allergenic foods.

Moderate to severe degrees of neutropenia have been reported in children with agammaglobulinemia.[1] This is believed to represent a secondary factor in this disease resulting most likely from toxic effects of the infections. It is cyclic, and recovery from leukopenia does not always parallel recovery of the patient from an infection. In most cases, however, there is a prompt rise in granulocytes with infections even in those children showing neutropenia.[1]

Recently attention has been drawn to the association of agammaglobulinemia with enlargement of the liver, spleen and lymph nodes. Citron[10] described two cases and reviewed six similar ones in the literature with hepatosplenomegaly and lymph node enlargement. The chief histological features were *diffuse reticuloendothelial hyperplasia and deficiency* of plasma cells. Pearce and Perinpanoyagam[7] reported hepatosplenomegaly and palpable cervical lymph nodes in a case of congenital agammaglobulinemia. Three cases of acquired agammaglobulinemia with hepatosplenomegaly have been described by Martin, et al.[10] Savacool and Landes[9] reported splenomegaly and lymph node enlargement in a 38-year-old male with acquired agammaglobulinemia. Zelman and Lewin[3] described reticuloendothelial hyperplasia of spleen and lymph nodes with granuloma formation in a patient with acquired Gamma globulin deficiency.

Of particular significance are the instances of arthritis and dermatomyositis which occur in patients with congenital and acquired agammaglobulinemia.[1,2] Cases of suppurative arthritis have been observed, but these probably are of the same general nature as the other infections which plague these patients. Of special interest is the nonsuppurative form of arthritis which has occurred in roughly a fourth of recorded cases.[1] Individual joints present clinical features quite consistent with rheumatoid arthritis, including effusions, slight local warmth, pain and tenderness, and slight to moderate limitation of motion. Synovitis consistent with rheuma toid arthritis has been described in biopsy specimens of synovial membrane.[1]

The occurrence of dermatomyositis in patients with agammaglobulinemia lends support to the view that this is truly rheumatoid arthritis,[42, 43] however, Vaughan and Good[44] tested patients with agammaglobulinemia by several procedures for demonstration of the rheumatoid factor or its inhibitor, and found neither agglutinator nor inhibitor in the sera of the patients studied. They could neither confirm nor exclude a possible relationship between the arthritis of agammaglobulinemia and rheumatoid arthritis. One suggestion as to the possible mechanism of causation is that the arthritis of agammaglobulinemia is an inflammatory response to non-living products of repeated chronic infections.[41] Fortunately, most attacks of arthritis accompanying agammaglobulinemia have subsided spontaneously over periods of several days to months without residual disability.

Acquired agammaglobulinemia differs from the congenital type in that the outstanding clinical features are pulmonary and gastrointestinal manifestations.[1] According to Mazzitello and Good,[45] pulmonary infection represents a *sine qua non* of the agammaglobulinemia syndrome. Bronchiectasis is a particularly threatening disease. Though it has been seen in patients with the congenital type it is more common in acquired agammaglobulinemia.[46] From this it follows that agammaglobulinemia should be ruled out in all cases of bronchiectasis.

DIAGNOSIS

A variety of laboratory studies have been suggested to assist in establishing the diagnosis of agammaglobulinemia. Barrett and Volwiler[9] have separated the tests into two groups: presumptive and definitive.

Presumptive Tests

Many routine laboratory tests suggest the diagnosis of agammaglobulinemia. The tests below aid in the diagnosis but do not definitely prove or exclude the presence of the condition.

Total Serum Globulins Since about one-half of the reported concentration is Gamma globulin, agammaglobulinemia may cause a low level of total globulin Of course, if the patient has a chronic disease causing an increase of the other globulin fractions, the level of total globulins may be within normal limits

Isohemagglutinin Titers Patients with agammaglobulinemia have shown a deficiency in isohemagglutinins, and demonstration of their absence during blood grouping has been suggested as a screening test for the disease in patients with blood groups O, A and B The test is of no value for patients with blood group AB A normal person who is not of type AB and who is over two years of age usually has a titer of anti-A or anti-B of 1 8 or higher A high titer would rule out the disease, and a low titer would strongly suggest it

Response to Antigenic Stimulation Patients with agammaglobulinemia have persistently positive Schick and Dick reactions This is true since lacking Gamma globulin they do not produce antibodies Antibody formation may be determined by injecting antigens such as typhoid and diphtheria vaccines A good antibody response, as measured by the Gruber Widal reaction and the Schick test, tends to rule out agammaglobulinemia

Zinc Sulfate Turbidity Test Good[23] recommends zinc turbidity as a simple screening test It gives a semiquantitative estimate of the concentration of Gamma globulin in the serum Values of zero to one unit are compatible with the diagnosis of agammaglobulinemia Higher values rule it out

X-ray Findings Margulis, et al , [illegible] report the important diagnostic Roentgen features of agammaglobulinemia as being extensive pulmonary parenchymal disease with paradoxical absence of hilar node enlargement and the striking deficiency of nasopharyngeal lymphoid tissue, in spite of recurrent upper respiratory infections Many of the patients showed pansinusitis Chest and sinus X-rays with a soft tissue lateral view of the nasopharynx should be routine on any patient suspected of having this disease

Definitive Tests

There are two definitive tests for agammaglobulinemia—electrophoresis of the serum and immunochemical quantitation.

Electrophoresis of Serum. This is a procedure for direct measurement of the Gamma globulin content of the serum plasma. Paper electrophoresis, as described by Durrum, is one of the most reliable screening tests for agammaglobulinemia. The technique is simple, and only a small quantity of blood is needed. The Gamma globulin is usually reported as a percentage of the total protein per 100 cc. of serum. Normal values range from 12 to 16 per cent. Free electrophoresis as described by Tiselius is also satisfactory, but more equipment and a larger sample of blood are required. By either of these techniques the only significant abnormality of the serum proteins in patients with agammaglobulinemia is an absence or marked decrease of the Gamma globulin.

Immunochemical Quantitation. Immunochemical methods offer the only satisfactory means of sensitively measuring low concentrations of Gamma globulin.[9] The precipitin method seems to be the best of these techniques. There is actually some Gamma globulin in the serum of these patients, but not enough to be demonstrated by electrophoresis.[4] Using the immunochemical techniques, analysis of the plasma of patients with congenital and acquired forms of the disease shows the presence of small amounts of Gamma globulin. Patients with acquired agammaglobulinemia have slightly more Gamma globulin than patients with the congenital type. In both types the concentration seldom exceeds 100 mg. per 100 cc. of serum (normal 600 to 1,200 mg. per 100 cc.).[4]

TREATMENT

In the therapy of agammaglobulinemia both antibiotics and concentrated Gamma globulin have proven of value.[1] The prime considerations in the treatment of a patient who is unable to produce antibodies are *1* the rapid control of acute infections, and *2* the prevention of recurrent infections.[9] In the treatment of acute bacterial infections the same principles as are efficacious in treating such infections in immunologi-

Total Serum Globulins. Since about one-half of the reported concentration is Gamma globulin, agammaglobulinemia may cause a low level of total globulin. Of course, if the patient has a chronic disease causing an increase of the other globulin fractions, the level of total globulins may be within normal limits.

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Response to Antigenic Stimulation. Patients with agammaglobulinemia have persistently positive Schick and Dick reactions. This is true since lacking Gamma globulin they do not produce antibodies. Antibody formation may be determined by injecting antigens such as typhoid and diphtheria vaccines. A good antibody response, as measured by the Gruber Widal reaction and the Schick test, tends to rule out agammaglobulinemia.

Zinc Sulfate Turbidity Test. Good[29] recommends zinc turbidity as a simple screening test. It gives a semiquantitative estimate of the concentration of Gamma globulin in the serum. Values of zero to one unit are compatible with the diagnosis of agammaglobulinemia. Higher values rule it out.

X-ray Findings. Margulis, et al.,[30] report the important diagnostic Roentgen features of agammaglobulinemia as being extensive pulmonary parenchymal disease with paradoxical absence of hilar node enlargement and the striking deficiency of nasopharyngeal lymphoid tissue, in spite of recurrent upper respiratory infections. Many of the patients showed pansinusitis. Chest and sinus X-rays with a soft tissue lateral view of the nasopharynx should be routine on any patient suspected of having this disease.

tions *1* the half-life of injected Gamma globulin in humans ranges between 13 and 32 days,[40] and *2* a level of 100 to 150 mg of Gamma globulin per 100 cc of serum seems to be the minimum necessary to prevent infection, a level easily obtained by the injection of 0 1 gm per kilogram of body weight [1,41]

In 1956, the Council on Pharmacy and Chemistry of the American Medical Association approved the use of immune globulin for the treatment of agammaglobulinemia [61] The Council pointed out that the immunity thus provided is temporary and passive, and emphasized that continued protection depends on routine and regular administration The Council recommended the deep intramuscular injection of this preparation, preferably in the gluteus

Although Gamma globulin is apparently the best means of prevention of recurrences of infections in these patients it has certain disadvantages *1* it necessitates the injection of a large volume of Gamma globulin solution at relative frequent intervals which is painful and unpleasant *2* continuous replacement therapy presents an economic problem, as Gamma globulin is quite expensive, *3* it provides only passive immunity, *4* and, finally, the long term effects of repeated injections of Gamma globulin have not been determined [43] Because of severe hypotensive reactions, the material cannot be administered intravenously [41] Chills fever, myalgia and faintness result from intramuscular injections of over 20 ml [41] That there is little danger of serum hepatitis, even with prolonged administration, should be emphasized, since the virus of homologous serum jaundice is not associated with the Gamma globulin fraction [41]

Just how long replacement therapy with Gamma globulin in these patients must be continued is not yet known So far there has been no report of a patient with agammaglobulinemia resuming the manufacture of Gamma globulin [60] In some of the patients with secondary agammaglobulinemia (nephrotic, nutritional dysproteinemic) resumption of Gamma globulin production can be expected to accompany recovery from the primary disorder [60] cally normal persons are regularly successful.[13] There should be accurate bacteriologic diagnosis and intensive specific treatment. In addition, time-honored procedures of surgical drainage in acute and chronic infections should be employed when indicated. Pooled Gamma globulin apparently does not alter the course of an acute disease in these patients.[9] This would be expected, since it does not provide enough specific antibody to a single bacterial antigen. Thus, Gitlin and Janeway[1] state that in their patients, the use of antibiotics has proven more effective than the use of Gamma globulin alone in the treatment of acute infections.

On the other hand, Gamma globulin has proved effective in the prophylaxis of infections in patients with agammaglobulinemia.[43] *Seltzer,*[13] *Rosenstiel,*[56] *Kuhns*[39] *and Bruton*[57] have reported improvement in their cases using Gamma globulin alone. Savacool and Landes[50] found Gamma globulin to be helpful in the treatment of a case of acquired agammaglobulinemia. They suggested that Gamma globulin be given only during times when exposure to infections is most likely and rely upon antibiotic prophylaxis at other times. Freundlich[58] reported improvement in a child with agammaglobulinemia using Gamma globulin; however, there was no improvement in an otitis media, and a mastoidectomy had to be performed. Latimer[59] reported cellulitis in one of his patients with agammaglobulinemia which did not respond to therapy until Gamma globulin was added to the therapeutic regimen.

A dose of 0.1 gm. of Gamma globulin per kilogram of body weight intramuscularly every 30 days has given adequate protection to most patients.[1, 60] Since Gamma globulin ordinarily is manufactured as a 16 per cent solution, this would represent 0.3 cc. per pound of body weight. In some cases, clinical improvement was not noted below a dosage of 0.15 to 2.0 gm. of Gamma globulin per kilogram of body weight.[1] That the prophylactic dose of Gamma globulin varies in different patients may be due to the fact that Gamma globulin is a heterogenous substance containing many different antibodies which have varying rates of degradation (ranging from 17 to 58 days).[49]

The dosage of Gamma globulin is based on two observathe treatment of children with asthma in preventing upper respiratory infections On the other hand, Abernathy, et al,[66] in a double-blind controlled study, found no evidence to support the view that treatment with Gamma globulin has a beneficial effect on infection and the course of chronic asthma in children

Recently, Good, et al,[67] reported that transplantation of four lymph nodes from a previously sensitized patient provided immunological reactivity to a patient with acquired agammaglobulinemia that lasted about 75 days Martin[68] has found by lymph node transplant immunologic capacity lasting up to 160 days Good, et al, suggested that in light of studies on skin transplantation, a more rewarding response may occur in children with congenital agammaglobulinemia They recommend the continued consideration of transplantation as a means of correcting the metabolic defect in these patients, and, in addition, suggest as possible approaches to this end, bone-marrow infusion, cancellous bone transplantation and production of splenosis peritonei

PROGNOSIS

The syndrome of agammaglobulinemia is a serious and potentially fatal defect which, if diagnosed and treated, appears to be compatible with normal life[69] Four of the original 19 patients reported by Gitlin and Janeway have succumbed to infection[1] The remaining patients have done fairly well, although those with anatomic changes secondary to infection suffer from permanent handicaps One of three patients described by Hayles, et al,[70] died of infection, although Gamma globulin was administered regularly Other cases of death from infection have been reported[27, 40, 46, 71] There is a great tendency to permanent damage in these patients, and there is a constant danger from infectious disease, thus, diagnosis should be established early and substitution and antibiotic therapy started before irreversible changes occur Certainly the prognosis in this newly discovered syndrome awaits further years of study

Antibiotics have been used successfully in the prophylaxis of infection in these patients. In some instances it may be necessary to supplement Gamma globulin prophylaxis with a program of continuous antibiotic therapy. Zinneman, et al.,[36] reported a case which did not respond to Gamma globulin, but responded to prolonged antibiotic prophylaxis. These workers concluded that prophylactic antibiotic therapy offers advantages over prophylactic Gamma globulin. Mazzitello and Good[13] reported effective prevention of recurrent infections in one case using antibiotics and Gamma globulin, and in several of their patients have used penicillin and tetracyclines alternately. Because of the cost involved, a case reported by Zbar, et al.,[62] received no supplemental therapy with Gamma globulin, but was treated with tetracycline alone, and remained free of significant infection for 20 months. Martin, et al.,[63] described an adult case of agammaglobulinemia who required sulfadiazine plus double the usual frequency of Gamma globulin injections for complete protection. Brem and Morton,[27] from the study of seven cases, concluded that the effects of this disease may be ameliorated by the administration of adequate amounts of Gamma globulin and the appropriate use of antibiotics.

Gamma globulin is only as effective as the amount and properties of the specific antibodies in the individual Gamma globulin preparation used.[1] These patients, in spite of Gamma globulin administration, will have the usual respiratory infections suffered by normal individuals; moreover, Gamma globulin and antibiotic prophylaxis will not reverse permanent anatomic changes, such as chronic ear, sinus and lung diseases. Again, this fact emphasizes the importance of proper surgical management of these conditions in patients with agammaglobulinemia.

As has been pointed out, there are definite disadvantages to the use of Gamma globulin; therefore, its indiscriminate use in unproved cases of agammaglobulinemia and in children with recurring respiratory infections should be frowned upon. Turner and Dobson[64] reported good results in the treatment of chronically ill children with repeated respiratory infections using immune globulin. Bernton[65] had gratifying results in the treatment of children with asthma in preventing upper respiratory infections On the other hand, Abernathy, et al ,[66] in a double-blind controlled study, found no evidence to support the view that treatment with Gamma globulin has a beneficial effect on infection and the course of chronic asthma in children

Recently, Good, et al ,[67] reported that transplantation of four lymph nodes from a previously sensitized patient provided immunological reactivity to a patient with acquired agammaglobulinemia that lasted about 75 days Martin[68] has found by lymph node transplant immunologic capacity lasting up to 160 days Good, et al , suggested that in light of studies on skin transplantation, a more rewarding response may occur in children with congenital agammaglobulinemia They recommend the continued consideration of transplantation as a means of correcting the metabolic defect in these patients, and, in addition, suggest as possible approaches to this end, bone-marrow infusion, cancellous bone transplantation and production of splenosis peritonei

PROGNOSIS

The syndrome of agammaglobulinemia is a serious and potentially fatal defect which, if diagnosed and treated, appears to be compatible with normal life[69] Four of the original 19 patients reported by Gitlin and Janeway have succumbed to infection[1] The remaining patients have done fairly well, although those with anatomic changes secondary to infection suffer from permanent handicaps One of three patients described by Hayles, et al ,[70] died of infection, although Gamma globulin was administered regularly Other cases of death from infection have been reported[27, 40, 46, 71] There is a great tendency to permanent damage in these patients, and there is a constant danger from infectious disease, thus, diagnosis should be established early and substitution and antibiotic therapy started before irreversible changes occur Certainly the prognosis in this newly discovered syndrome awaits further years of study

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HISTORY AND REVIEW OF CASES

An effort has been made to review a great many of the reported cases of agammaglobulinemia. In the cases described, emphasis has been placed on the symptoms and signs referable to the nose, sinuses, ears, throat and lungs—the areas which are of great interest to one practicing the specialty of otolaryngology.

In 1952, Bruton[44] reported the first case of agammaglobulinemia in a male child as a syndrome of recurrent bacterial infection associated with a lack of Gamma globulin, despite an otherwise relatively normal plasma protein pattern. Prior to this time, several patients with decreased concentration of serum globulin, and in some instances with increased susceptibility to infections had been reported.[illegible] Bruton's[2] patient was an eight-year-old boy who, in four years, had had at least 19 episodes of clinical sepsis, in which some type of pneumococcus was recovered from his blood stream on ten occasions. He was seen initially with a clinical picture of acute osteomyelitis. He had frequent episodes of otitis media and pneumonia. Studies demonstrated that he did not produce antibodies for pneumococcus, diphtheria or typhoid when given specific antigens. Clinical and laboratory studies revealed no deviation from normal until a Tiselius electrophoretic analysis revealed the absence of serum Gamma globulin.

In 1953, Kerdan, et al.,[5] reported agammaglobulinemia in a fatal case of generalized vaccinia in a seven-year-old girl. In this same year, Janeway, et al.,[28] were able to confirm Bruton's findings in nine cases. These children had multiple infections, and were unable to produce circulating antibodies. The determination of the absence of serum isohemoagglutinins in patients of blood groups O, A, or B was used as a simple screening test for this condition.

Lowered serum globulins have been observed in patients with edema due to a lowered serum albumin.[illegible] Hypogammaglobulin has been reported in patients with nephrosis[illegible] or malnutrition[illegible] with edema.

Allen[illegible] treated a middle-aged woman with agammaglobulinemia with several hundred plasma transfusions During this period this patient underwent several successful pregnancies Zinneman, et al ,[11] reported agammaglobulinemia in two men and one woman who had normal childhood, but who had recurrent respiratory infections, pneumonia, bronchiectasis, and pneumococcic meningitis during early adulthood In one of these patients the cause of the disease was thought to be secondary to generalized sarcoidosis The patients were unable to produce humoral bacterial antibodies Biopsies of the liver, spleen and lymph nodes of one patient showed complete absence of plasma cells, which are thought to be the source of Gamma globulin

Young and Wolfson[35] reported four men with agammaglobulinemia, three of whom had no lymph node germ centers All four were extremely susceptible to infection Arends, et al ,[29] described a 53-year-old woman with agammaglobulinemia who had malignant lymphoma and repeated respiratory and skin infections Sanford, et al ,[30] described a female adult with no Gamma globulin who had repeated infections of a sprue-like syndrome Rosecan, et al ,[3] reported two adult male cases of agammaglobulinemia one experienced repeated upper respiratory infections and diarrhea, and the other only repeated intestinal difficulties

Prasad and Koza[17] reported the first case of agammaglobulinemia in a female adult She had a history of recurrent illnesses beginning with sinusitis She had attacks of pneumonia and meningitis Clinically, she had lymphadenopathy, hepatomegaly and splenomegaly Numerous nonspecific granulomata were demonstrated on histologic sections of the spleen and the biopsied axillary lymph node These workers thought that this finding implicated a disease of the reticuloendothelial system Prasad and Koza suggested that some infection may have resulted in granuloma formation thereby damaging the reticuloendothelial system and producing agammaglobulinemia

Wall, et al ,[34] reported two cases of agammaglobulinemia in adults One patient had repeated bacterial pneumonias and one had repeated infections associated with a sprue-like picture Of interest is the fact that the first case had a repaired cleft palate and congenital hip dislocation, as several cases of agammaglobulinemia have been reported in individuals having congenital anomalies[32] In addition, he had had two episodes of erysipelas The second case had recurrent episodes of nasopharyngitis, maxillary sinusitis, bronchitis and diarrhea A lymph node biopsy incision failed to heal for two and one-half months despite vigorous antibiotic therapy

Seltzer, et al ,[13] reported a case of acquired idiopathic agammaglobulinemia in a 27-year-old male with recurrent upper respiratory infections and pneumonia They were reasonably convinced that the patient at one time had histoplasmosis and raised the question as to whether histoplasmosis and resulting infection of the antibody-forming reticuloendothelial system could have resulted in permanent depression of antibody formation, or possibly the depressed antibody production made him susceptible to histoplasmosis From their studies they concluded that the Gamma globulin half-life in idiopathic hypogammaglobulinemia and agammaglobulinemia is normal regardless of the age group

Young, et al ,[41] described four cases of agammaglobulinemia in male adults In three cases there was a deficiency in tissue and peripheral blood lymphocytes An interesting finding was a marked hypergammaglobulinemia in a mother and brother of one case The first case had a stormy course with frequent infections of purulent otitis media, beginning in childhood, acute mastoiditis, suppurative cervical adenitis, infectious hepatitis, lobar pneumonia and bronchiectasis At 17 he had an operation for chronic sinusitis and at 18 a lobectomy for bronchiectasis The other three cases had frequent infections since childhood with bronchopulmonary involvement predominating

Latimer, et al , [9] reported a severe wound infection following the surgical excision of a benign breast lesion in an adult female with a proved agammaglobulinemia The cellulitis responded to therapy only after Gamma globulin administration

Brem and Morton[27] described their experiences with seven patients in whom Gamma globulin was not demonstrable electrophoretically The first three were adults who exhibited the complete pattern of agammaglobulinemia, with absence of antibody production and susceptibility to infection All three had striking splenic and lymph node enlargement, apparently secondary to agammaglobulinemia or to the resulting infections Two of them developed advanced liver disease, presumably due to chronic viral hepatitis, possibly of the homologous serum variety The first patient suffered from sinusitis, otitis media and bronchitis The second patient had sinusitis, otitis media, mastoiditis, bronchitis and pneumonia During the course of his infections he had a right and then a left mastoidectomy His sinuses were surgically drained The third case had many respiratory infections and bronchiectasis following whooping cough He had had a bilateral pulmonary lobectomy The next two cases, a 25-year-old male and a nine-year-old boy, showed marked depression of Gamma globulin electrophoretically, but were able to produce antibodies and were not particularly subject to infection, both had isohemagglutinins The adult male complained of recurring episodes of abdominal pain with nausea and vomiting The boy had a peculiar undiagnosed episode of edema lasting about one month The next two cases were elderly brothers of French descent They had long histories of upper respiratory infections, bronchitis and pneumonia Both subsequently developed chronic lymphocytic leukemia The infections antedated by years the appearance of the leukemias

Hutchison[61] reported a case of fatal pneumonia in a young child with congenital agammaglobulinemia The patient's brother had died previously of pneumonia The writer assumed that since the histologic picture of the lungs in both were similar that both had agammaglobulinemia

Martin, et al,[63] described agammaglobulinemia in a 63-year-old woman She had recurrent upper respiratory infections with sinusitis, tonsillitis and adenitis, oral moniliasis and pleurisy There was an absence of plasma cells in the bone marrow She responded only to Gamma globulin and sulfadiazine

Hinds[69] reported a case of agammaglobulinemia in a 21-year-old male He had repeated infections He had laryngitis and sinusitis, which required antral windows He had had myringotomies for otitis media He had episodes of pneumonia, and finally bronchiectasis requiring lobectomy

O'Brien and Sereda[80] reported agammaglobulinemia in male siblings, ages three and five years The first child was seen initially because of a painful swollen right ankle, and the second because of a painful left knee Both patients had a history of repeated respiratory infections, the three-year-old having had severe almost continuous respiratory infections with acute tonsillitis, pharyngitis and acute otitis media, and, finally, pneumonia at 11 months An uncle on the maternal side died at age five Autopsy findings suggested that he, too, was afflicted with the disease, thus giving support to the theory that the defect is a recessive sex-linked characteristic similar to hemophilia

Wechsler and Wolf[81] reported a case of agammaglobulinemia in a 53-year-old female with typical findings of this disease She had been well until the age of 27 Her illnesses included bronchitis, pneumonia, eyelid infection, meningitis, otitis media and mastoiditis Because of a persistent otitis media following meningitis, she had a simple mastoidectomy

Rosenstiel[56] described a case of agammaglobulinemia in a 38-year-old white male veteran of World War II For many years he had had repeated respiratory infections requiring antibiotic therapy and on many occasions was hospitalized A review of his past medical history revealed that he had had 20 attacks of pneumonia, one attack of erysipelas, innumerable colds and sore throats, and many attacks of sinusitis requiring surgical drainage

Congenital agammaglobulinemia in two brothers has been reported by Elphinstone, et al [82] The first case had bronchopneumonia at the age of two months, left chronic otitis media at the age of three years, left mastoidectomy at the age of seven, and a right mastoidectomy at the age of eight Mucopurulent material was obtained from antral irrigations He contracted pulmonary tuberculosis between the ages of seven and eight years He had repeated episodes of pneumonia

The second case had empyema, staphylococcal infections of the nose and bronchitis

Zbar, et al,[illegible] reported agammaglobulinemia in a 27-year-old white married housewife who had had ten attacks of pneumonia A case of primary agammaglobulinemia in a four-year-old male with a history of repeated pyogenic respiratory infections was reported by Sorensen and Partington [illegible] He had mild bronchitis at the age of three months, bronchitis and otitis media at seven months, groin abscess at two years, cough and mucopurulent nasal discharge at three years, and pneumonia at the age of four

Agammaglobulinemia in a 36-year-old male has been reported by Savacool and Landes [illegible] At the age of 21 this patient developed prolonged colds, each associated with stuffiness, nasal discharge and general weakness and fatigue He had two episodes of otitis media lasting about two months He had chronic sinusitis and recurrent attacks of pneumonia He had a lobectomy for an unresolved pulmonary infection He was noted to have splenomegaly and generalized lymphadenopathy

In 1957, a case of congenital agammaglobulinemia was described by Freundlich[illegible] in a five-and-a-half-year-old boy who had a Gamma globulin level between 0 5 and 1 mg per 100 ml plasma, which was lower than any other case reported in the literature up to that time The patient underwent an unusually prolonged varicella infection At the age of one, he had a prolonged case of chickenpox and at one-and-a-half, meningitis He had episodes of diarrhea and a chronic cough with frequent periods of high temperature X-rays showed pulmonary changes and sinus disease An interesting finding was noted, namely, the absence of adenoid tissue in the nasopharynx He developed a chronic bilateral suppurative otitis media and eventually had a right mastoidectomy He had recurring bouts of sinusitis

Martin, et al,[49] have reported three additional cases of agammaglobulinemia The first case was that of a 13-year-old Negro male He had repeated episodes of respiratory infections, pneumonia with empyema and bacteremia, acute otitis media and bronchitis In addition, two episodes of septic arthritis occurred At the time of study, he had moderate bilateral bronchiectasis, chronic pansinusitis, chronic left otitis media and hepatosplenomegaly The second case was that of a 36-year-old white female who had a history of pneumonia, appendicitis with rupture, sinusitis, bronchitis, pharyngitis and cutaneous furunculosis While under study, the patient was found to have hepatosplenomegaly, early bilateral bronchiectasis, chronic frontal and maxillary sinusitis, and scattered skin lesions The third case was that of a 54-year-old white female, who following an episode at the age of 40 years of septic arthritis and bacteremia due to streptococcus pyogenes, developed 11 attacks of pneumonia and bronchopneumonia, staphylococcus septicemia, meningitis and otitis media, generalized herpes zoster, and innumerable episodes of cellulitis, sinusitis, pharyngitis and bronchitis Studies disclosed severe bilateral bronchiectasis, pansinusitis, and hepatosplenomegaly

Two additional cases of agammaglobulinemia have been described in adult females by Cooke, et al[84] Both cases had recurrent pancreatitis and steatorrhea Citron[10] draws attention to the association of acquired idiopathic agammaglobulinemia with enlargement of the spleen and lymph nodes He describes two cases and reviews six similar ones in the literature His two cases had pronounced hepatosplenomegaly and lymph node enlargement The first case, a 33-year-old man, had a history of gingivitis, laryngitis, pleurisy and pneumonia The second case, a 17-year-old girl, had a history of pneumonia and had terminal meningitis

In 1957, Pearce and Parinpanayagam[6] reported the first case of congenital idiopathic hypogammaglobulinemia in a girl in England The child was three years, eight months of age and had a history of constant infections Repeated attacks of left otitis media developed into chronic otitis media She had recurrent attacks of stomatitis, purulent rhinitis and pneumonia In addition, she had hepatosplenomegaly and palpable cervical lymph nodes A cervical lymph node exhibited the characteristic feature of absence of plasma cells

Kuhns and Solow[39] reported the occurrence of little or no

Gamma globulin in a 19-year-old female patient with a long history of susceptibility to infections with other long-standing abnormalities, most of them related to bones or skin. She had recurrent urinary infections and upper respiratory infections. She had frequent colds and a clogged nose, and she became a mouth-breather at an early age. She had a chronic cough. X-rays showed a chronic hyperplastic pansinusitis. She had a crooked spine, one leg was shorter than the other, and she had a slight bilateral hallux valgus deformity. The skin of her body showed numerous areas of depigmentation.

Barrett and Volwiler[9] described a case of acquired agammaglobulinemia in a husky 19-year-old sailor, and a case of secondary agammaglobulinemia in a 67-year-old male suffering from multiple myeloma. The sailor had two episodes of pneumonia within two months, but had no previous history of susceptibility to infection. His Gamma globulin levels were on the borderline between safe and dangerous hypogammaglobulinemia. The elder patient concurrently developed multiple myeloma and extreme susceptibility to infection. In this case the question was raised as to whether the loss of antibody production or of leukocyte production, or the failure of some other defense mechanism was responsible for the recurring infections.

A case of agammaglobulinemia in a 53-year-old housewife has been reported by Domz and Dickson.[60] The interesting aspect of this case is that she had repeated attacks of pneumonia, despite antibiotic and Gamma globulin prophylaxis.

Zelman and Lewin[37] describe a case of adult agammaglobulinemia with multiple congenital anomalies. Clinically this patient had recurring pyogenic infections, a sprue-like syndrome, and reticuloendothelial hyperplasia of the spleen and lymph nodes with granuloma formation. This association suggests the possibility that such adult "acquired" agammaglobulinemia may, in fact, represent another congenital anomaly. A hereditary basis is suggested by the presence of hypergammaglobulinemia in both parents of this patient.

CASE REPORTS

Case 1. The patient was an eight-year-old white girl who was admitted to the hospital because of a persistent left otitis media and the sudden appearance of swelling and tenderness of the left knee and right wrist. She had a history of recurrent occurrences of respiratory infections since earliest childhood. For several months she had had purulent drainage from the left ear which had failed to respond to the usual measures, including intensive antibiotic administration and radium therapy to the nasopharynx. For several years she had suffered recurrent ear infections and purulent sinusitis. She had a chronic productive cough.

She was normal at birth, however, at the age of four weeks she had an upper respiratory infection with a temperature of 103.8° F., which responded satisfactorily to intramuscular penicillin. At the age of two, she was seen many times by her family doctor for upper respiratory infections. On one of these occasions, when she had acute bronchitis and otitis media, an axillary temperature of 106° F. and rather prolonged febrile convulsions were recorded. Of interest is the fact that at the age of five she had chickenpox with secondary infection requiring intramuscular penicillin and aureomycin ointment locally. When she had the smallpox vaccination, she had a secondary reaction on the right eyelid.

At the age of six, she had many upper respiratory infections with a "stuffy nose" and discharge. This same year, she had several episodes of otitis media and pneumonia. Because of the persistent infections, arrangements were made for her to have a tonsillectomy and adenoidectomy. A significant observation was made at the time of surgery, namely the complete absence of adenoid tissue. Her tonsils were slightly enlarged, septic and because of adhesions had to be removed by dissection.

Early in 1955, at the age of seven, she was seen many times by her family physician with ear, nose, throat and lung infections. For the first time splenomegaly was noted. X-rays of the chest, made in March of this year, showed pneumonitic changes in the central and mesial lung fields which did not completely resolve. In the Spring of this year she was hospitalized by a pediatrician in a neighboring city. He reported to the family that all tests were negative; however, he gave her two transfusions, after which she showed a great deal of improvement, and, consequently, was not seen by her family physician from April, 1955 until February, 1956. From this time until her admission to the hospital she was treated for many episodes of bronchitis, purulent sinusitis and otitis media.

Physical examination revealed the following findings: temperature 98.6° F., pulse 100, respirations 20, blood pressure 100/60. The patient appeared slightly anemic, but was not in acute distress. Her weight was 55 pounds. The pupils were round and equal and reacted to light and accommodation. The nasal membranes were moderately congested and a purulent drainage was present. The maxillary sinuses were cloudy to transillumination. The right ear drum was retracted and scarred. A profuse sticky discharge was noted to be exuding from a central perforation in the left ear drum. No mastoid tenderness was present. The tonsils were absent and the fossae clear. A thick postnasal drainage was noted. On palpation of the neck several small non-tender lymph glands were noted bilaterally. The lungs were clear. The heart sounds were normal. Examination of the abdomen revealed rather marked splenomegaly. There was slight swelling and tenderness of the left knee and right wrist.

Laboratory examination revealed the following findings: urine negative, hemoglobin 10.5 gm., white blood cell count 4,200 with normal differential. Erythrocyte sedimentation rate was 12 millimeters in one hour. Serology negative, normal prothrombin time, fibrinogen normal, clot retraction two hours, prothrombin consumption 38 per cent, platelets 222,000. The antistreptolysin titer was negative. No L.E. cells were found. All agglutinations were negative. A culture from the left ear revealed a pseudomonas aeruginosa and hemolytic staphylococcus which were not sensitive to any antibiotics. Alpha streptococci and Gram negative diplococci, which were highly sensitive to a number of antibiotics were cultured from the throat. An electrocardiogram was normal. A bone marrow examination was reported as showing slight hyperplasia with increase in the percentage of nucleated red cells. A chest X-ray showed extensive changes in both lung fields with a large amount of increased density radiating out from the hilar regions. A lateral soft tissue film showed no lymphoid tissue in the nasopharynx. Sinus X-rays showed clouding of all of the paranasal sinuses. X-rays of the right wrist were normal, except for slight soft tissue swelling. Skull films showed stippled punctate calcium deposits in the skull in the region of the choroid plexus bilaterally. Films of the mastoids showed very poor cell development bilaterally with sclerosis present, but no evidence of cell break down or bone destruction.

Other laboratory findings were as follows: total serum proteins were 4.9 gms per 100 cubic centimeters, of which albumin was 4.9 and globulin 1.1 gms. The A/G ratio was 4.5/1. Serum electrophoresis showed the total protein to be markedly decreased 5.2 gms per 100 cc (normal 7.35 gm per 100 cc.). There was a complete absence of Gamma globulin. By the immunochemical method she was shown to have a serum Gamma globulin level of 100 mg per 100 cc.* There was a relative increase of the albumin fraction—72.0 per cent (normal 59.4 per cent). The patient was found to be in blood group O, Rh positive. There were no anti-B isohemagglutinins and only very weak isohemagglutinins demonstrated in her serum.

A diagnosis of agammaglobulinemia was made and the intramuscular administration of Gamma globulin was begun, the patient receiving 16 cc as an initial dose. The symptoms relative to the wrist and knee disappeared in several days following admission to the hospital. In spite of intense antibiotic therapy, the drainage from the left ear persisted, consequently, a complete simple mastoidectomy was performed. Her postoperative course was uneventful, and she was discharged 11 days following surgery on Dec. 11, 1956, in good condition, with a dry left ear. Since this hospital admission, she has received 10 cc of Gamma globulin bimonthly. In addition, she has been maintained on prophylactic doses of penicillin.

For the next six months the patient did well generally, except for a chronic maxillary sinusitis. At the age of nine, in July, 1957, bilateral antral windows were made. Much pus and hyperplastic tissue were removed from each maxillary sinus. In November of the same year, she was admitted to the hospital for treatment of otitis media and bronchitis. A chest X-ray showed progression of the infiltrative changes in both lungs. With this admission she had a leukopenia, the white blood cell count being 3,000. Serum protein analysis revealed a total serum protein of 5.4 gm per 100 cc and a Gamma globulin of 2.5 per cent (practically absent). The drainage from her ears cleared promptly and the bronchitis improved on chloromycetin therapy.

For the past ten months the patient has done very well as far as her sinuses and ears are concerned, however, at the time of her last admission in July, 1958, her pulmonary symptoms had shown no improvement. Her nasal membranes were normal, no pus was present in her nose, and there was no postnasal drainage. Her maxillary sinuses were clear to transillumination. Small central perforations were present in both ear drums, but no drainage was present. An interesting observation was that in the past year, intermittently, she has had painful, feverish swelling of her knees, wrists, shoulders and hips. These symptoms subside spontaneously in about five days. Serum electrophoresis revealed a total protein of 5.6 gm. per 100 cc., and a serum Gamma globulin of 3.8 per cent. The zinc sulfate turbidity test was reported as being 0.25 units (values of 0 to 1 unit are compatible with agammaglobulinemia). The sweat test was negative. A chest X-ray showed further progression of the pulmonary changes. A chest consultant advised against definitive procedures on the lungs, as he felt that if the changes were those of bronchiectasis they were almost surely widespread and not amenable to any but general measures. Electrophoretic studies revealed normal serum proteins in all of the members of her family except for her father whose Gamma globulin was in the lower limits of normal.

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*Dr. David Gitlin, Children's Hospital, Boston, Mass., kindly performed this test.

Case 2. The patient was a four-year-old white boy, who was admitted to the hospital Jan. 20, 1958, for a tonsillectomy and adenoidectomy. He had suffered from repeated ear and upper respiratory infections, and his mother stated that he kept a "stuffy" nose through the Winter months. He was small and underweight. For about two months he had had a left otitis media, the drainage having ceased about one week prior to this admission.

He was born at home and was premature, weighing four pounds at birth. At the age of two weeks he was hospitalized with pneumonia. His temperature went up to 107° F., and he had convulsions for five days. After six weeks in the hospital, he was discharged, weighing five pounds and eight ounces. At the age of six months he was again admitted to the hospital with a temperature of 104° F. He cried continuously during his eight day stay,, and his mother was told that he was being treated for "gas on the stomach." At the age of 15 months, he was treated at home for pneumonia.

His present family physician saw him for the first time at the age of 18 months. His history mentions the fact that up to this time he had had about ten episodes of acute purulent otitis media. He weighed 19 pounds and three ounces, and his hemoglobin was 6.5 gm. The anemia was apparently nutritional, as it responded satisfactorily to iron therapy. His mother states that he had "three day measles" (Rubella) at the age of two.

At the age of 27 months, in January, 1956, he was hospitalized briefly for gastroenteritis, bronchitis and otitis media. A urinalysis was normal. His hemoglobin was 14.5 gm. and his white blood cell count was 8,050. A chest X-ray was negative.

For the next two years he was taken to a pediatrician and his family doctor on many occasions because of poor appetite, failure to gain weight and repeated colds and ear infections. During this period he had seven recorded episodes of otitis media.

Physical examination revealed the following findings: temperature 98.6° F., pulse 80 and respirations 20. The patient appeared small for his age and undernourished. His weight was 34 pounds. The pupils were round, equal and reacted to light and accommodation. The nasal membranes were moderately congested. The maxillary sinuses were cloudy to transillumination. A central perforation measuring 2 mm. by 1 mm. was present in the left ear drum. No discharge was present. The tonsils were small, fibrotic and imbedded. The adenoids were not visualized, but on palpation were thought to be small. A few small non-tender lymph glands were palpated in the superior cervical areas on each side. The lungs were clear and the heart sounds normal.

Laboratory examination revealed the following findings: urine negative, hemoglobin 13 gms., white blood cell count 9,900, with a normal differential, bleeding time two and a half minutes, and coagulation time ten minutes.

At operation the tonsils were found to be small, fibrotic and many adhesions were present. Only a minimal amount of adenoid tissue was present in the nasopharynx. The microscopic examination of the tonsils was reported as follows: "The tonsils are covered in part by a benign stratified squamous epithelium, but the greater portion of the epithelium is missing. No significant exudate is present within the crypts. The lymphatic tissue does not appear significantly hyperplastic." The patient did well following surgery, and was discharged on his first postoperative day.

Because of the paucity of adenoid and tonsil tissue and the clinical history of recurrent ear and respiratory infections, agammaglobulinemia was suspected. After recuperating from the tonsil surgery, further studies were done. By the paper electrophoretic technique, the total protein was slightly decreased, 6.2 gms. per 100 cc., of which albumin was 4.3 gms. and globulin 1.9 gms. The Gamma globulin was 6.9 per cent (normal 14.4 per cent). There was a relative increase of the albumin fraction—69.4 per cent (normal 59.4 per cent).

These studies indicated an abnormality of the serum proteins and a very low Gamma globulin. Although a definite diagnosis of agammaglobulinemia could not be established, because of the clinical history administration of 8 cc. of Gamma globulin each month was started in March, 1958. At this time X-rays of his sinuses, lungs and mastoids were made. The maxillary sinuses were almost completely opaque. The lung fields were clear, and the heart, aorta and mediastinum were normal. The mastoids were normally aerated and showed no definite evidence of infection.

For the last six months the patient has done well except for an occasional attack of sinusitis. The left ear has remained dry but the drum perforation has not healed.

Additional laboratory studies were done in August, 1958. The total serum protein by chemical analysis was 6.4 gm. per 100 cc., of which albumin was 4.65 gm. and globulin 1.75 gm. By serum electrophoresis the Gamma globulin was shown to be 1.9 per cent (normal 12.2 per cent), which was much less than the initial determination of 6.9 per cent, despite Gamma globulin administration. The patient was found to be in blood group O, Rh positive. Only very weak anti-A and anti-B isohemagglutinins were demonstrated in his serum; his anti-A isohemagglutinin titer was 1:1 and his anti-B titer was 1:4. The zinc sulfate turbidity test was reported as being 0.25 units (values of 0 to 1 unit are compatible with agammaglobulinemia). These tests confirmed the diagnosis of agammaglobulinemia.

COMMENT

Cases of agammaglobulinemia in a young girl and a young boy have been reported, exhibiting the complete pattern of this syndrome. The repeated infections in these patients must be attributed to their deficiency of Gamma globulin and concomitant poor antibody response.

The initial examination of the serum of the first case by paper electrophoresis showed a complete absence of Gamma globulin. After the administration of Gamma globulin, subsequent electrophoretic studies revealed small quantities of this serum component. The first examination of the serum of the second case showed a Gamma globulin level of 6.9 per cent, which was not considered low enough to be considered diagnostic of agammaglobulinemia. In spite of Gamma globulin administration, serum electrophoresis six months later showed the Gamma globulin to be almost completely absent. A diagnosis of agammaglobulinemia was then confirmed.

Both of these patients demonstrated weak or absent isohemagglutinins. The zinc sulfate turbidity test in each case was compatible with agammaglobulinemia.

Although the girl has had repeated and varied bacterial infections, she apparently has developed immunity to the common viral infections, which is characteristic of this syndrome. The boy has had only Rubella, and so far has escaped the common acute infectious viral diseases of childhood so that his ability to develop immunity to viruses has not been tested.

Of course, the striking feature in these patients is the occurrence of repeated infections of the nose, sinuses, ears, throat and lungs, the diseases which lie within the field of the otolaryngologist.

The absence of adenoid tissue in a four and a six-year-old child with repeated respiratory infections is a most unusual finding; consequently, when one discovers very little or an absence of lymphoid tissue in the nasopharynx of a child, agammaglobulinemia should be strongly suspected.

The first patient has splenomegaly and lymph node enlargement implying disease of the reticuloendothelial system, which is a common finding in this disease. On two occasions she has had neutropenia and never responded with leukocytosis to acute infection. Granulocytopenia during infection has been reported in several patients with agammaglobulinemia.[3] One patient was regarded for years as having recurrent splenic neutropenia before the diagnosis of agammaglobulinemia was made[3] This patient has had the polyarthritis which has occurred in about one-fourth of the reported cases The second case has had very minimal lymph node enlargement, but no splenomegaly, neutropenia, or polyarthritis

In spite of Gamma globulin administration and penicillin prophylaxis, the time-honored surgical procedures of the otolaryngologist were necessary to eradicate ear and sinus disease in the first case The progression of the pulmonary changes despite Gamma globulin administration, antibiotic prophylaxis and surgical correction of sinus and ear disease should also be emphasized

The first case, a girl, is believed to represent acquired agammaglobulinemia, although the onset of infections shortly after birth would lend support to a diagnosis of the congenital type The advanced pulmonary pathology is more commonly described in the acquired type Her sex is in favor of the acquired type, since congenital agammaglobulinemia is supposed to occur only in males A few cases have been reported in females,[6,15] and statistically there is a rare chance of this happening[1] The second case, a boy, is thought to represent the congenital type.

The writer has two additional cases with very low Gamma globulin levels under observation Both of these patients have chronic ear and mastoid disease Perhaps all patients with recurring and chronic ear and mastoid infections should have electrophoretic serum studies as has been recommended for all cases of bronchiectasis [43 55]

SUMMARY AND CONCLUSIONS

1 The disease entity agammaglobulinemia has been described in detail and the literature reviewed

2 Typical cases of acquired agammaglobulinemia in a young girl, and congenital agammaglobulinemia in a young boy have been reported The possibility of the disease in the girl being of the congenital type has been discussed

3 Six years of case reports and study by competent workers

has greatly widened our knowledge of an unusual and important syndrome

4 The otolaryngologist often has the first opportunity to make the diagnosis of agammaglobulinemia, whether it is the congenital or the acquired type He should strongly consider this diagnosis in any patient with recurring acute or chronic infections of the respiratory tract He should also think of this diagnosis in any patient showing a striking deficiency or absence of adenoid tissue

5 Paper electrophoresis of the serum proteins, which is now generally available, will usually establish the diagnosis Other presumptive clinical, laboratory and X-ray findings have been reviewed

6 As agammaglobulinemia is closely tied up with the immunologic mechanisms, there are many unsolved problems in medicine whose nature may be clarified with further study of this disease, particularly in the field of protein metabolism and resistance to infection

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209 Medical Arts Bldg

CYTOMEGALIC INCLUSION DISEASE OF THE NOSE*

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Cytomegalic inclusion disease, or generalized salivary gland virus infection, is ordinarily thought of as a multisystem virus disease occurring mainly in children and stillborn infants, however, it can manifest itself in organs of interest to the otolaryngologist. It is the purpose of this thesis to aid in the diagnosis and elucidation of an occasional unusual salivary gland or nasal disease. The disease has been characterized by "owl-eyed" giant cells with nuclear and cytoplasmic inclusions. The cells are seen in the epithelium of various viscera and the cerebrum. Although such cells had been described as long ago as 1904 by Jesionek and Kiolemenoglou,[1] who observed them in kidneys of a stillborn infant, it is only recently that the disease has been recognized as an entity of some importance which can occur in any age group in a mild localized form or a fatal disseminated type. Following the original work, Ribbert[2] reported the same inclusions occurring in a newborn infant and in the salivary glands of two older children. The term "cytomegalic" was suggested first by Goodpasture[13] to differentiate this entity from other inclusion diseases. Since then, numerous reports of this disease have appeared in the literature. The cases described were usually diagnosed at autopsy. Smith and Velios[7] reviewed the literature comprehensively in 1950. They found 69 cases and added 20 previously unreported cases. Medearis[20] has presented an excellent review of 132 cases.

The characteristic large and pathognomonic mononuclear cell, upon which the diagnosis is usually based, has been well described by Cappell and McFarland,[3] and by Watt, Saxton, and Lee.[4] This large cell is usually epithelial, and rarely mesenchymal or endothelial. It is considered by most pathol-

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ogists to be easily recognized and distinct from other diseases with intracellular inclusions. The extraordinary size of the cell, up to 35 microns, and the deeply staining acidophilic or purplish nuclear inclusion with a surrounding clear halo extending to the nuclear membrane are characteristic. The nuclear membrane is distinct, and near it are dark chromatin masses of different sizes. The cytoplasm is abundant and either acidophilic or amphophilic. In it are basophilic inclusions which usually arrange themselves in a crescentric pattern near the periphery.

The etiology of the disease is considered by most investigators to be a virus, related to the salivary gland virus found in laboratory animals, because the cytoplasmic and intranuclear inclusions characteristic of cytomegalic disease are similar to inclusions found in other human and animal disease of known virus etiology. There is evidence of species specificity of the virus, because to date, there has been no reported successful transfer of the disease to animals, however, this is to be expected, since the salivary gland inclusion disease occurring in rodents and other animals has been found to be species specific.[6] This isolated filterable virus, apparently of low virulence in the rodents, was capable of transmitting the disease to other animals, with intranuclear inclusions found at the site of injection, and salivary glands. At times, a generalized infection was produced with pathological changes in the lungs, abdominal organs, and brain.[5] This appears to be analogous to the situation described in man. Weller[10] has isolated the virus from liver biopsies (showing cytomegalic inclusions) and from the urine of children with cytomegalic inclusion disease, propagated it in human fibroblasts and produced *in vitro* cellular changes with nuclear inclusions. Rowe[21] and Smith[22] have isolated from human tissuse the filterable virus capable of producing the typical cytopathological inclusions of this disease. The virus also produced complement-fixing and neutralizing antibodies with species specificity. The presence of antibodies to the virus in a high percentage of the population indicates that it is of wide distribution, therefore, it exists in a latent or asymptomatic form. The virus has been further described and studied by filtration and the electron microscope.[16]

In man, the salivary gland virus infection is often transmitted from an asymptomatic mother to her fetus The newborn infant may appear healthy but have a latent infection with salivary gland involvement, or the fetus may have a disseminated infection apparently because of greater susceptibility of fetal tissues [3] Debility of the child or adult by a concomitant disease, such as avitaminosis, is felt to predispose to generalized infection Fatal generalized infection most often occurs in the first several years of life

Pathologically, in man, various systems and organs are involved There is epithelial involvement of the salivary glands, alveoli, and bronchi of the respiratory tract, the tubular epithelium of the kidney, liver, and gastrointestinal tract The brain, eye, pancreas, and adrenal glands are less commonly involved Apparently the organs may be involved individually or in any combination In addition to the large incision-bearing mononuclear cell, the histological picture shows infiltration of lymphocytes and macrophages, a fibroblastic reaction, and occasionally necrosis

In the kidney there is a focal interstitial nephritis and tubular dilatation with desquamation The nephrosis or nephritis produced is rarely severe Because of the renal tubular involvement with exfoliation of cells into the urinary tract, Wyatt,[4] in 1950, first suggested the use of urinalysis and identification of the characteristic cytomegalic cell as a diagnostic aid Actual demonstration of the cell in cytologic preparations of urine sediment was not made until 1951 by Fetterman,[9] and later by Mercer [8] Chronic or interstitial pneumonitis is one of the most frequent manifestations Pulmonary cysts, atelectasis, and necrosis have been reported [11 14] The alveolar cells are swollen and many contain inclusions and have desquamated into the lumen with a protein coagulum [12] In the brain, focal areas of encephalitis with mononuclear infiltration with intranuclear inclusions have been found in ganglia, glial cells, and also meninges Necrosis and hemorrhage in the basal ganglia, hydrocephalus, and diffuse intracranial calcifications have also been seen Liver damage results from periportal inflammation, necrosis, biliary obstruction, and fibrosis Inclusions in the cord and bile duct cells, leukocytic infiltration, and accumulation of fat in the parenchymal cells has been seen[12] Biliary cirrhosis has been reported A blood dyscrasia is a rather frequent finding in infants with some of the following manifestations purpura, splenomegaly, anemia, petechiae, thrombocytopenia, hemorrhage, and extramedullary blood formation The large and small intestine have at times shown infiltration with cytomegalic cells and ulceration Ocular pathologic changes have been uveitis, cataract, and retinal infiltration with inclusion cells

Inclusion disease in the salivary glands and pancreas has been rather frequently reported with cellular infiltration, interstitial fibrosis, and small foci of necrosis As yet, there has been no correlation with newer concepts of salivary gland disease, brought about by refinements in sialographic technique[10] One may only speculate what relationship exists between non-obstructive sialectasis, Mikulicz's disease, "chronic asymptomatic enlargement," and cytomegalic inclusion sialadenitis

In the diagnosis of cytomegalic inclusion disease there has been confusion with syphilis, toxoplasmosis, hemolytic disease of the newborn, sepsis, measles, and serum hepatitis The differential diagnosis from most of these diseases is made by the appropriate blood test The disease pattern and resulting clinical picture varies widely with the type of involvement It may be either an acute or chronic, occasionally febrile disease, which may feature interstitial pneumonitis, nephrosis, cirrhosis, blood dyscrasia, or central nervous system manifestations The signs and symptoms may include fever and weight loss, dyspnea, cough, jaundice, hepatomegaly, and splenomegaly, purpura, petechiae, hemorrhage, mental and motor retardation, vomiting and diarrhea Skull X-rays may show diffuse calcifications outlining the ventricles Liver and lung biopsies have been successfully used for diagnosis[12] The less traumatic examination of body fluids seems to have more promise The diagnosis may be confirmed by identification of the inclusion-bearing cell in stained smears of urinary sediment, salivary and bronchial secretions, or gastric washings[24] The smears are fixed on slides in equal parts of ether and 95 per cent ethyl alcohol for 20 minutes, then stained with hemotoxylin and eosin or Wright's stain A negative test does not invalidate the diagnosis

The treatment is mainly symptomatic, since there is no specific vaccine and a specific anti-viral therapeutic agent is unavailable Birdsong[17] feels that there is a rationale and evidence to support the use of Cortisone, and that in infants exchange transfusion may be valuable Gamma globulin for its antibodies and antibiotics may be used in addition to supportive therapy, also of value is the occasional removal of diseased tissue, as will be seen in the following patient who typifies an apparent localized disease

Mrs J J a 56-year-old colored schoolteacher, was first seen Oct 9 1954 complaining of a burning sensation in her nose of two months' duration There was also frequent sneezing bloody and yellow thin drainage, and nocturnal nasal obstruction Examination revealed erythematous crusted, and weeping lesions of the nasal vestibule The nasal mucosa appeared about normal in color but was slightly swollen and the surface granular The remainder of the examination was not unusual A diagnosis of non specific nasal vestibulitis was entertained and the patient was instructed in the topical application of a neomycin and bacitracin ointment Routine X-ray examination of the paranasal sinuses was negative She did not keep her next appointment, and was not seen again until January 29, 1957, at which time she stated she had progressive nasal obstruction and continued rhinorrhea Examination revealed the nasal vestibulitis again but now the nasal chambers were filled by pale hyperplastic mucosal changes The appearance differed from that of the allergic nose with polyps in that the lesions varied in size but were generally smaller than allergic polyps, and arose from the entire lateral wall as well as the septum Although the larger growths had a smoother surface, the smaller ones appeared nodular or papillary

On Feb 24 1957 she was admitted to the hospital, where she gave a history as above and that she had constant obstruction of the left nostril for the past year and frequent 'colds" She complained of mild exertional dyspnea and occasional precordial pain She had previously been treated for hypertension and nervousness She denied having a cough bronchial sputum, ankle edema weight loss jaundice or melena There was nocturia one or two times Her only operation had been a hysterectomy and bilateral salpingo-oophorectomy in 1945 She had two pregnancies and had borne two children who were living and well There was no known history of allergy either personally, or in her family Except for the rhinological findings a blood pressure of 180/93 and on retinoscopy vasospasm and arteriovenous nicking the physical examination was not remarkable The liver and spleen were not palpable

Examination of the blood revealed 12 6 grams of hemoglobin, red blood cell count of 4 48 mil/mm^3, hematocrit 39 per cent The white blood cell count was 9 350 with 55 segmented polymorphonuclear leukocytes six monocytes and one eosinophil

On Feb 25 1957, under local anesthesia an extensive intranasal removal of these lesions was carried out without difficulty except for greater bleeding than was expected The pathological report is as follows

"Grossly the specimen consists of multiple pieces of edematous soft membrane which in some areas is almost translucent. The membranes together form a mass measuring 5x4x.5 cm. Multiple small polypoid nodules arise from the surface of the membrane. On microscopic examination the sections consist of a papillary and polypoid structure, which is lined by pseudostratified columnar epithelium, showing areas of squamous metaplasia and transitional type of epithelium. The papillary projections consist of moderately edematous stroma containing dilated blood vessels and lymphatics and showing marked infiltration of lymphocytes, and in some areas occasional neutrophiles. The epithelium is everywhere well demarcated from the stroma by a fairly distinct basement membrane. In the epithelium are many large cells with nuclear inclusions surrounded by a clear halo (see Fig. 1). There are remnants of nuclear material at the periphery contained by a very distinct and sharp nuclear membrane. In the more advanced lesions the epithelial cells appear distended by large inclusion bodies which are sharply outlined, round and show moderate variation in size. The inclusion bodies are much larger than ordinary cells and sometimes only an outline of the cell membrane and nucleus is recognizable (see Fig. 2). In the more advanced lesions, apparently the entire cell is destroyed by the inclusion body. The diagnosis was cytomegalic inclusion disease.

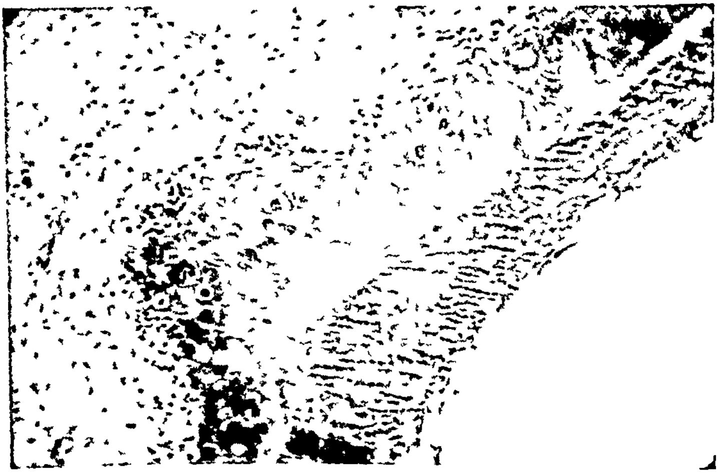

Fig. 1. One can see a number of cytomegalic cells in epithelium with the submucosal reaction.

Postoperatively she received penicillin for several days. Except for a nodular recurrence on the left inferior turbinate a month later the nose cleared remarkably well. Six months postoperatively there was a recurrence of the vestibulitis lasting two weeks. A year and a half postoperatively she was asymptomatic. Examination of the nose revealed only a mild pale granular hyperplastic mucosal change of the middle turbinates.

Complete blood count was as follows: hemoglobin 12.2 gm. or 84 per cent, red blood cells 4.6 million, micro hematocrit 39, white blood cell count, 8,150, with 45 polymorphonuclear leukocytes, 49 lymphocytes, five monocytes, and one stab The urinalysis revealed numerous epithelial cells but none with inclusion bodies, occasional granular casts, three to five white blood cells and an occasional red blood cell per high power field A trace of occult blood was found in the urine One might wonder whether early renal cytomegalic disease were developing

SUMMARY

The history of cytomegalic inclusion disease has been traced since the finding of an unique cell in 1904 Laboratory and research work establishing the etiologic agent as the salivary gland virus has been reviewed Our present knowledge of the epidemiology of the disease is not complete Cellular and organ pathology has been tabulated The clinical picture, differential diagnosis, diagnostic tests, and treatment were outlined

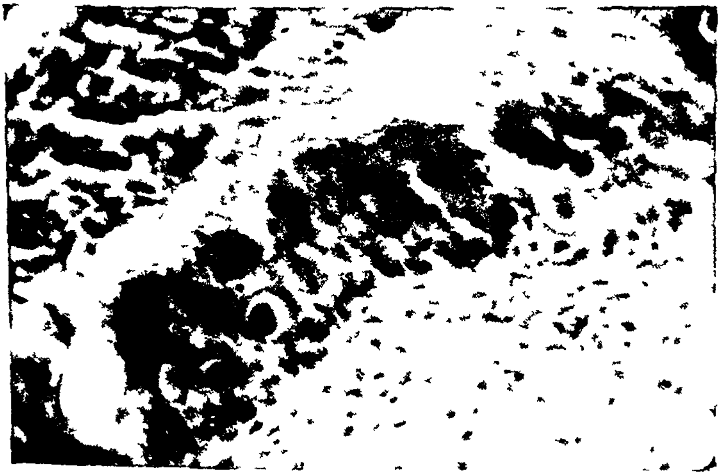

Fig 2 High power magnification showing the dark nuclear inclusion with the surrounding clear halo

In conclusion, evidence has been presented of the existence of an unusual specific virus disease entity which may infect the nasal or other respiratory passages, or the salivary glands When confronted by a patient with unusual hyperplastic and obstructive nasal mucosal changes, cough, or unexplained salivary gland disease, cytomegalic inclusion disease should be considered and excluded by a careful history and system review, general physical examination, laboratory studies with examination of stained fresh nasal and salivary gland secretions, and the urinary sediment for the diagnostic cell

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1838 Parkwood Ave

OTOLOGICAL FUNCTIONAL MICRO-SURGERY POST GRADUATE COURSE

A practical course of surgical techniques in otological surgery will be conducted by Dr Michel Portmann at the University of Bordeaux, France, Ear, Nose and Throat Department, June 26 to July 5, 1960 This course will be given in English

The number of assistants will be limited to twenty, divided into two groups The fee of $100 00 is to be paid at the Faculty of Medicine of Bordeaux, at the beginning of the course Applications should be sent to Dr Michel Portmann, 45 Cours Foch, Bordeaux, France

MULTIPLE NEUROFIBROMATOSIS INVOLVING THE LARYNX

EUGENE L YURICH, M D ,
and
G JAN BEEKHUIS, M D ,

Madison, Wis

INTRODUCTION

Multiple neurofibromatosis was classically described by von Recklinghausen in 1882 It is known mainly to dermatologists because of the typical skin manifestations of multiple cutaneous tumors and "cafe-au-lait" spots In addition, these tumors may be found to involve the viscera and the central nervous system There may also be associated malformation of bone and mental disturbances of the deficiency type

In order to avoid a confusion in terminology, one might best refer to this disease entity as multiple neurofibromatosis, or "von Reckinghausen's disease of the nervous system "[1] In so doing, one would differentiate it from von Recklinghausen's disease of bone (generalized osteitis fibrosa cystica)

Crowe and his associates[2] in 1956, while reviewing 227 cases over a 16 year period, found the incidence of this disease to be one per 2,500-3,300 births Despite his relative frequency, concomitant involvement of the larynx has been extremely rare

REVIEW OF THE LITERATURE

In reviewing the literature, we found eight previously reported cases of multiple neurofibromatosis with associated involvement of the larynx In one of these as reported by Hoover,[3] there were no skin manifestations, however, as reported by one author,[1] neither cutaneous tumors nor "cafe-au-lait" spots need be present to establish the diagnosis

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Author	Age	Sex	Site	Treatment	Comment
Colledge[4] 1930	44	F	Right Aryepiglottic Fold and Pyriform Sinus	Lateral Thyrotomy	Symptoms of Hoarseness and Dysphagia
Hoover[1] 1940	Teens	F	Right False Cord and Aryepiglottic Fold	Thyrotomy	Lesions of Pharynx, Retro tracheal Area and Mediastinum but No Skin Lesions
Helg[5] 1950	7	F	False Cord and Aryepiglottic Fold	Tracheotomy and Thyrotomy	
Holinger and Cohen[6] 1950	27	M	Left Pyriform Sinus	Removed with Snare Forceps	Pedunculated, Very Vascular Tumor, Dyspnea and Dysphagia
Figi and Stark[7] 1953	41	M	Both Arytenoids, Aryeppiglottic Folds and Retrocricoid	Biopsy	Hoarseness
Figi and Stark[7] 1953	50	F	Left Arytenoid and Retrocricoid	Cutting Diathermy Under Suspension Laryngoscopy	Hoarseness
Gibbs Taylor and Young[8] 1957	3 mo	M	Retrocricoid	Tracheotomy	Progressive Stridor
Burgdorf[9] 1954	17	F	Retrocricoid	Biopsy	Dysphagia

Fig 1

The reported cases and pertinent data are demonstrated in the following chart (see Fig 1) We have purposely omitted cases of solitary neurofibroma of the larynx in our review

PATHOLOGY

In multiple neurofibromatosis, there appears to be a distinct hereditary factor present, with several persons in one family at times being affected There is an increased incidence of other congenital malformations and a greater tendency toward the development of pheochromacytoma of the

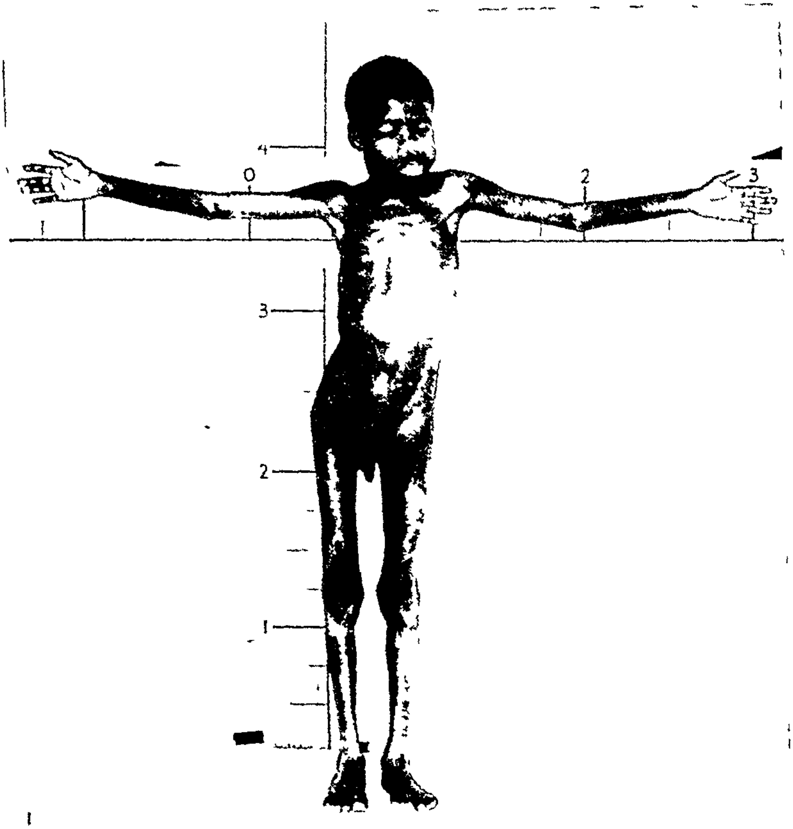

Fig 2 The patient demonstrates shortness in stature and obvious bone deformities

adrenal gland[1,10] Harrison[10] states that 5 per cent of patients with pheochromacytoma have associated neurofibromatosis

Grossly, the tumor involving the larynx may or may not be encapsulated, with a greater tendency toward poor encapsulation in multiple neurofibromatosis as compared to the usual well encapsulated solitary neurofibroma The tumor apparently grows along the nerve trunk and the common sites of laryngeal involvement are the arytenoids and the aryepiglottic folds

Although the histologic appearance includes both neural (ectodermal) and connective tissue (mesodermal) elements, it is now generally accepted to be neuroectodermal in origin The tumor arises from the Schwann cells which sheathe all the nerve fibers outside the central nervous system

These tumors are usually benign and become clinically significant when encroaching on vital structures Helg[illegible] disputes the possibility of malignant degeneration and recurrence following removal Harrison[10] feels that 5 to 10 per cent of neurofibromata may undergo malignant change Burgdorf[9] is of the opinion that incomplete removal or surgical trauma may act to precipitate malignant degeneration in these tumors

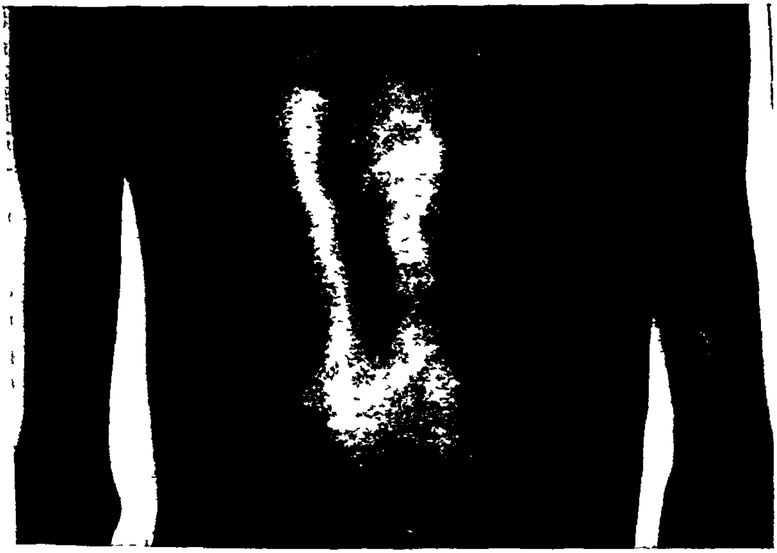

Fig 3 Multiple cutaneous nodules are evident over the trunk

DIAGNOSIS

In a patient with generalized neurofibromatosis and symptoms of hoarseness, and/or dyspnea, and/or dysphagia, one strongly suspects the possibility of laryngeal involvement On examination of the larynx, a smooth, submucosal mass is evident, usually in the area of the arytenoid and aryepiglottic fold The extent of the lesion may be further evaluated by direct laryngoscopy and roentgenograms of the neck, including

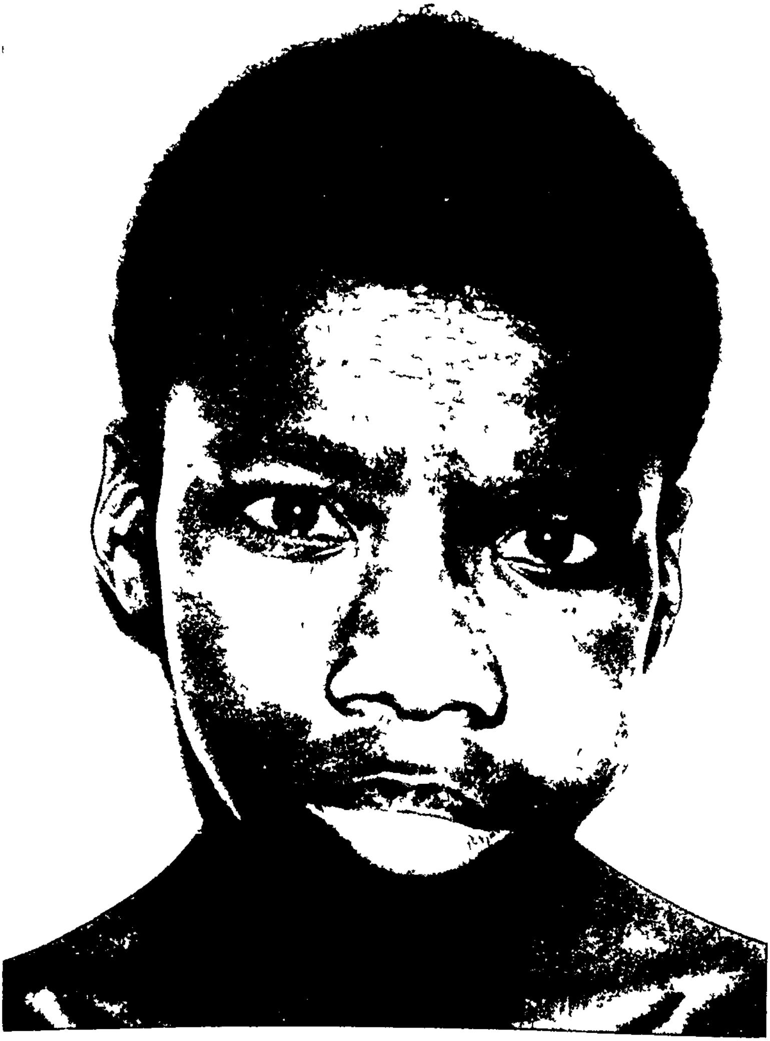

Fig 4 Here the patient exhibits the cutaneous nodule over the left cheek

laryngograms and laminograms In cases where clinical findings are doubtful, a biopsy should be obtained for confirmation

REPORT OF A CASE

J G is a 19 year old Negro male who was first seen in the Department

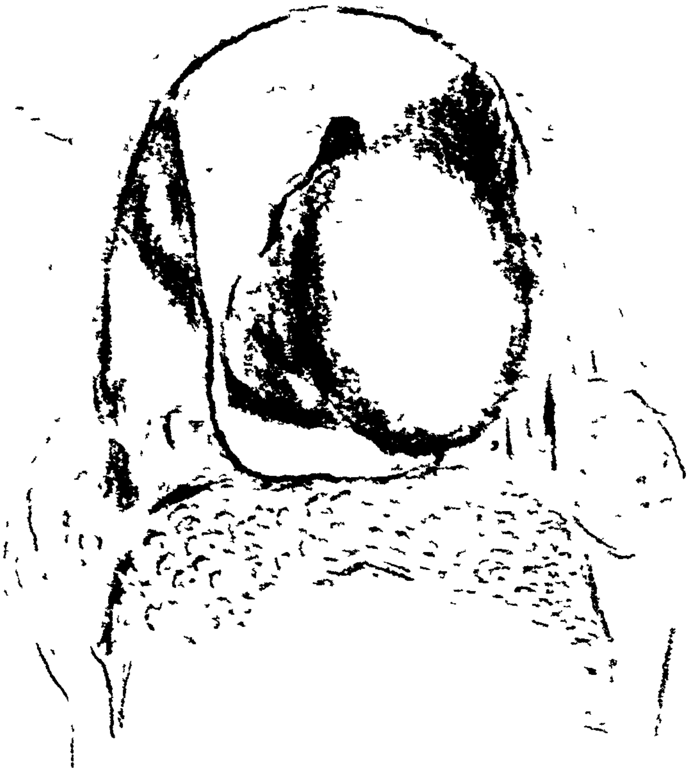

Fig 5 An artist's sketch reveals the extent of the lesion in the larynx Note the encroachment of the laryngeal lumen by the mass

of Otolaryngology on Nov 5, 1957 with a two year history of progressive hoarseness and "noisy" breathing Prior to that time, he had been followed in the Endocrine Department because of selective hypofunction of the pituitary gland and hypoadrenalism A diagnosis of multiple neurofibromatosis was made in 1948 when biopsies from the left cheek and other cutaneous surfaces were taken

Past History He was found to be mentally and developmentally retarded but nevertheless completed the ninth grade in school

Family History His maternal grandfather was four feet, ten inches in height and had many skin tumors" His mother has typical mollusk-like neurofibromata over the posterior neck area

Physical Examination General examination revealed a thin short (four

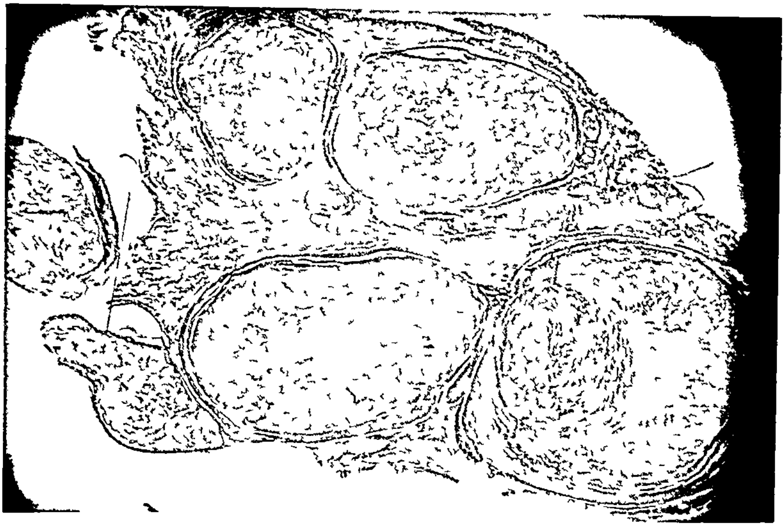

Fig 6 Microscopic appearance of lesion under low power

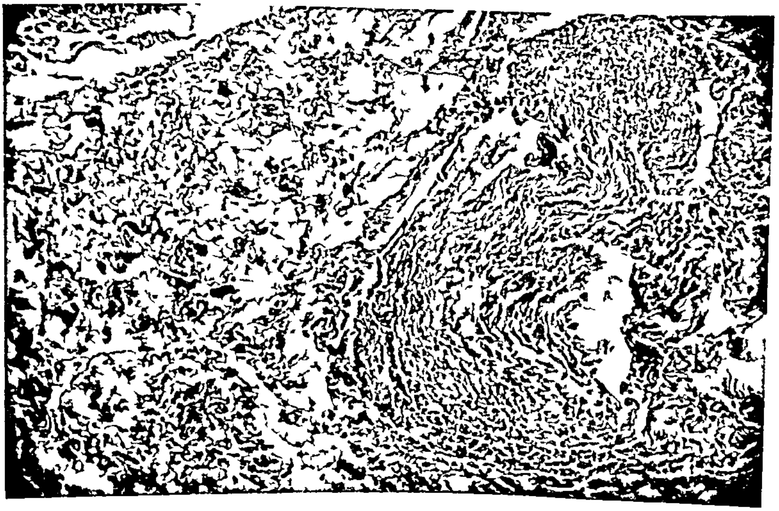

Fig 7 Microscopic appearance of lesion under higher power

feet, eight inches), colored male weighing 50 pounds (see Fig 2) There was an obvious anterior chest deformity and scoliosis Sensorium was clear but below chronological age Over the trunk and extremities were multiple 1x2 cm cutaneous nodules (see Fig 3), with pigmented macules on his back measuring 1 to 5 cm in diameter There was also a 3x4 cm mass involving the left cheek (see Fig 4) He exhibited marked hoarseness and inspiratory stridor Indirect laryngoscopy revealed a large, submucosal mass involving the left arytenoid, left aryepiglottic fold with almost complete filling of the lumen of the larynx and obliteration of the pyriform fossa

On Nov 27, 1957, a preliminary tracheotomy was performed, after which, under general anesthesia, a horizontal skin incision and dissection through the thyrohyoid membrane exposed the tumor mass It was directly continuous with the left superior laryngeal nerve and occupied the entire region of the left arytenoid, left aryepiglottic fold and left ventricular band (see Fig 5) The tumor was poorly encapsulated and firm adherence to the mucosa necessitated entry into the pharynx at several points A portion of the left arytenoid cartilage was also removed with the specimen Mucosal surfaces were approximated to afford primary healing

Pathology Grossly, the specimen had a pearly white color, nodular surface and was pyramidal in shape, measuring 8x4x2 cm Microscopically, there were hypertrophied masses of loose tissue with sheets of cells arranged in whorled like fashion (see Figs 6, 7) The smaller masses were similar to nerves

Diagnosis Plexiform neurofibroma

COMMENT

The case presented gives a typical picture of multiple neurofibromatosis with generalized cutaneous nodules, "cafe-au-lait" spots, bony deformities and mental deficiency The family history points to the hereditary tendency of this disease In addition, there is the selective pituitary hypofunction with dwarfism and adrenal insufficiency The small stature of the maternal grandfather is an interesting historical fact

The presence of laryngeal involvement is a rare concomitant finding, but the location and gross observations herein described are in accord with those few cases reported in the past

Surgical excision via a lateral pharyngotomy or thyrotomy approach appears to be the safest and surest means of complete removal of this type of tumor, especially in our case, where it was so poorly encapsulated

SUMMARY

1 Multiple neurofibromatosis with associated laryngeal involvement is a rare finding

2 A case is presented of multiple neurofibromatosis with

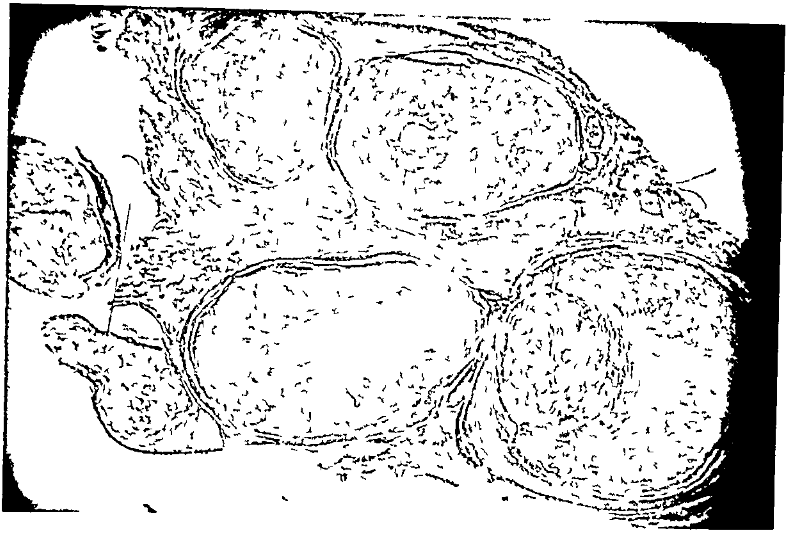

Fig 6 Microscopic appearance of lesion under low power

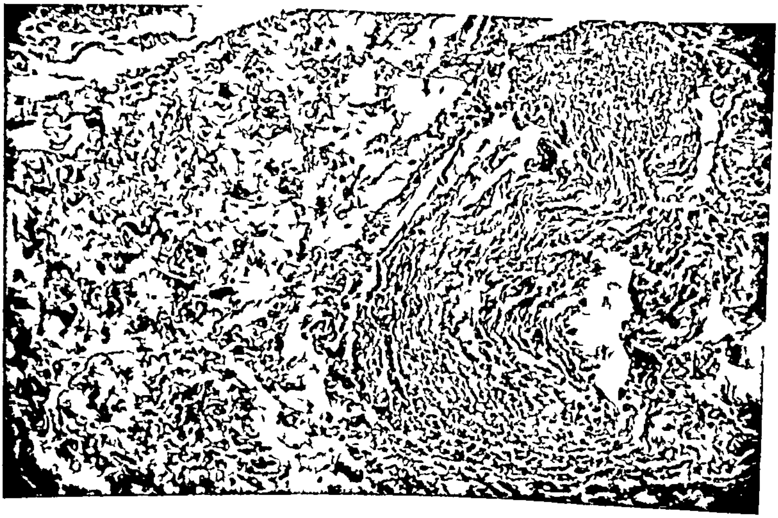

Fig 7 Microscopic appearance of lesion under higher power

feet, eight inches), colored male weighing 50 pounds (see Fig 2) There was an obvious anterior chest deformity and scoliosis Sensorium was clear but below chronological age Over the trunk and extremities were multiple 1x2 cm cutaneous nodules (see Fig 3), with pigmented macules on his back measuring 1 to 5 cm in diameter There was also a 3x4 cm mass involving the left cheek (see Fig 4) He exhibited marked

GLOMUS JUGULARE TUMORS

A Report of Seven Cases*†

CHARLES E KINNEY, M D,
and
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Cleveland, O

Glomus jugulare tumors are rather rare lesions arising from cells first identified and described by Stacy Guild in 1941,[1] situated in the adventitia of the jugular bulb and the ramus tympanicus of the glossopharyngeal nerve

Following the first documented case of this tumor reported by Harry Rosenwasser in 1945,[2] the literature on this subject has progressively enlarged from the 18 cases reported by the Armed Forces Institute of Pathology in 1951,[3] to a single series of 23 patients documented in 1958[4]

This article is to report a series of seven glomus jugulare tumors accumulated from 1947 to 1958 in the office practice of Dr C E Kinney

Case 1 This 43 year-old white female was first seen in 1945 with a chief complaint of left ear pain All symptoms were referred to the introduction of a hairpin into the ear 15 years prior, following which fragments of tissue were intermittently removed from the ear for about one year Four years later a biopsy was performed and the patient received multiple X-ray treatments for three years Two episodes of bleeding were recalled in addition to a frequent ear discharge

When examined, there was dry blood on the canal floor and a pink colored thickened drum which appeared to pulsate No true granulations were present Hearing was 50 per cent of normal in the left ear

Frequent examinations for the next two and a-half years showed little subjective or objective change until pain developed, and the drum became redder and bulged A radical mastoidectomy was performed in 1947 for a tumor mass filling the middle ear, mastoid cavity and eroding the wall of the lateral sinus Bleeding was severe and was controlled by cautery and packing

The patient is 12 years postoperative and has no recurrence of the tumor Microscopic diagnosis was granulation tissue until the second case alerted the observers to the glomus jugulare diagnosis

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*Read before the Detroit Otolaryngological Society March 1959

†From the Otolaryngological Service Saint Luke s Hospital Cleveland O

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concomitant laryngeal neurofibroma, selective pituitary hypofunction and hypoadrenalism

3 Pathologic factors, diagnosis and treatment are discussed

ACKNOWLEDGEMENT

Our sincere thanks and appreciation to Dr James E Croushore for his aid in the preparation of this paper

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SIXTH INTERNATIONAL CONGRESS ON DISEASES OF THE CHEST

The Sixth International Congress on Diseases of the Chest will be held at the University of Vienna from August 29 to September 1, 1960

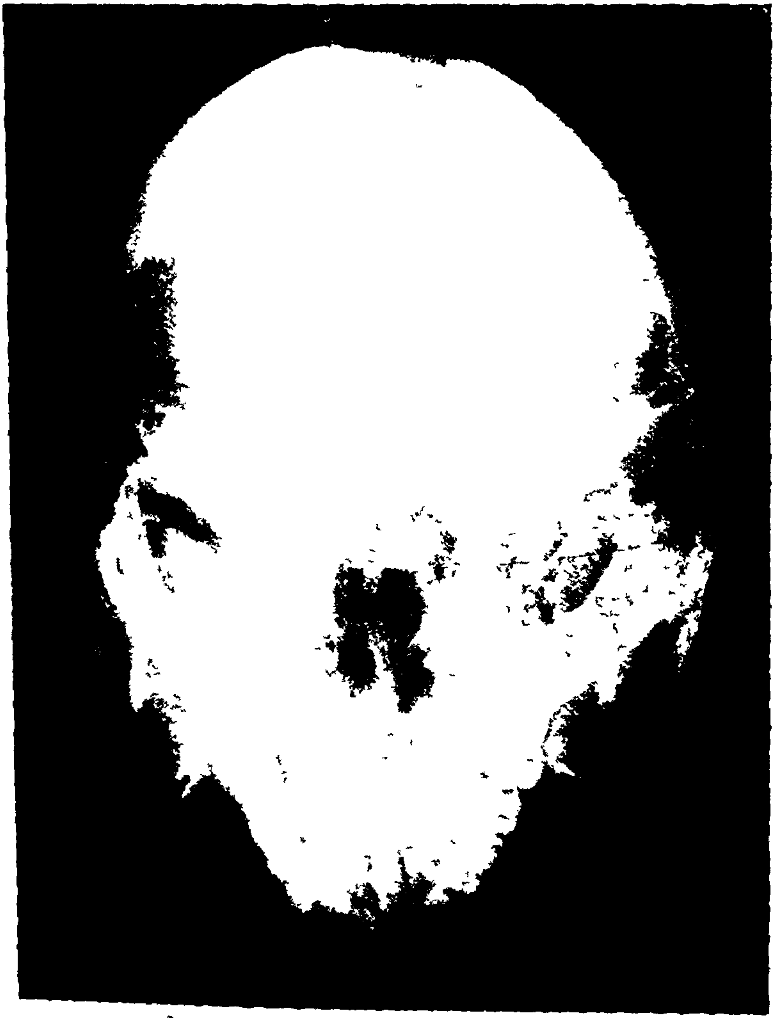

Fig 1 Radiogram showing destruction of the floor of the left posterior and lateral middle cranial fossa and base of the petrous portion of the temporal bone

mately three months Four days prior to the examination, the patient had a right myringotomy performed which resulted in considerable bleeding

The hearing on the right was 89 per cent of normal There was dry blood near the posterior aspect of the drum which was thickened and red X-rays were normal of the mastoid bone A biopsy, using the Rosen technique, diagnosed a glomus jugulare tumor Four days later a right complete radical mastoidectomy was performed for the tumor in the hypotympanic space and extending to the Eustachian tube

Case 2. This 46-year-old female was seen in 1950, complaining of intermittent but increasing cricket sounds in the right ear of six months' duration, and a loss of hearing on that side.

The only abnormal finding on complete visual otolaryngological examination was a complete paralysis of the right vocal cord. Pure tone air conduction audiometry showed that she had 40 per cent hearing in the involved ear, and bone conduction testing showed that this loss was mainly of the perceptive type. Caloric vestibular tests on this side showed that there was hypoactivity of this labyrinth. Mastoid and occipital view X-rays of the skull showed nothing abnormal.

It was advised that she be hospitalized for further study but she chose to go to a large clinic in another state. Here a diagnosis of probable intramedullar lesion was made, and she was told that the operative mortality in such cases was over 90 per cent.

Two years later and with further hearing loss, headache, dizziness, nausea, and vomiting, the patient was admitted to our hospital. Neurological consultation was secured and a jugular foramen tumor diagnosed. A suboccipital craniotomy was performed, and a glomus jugulare tumor was removed from the right cerebellar pontine angle and jugular foramen. Postoperatively the course was satisfactory, except that one year later a redness of the lower aspect of the right drum developed which was typical of a glomus jugulare. The ear was treated by irradiation.

This patient is seven years postoperative and has residual neurological changes as well as evidence of tumor in the middle ear.

Case 3. This 60-year-old white female was seen in 1950 with a chief complaint of left earache and discharge. From about the age of 33 there had been a hearing loss on that side. A growth was found in the ear two years prior to the admission examination, and for the recent three weeks there was constant aching and discharge.

Auditory acuity was 35 per cent of normal. The ear canal was completely filled with a large granular and necrotic polyp surrounded by a mucopurulent discharge.

The patient was admitted to the hospital and following removal of the polyp, the diagnosis of an ulcerated, polypoid adenoma of the ceruminous glands of the ear canal was returned. Postoperative visits revealed persisting tumor in the floor of the anterior wall of the ear canal, although the drum was normal except for an anterior inferior perforation. Ten months later a recurrent tumor of similar diagnosis was removed.

Two years thereafter there was a severe hemorrhage from a smooth polypoid tumor of the canal which clinically was diagnosed a glomus jugulare. A review of the previously biopsied material proved the diagnosis to be correct. In 1953 the patient was hospitalized, and a left open carotid arteriogram showed some filling of the tumor mass, following which the external carotid artery was doubly ligated. Four days thereafter a radical mastoidectomy was performed with moderate bleeding from the tumor extending posterior to the lateral sinus to the digastric groove, the posterior canal wall, the paralabyrinthine cells, and filling the middle ear. Approximately 98 per cent of the tumor was removed and the ear packed. Tissue examination substantiated the clinical diagnosis. The ear was irradiated postoperatively but eight months later required further similar treatment because of tumor recurrence.

The patient is now living and without recurrent tumor after six years.

Case 4. The chief complaint of this 39-year-old white female was continuous buzzing and decreased hearing in the right ear for approxi-

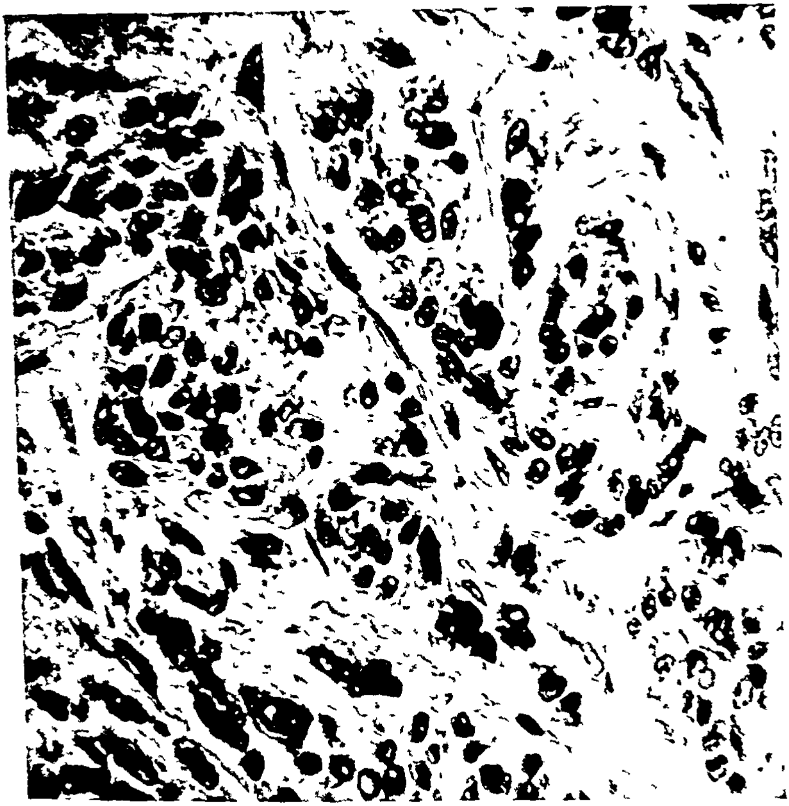

Fig 3 Photomicrograph of tumor composed of nests of compactly arranged cells in acinar formation in a richly vascularized stroma (H and E X500)

middle ear cavity The patient was hospitalized and although a left open carotid arteriogram failed to delineate the tumor the external carotid artery was doubly ligated The following day a tympano-attico apictomy was performed with little bleeding for a tumor in the mastoid cavity middle ear and Eustachian tube orifice Pathology reported a glomus jugulare

The patient is living without recurrence four years postoperative

Case 6 Of considerable interest is this 58 year-old white female seen in 1957 Eighteen months prior to the first visit a spontaneous left facial paralysis had developed followed in ten months by considerable intermittent pain in and about the left ear A hearing loss of two months was observed by the patient One month before the visit, the patient had severe pulsating pain in the ear and a left ear operation was performed in Europe

The first examination disclosed marked swelling of the post auricular incision and when cleared away a red granular friable bloody tissue was found X-ray of the chest was normal but the skull showed destruction of the floor of the left posterior and lateral middle cranial

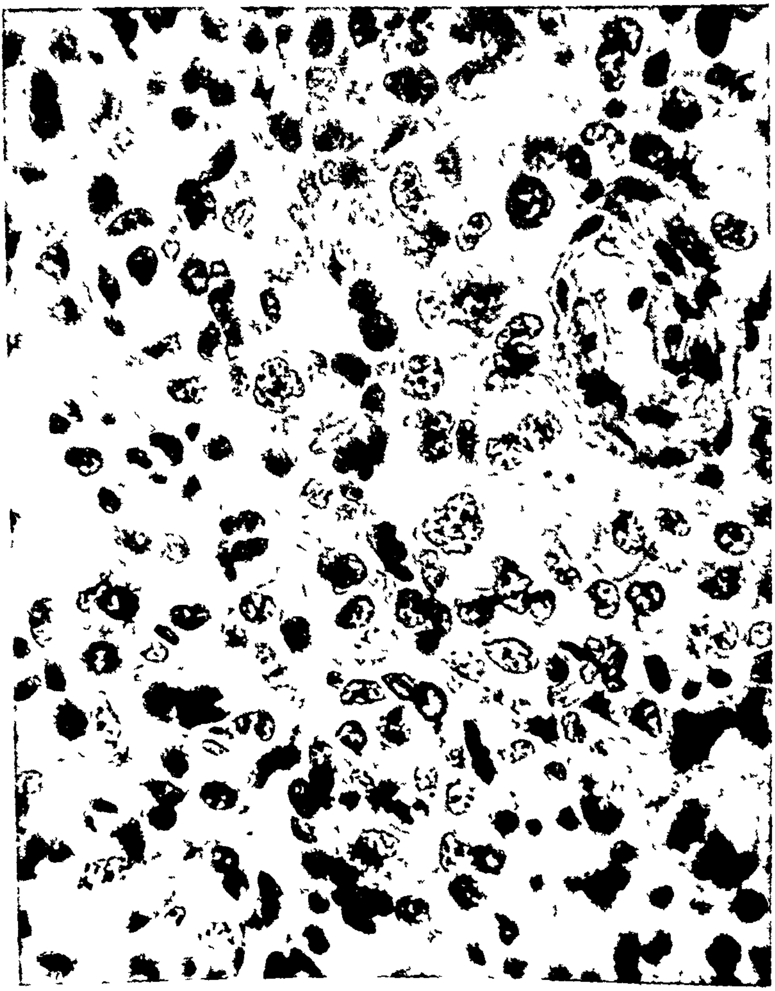

Fig 2 Photomicrograph of malignant tumor composed of lobulated sheets of irregular cells containing frequent mitotic figures with no attempt at differentiation (H and E X500)

The patient after four years is without evidence of recurrence

Case 5 This 48 year old white female complained of decreased hearing fullness and pulsation in the left ear when first seen in 1955 Nine years prior the patient was treated for tinnitus on the left and three years prior, a biopsy of a growth coming out of the drum was reported as granulation tissue

The left audiogram disclosed the level to be 56 per cent of normal Two red pulsating masses were found protruding from the posterior junction of the drum and canal wall Temporal bone X rays were positive for areas of cellular destruction near the antral portion adjacent to the

Several cases presented the difficulties encountered in the microscopic diagnosis of this tumor The first, removed early in the period of recognition of this entity, was diagnosed an endothelioma until the second case altered the observer's opinion Another case biopsied was reported as a granuloma until the entire tumor was removed and examined, revealing a deep glomus jugulare tumor In the two cases in which the external carotid artery was ligated, the bleeding was presumed to be reduced by the procedure

These cases present the problems encountered in early diagnosis, as well as the importance of prompt and adequate treatment, careful follow-up and the recognition of the malignant potential of this tumor When adequately studied, diagnosed, and treated, we believe the prognosis for these patients is good

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10515 Carnegie Ave

THE UNIVERSITY OF MICHIGAN MEDICAL CENTER

The annual Otolaryngology Conference at The University of Michigan will be held April 21, 22, and 23, 1960, under the direction of Dr James H Maxwell, Chairman of the Department of Otolaryngology

Applications may be addressed to the Department of Postgraduate Medicine, University Hospital, Ann Arbor, Mich fossae, as well as the base of the petrous portion of the temporal bone and mastoid process (see Fig 1) A biopsy returned as undifferentiated malignant tumor cells of the ear canal

A left post-auricular exploration of the mastoid area was performed, and a partial removal of the tumor mass accomplished The entire mastoid cavity was filled with tumor which had eroded the tip and lateral sinus The area was packed and closed Microscopically the tumor consisted of lobulated sheets of cells approximating acinar formation Numerous mitotic figures were present (see Fig 2) The diagnosis was glomus jugulare tumor, malignant, of the left mastoid region The tumor was a sharp comparison to the benign character usually seen (see Fig 3) Irradiation therapy was begun, but the tumor continued to increase in size despite therapy

The patient expired in 1958, 11 months after surgery, with symptoms suggesting rapid intracranial invasion This occurred in northern Vermont and no autopsy was obtained

Case 7 This 65 year old white female presented herself with a pain and pressure in the right ear of two months' duration Eight years previously, a sudden dizzy spell and hearing loss developed and she was informed that a blood vessel had broken in the right ear

The right ear hearing was 20 per cent of normal A partially bleeding, fibrotic tumor was found filling the entire canal to the external meatus A severe hypertension was present

Biopsy of the mass was reported as a granuloma pyogenicum and the following day the entire tumor was snared from the canal This specimen was reported as a glomus jugulare tumor, and the patient was treated by irradiation rather than surgery, due to her poor physical condition

Two years since treatment with over 5,000 r there is beginning regrowth of the tumor

These seven female patients varied in age from 39 to 65 years, with an average age of 51 years This corresponds well with the incidence of this tumor in the young and middle aged adults A pulsating tinnitus and an early conductive hearing loss were the most frequent complaints Other symptoms included otorrhea, bleeding from the ear, facial paresis, vertigo, inner ear deafness, and headache Symptoms were present from three months to 17 years prior to the diagnosis of the tumor

All cases except Case 2 presented a tumor mass in the auditory canal or behind the tympanic membrane In these cases the Brown test proved of considerable value in the clinical diagnosis prior to biopsy In one case, not here documented, the tumor failed to blanch on positive pressure with the Siegel otoscope, and no increase in pulsation or redness was noted with negative pressure Subsequent biopsy diagnosed a squamous cell carcinoma

It is the purpose of this report to describe generally the time and frequency performances of six superior esophageal speakers utilizing speech samples of adequate length for which norms for rate, breathing and frequency are already available for normal or superior speakers

SUBJECTS

Superior esophageal speakers were selected so that any generalizations might serve as suitable and attainable goals for those learning to speak without a larynx An initial group of 52 satisfactory performers from Southern California was obtained through contact with otolaryngologists, speech therapists, the Veterans Administration, The American Cancer Society, The Lost Chord Club of Los Angeles, and the New Voice Club of San Diego The senior author screened this group successively to 23 and ten Eight skilled and experienced judges selected six of the ten performances as best on a rating scale of general effectiveness The Fairbanks "Rainbow Passage"[2] was recorded for each performer, "as though speaking to an audience of 25 people"

All of the selected speakers were males with at least four years of experience with esophageal speaking Three of the speakers were experienced teachers of esophageal speech There were excellent female speakers in the original group, but none appeared to match the performance of the male subjects who were finally selected Six judges were requested to select female voices from the male voices on the basis of recordings alone The complete lack of success on the part of the judges tends to indicate marked similarity in the voices of male and female esophageal speakers

Each of the six speakers performed at the highest level of esophageal speech, as defined by Wepman, MacGahan, Rickard and Shelton[5], in other words, they spoke with continuity and without consciousness of the act of swallowing and eruction The speech was easily and naturally produced without apparent thought or effort

RESULTS

The answers to our four basic questions can be presented

HOW EFFECTIVELY CAN THE LARYNGECTOMEE EXPECT TO SPEAK?

Norms for Effective Esophageal Speech

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and

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The present sources of materials related to esophageal speech have little to offer in the way of direct information on how effectively the laryngectomee may reasonably expect to speak The typical laryngectomee is concerned with such vital questions regarding his speech performance as the following

1 How many words and syllables can be spoken per charge of swallowed air?

2 How long does it take to swallow a charge of air?

3 What rate in words per minute can be achieved relative to normal speakers?

4 What possibilities of pitch level and pitch variety can be anticipated for esophageal speakers?

The word *frequency* will hereafter be used in place of *pitch* because we measure frequency, which in turn is perceived as pitch By way of example, we hear middle C and the octave below it as perceived pitches, but these are represented physically as 256 cycles per second and 128 cycles per second

The laryngectomee would be benefited by some practical normative statistical information about the possibilities he could reasonably expect to achieve in successful esophageal speaking

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2. How long does it take to swallow a charge of air? Esophageal speech, despite frequent air charges, is relatively efficient and pleasant to hear. In order to understand how this can be true, we need to know that the skilled esophageal speakers gulped air in from .42 to .80 seconds, mean values considered. This is unbelievably rapid "breathing," as rapid in fact as most normal speakers breathe for speech. It should be pointed out that superior speakers usually breathe only at phrase limits. The esophageal speaker utilized his air charge for approximately one and one-half seconds, during which time he averaged 2.8 to 6.3 words, and so it is obvious that he needed air charges not only at phrase limits, but also many times in between. This is contrasted with the normal speakers who utilized their breath for 4.19 seconds and spoke 12.5 words. If relative time or words per air charge is taken as a measure of efficiency, the normal speaker was from 2½ to 3 times more efficient than the esophageal speaker.

3. What rate in words per minute can be achieved relative to normal speakers? It is probably most reasonable to combine the factors of Questions *1* and *2*, and to use total words per minute as a measure of efficiency. When this was done, the average for the superior esophageal speakers was 113 words per minute, compared with a rate of 166 words per minute[1] for the average normal speaker for the same reading material. Considering this measure, the normal speaker spoke only 1.47 times the rate of the esophageal speaker. Stated another way, the esophageal speaker was 80 per cent as rapid as the average normal speaker. Although the esophageal speaker would be judged as "too slow" compared to normal speakers, this is of little consequence if his efficiency is relatively satisfactory. The superior esophageal speakers did perform efficiently with the 300-word speech samples in the present study.

Preliminary study indicated that the skilled esophageal speakers could not speed up their rate without sacrificing either loudness or suitable phrasing. Even with the superior speaker the ability to modify rate is severely limited by the need for frequent air charges.

4. What possibilities of frequency level and frequency varifrom the data obtained with the six selected speakers. Because of the brief nature of this report, the sensitive electropneumography, and phonophotographic techniques have not been described in detail.

1. How many words (or syllables) may the laryngectomee expect to speak on each charge of swallowed air? The answer to this question must be interpreted in the light of one feature of the particular experimental situation involved in this present study. The superior esophageal speakers in this study were held to a loudness level suitable for an audience of 25 people. A constant check of sound pressure levels indicated that the speakers did maintain this level. A lowered intensity might well have increased somewhat the number of syllables spoken. The experimental study indicated that without exception these superior speakers took small charges of air efficiently and unconsciously. It should be noted that the normal superior speakers while reading the same "Rainbow Passage," did not begin to produce the syllables per breath claimed by some esophageal speakers.

Our study indicates the mean words per air charge range from 2.8 to 6.3 for esophageal speakers; this should be compared with 12.5 mean words per breath for normal breathing superior speakers. The extreme range for one esophageal speaker was 15 words (22 syllables), contrast with this one exceptionally superior normal speaker who, under identical conditions, spoke 36 words (50 syllables). The normal speaker read aloud with an average of 12.54 words per breath. This speaker had a wide range of performance and tended to utilize breathing phrases of 7 and 14 words. On the other hand, the esophageal speaker averaged 4.98 words per air charge and had a much narrower range of performance.

The syllabic values found in this study are somewhat higher than those found by Stetson,[1] who stated a limitation of from three to four syllables. The lowest average value in the present study was 3.8 syllables and the highest 8.7 syllables per air charge. These values, even when highly efficient performances are considered, are much more conservative than some information given recent wide publicity.[3]

no more conscious of the "breathing" function than were normal speakers

2 The speakers of this study averaged 5 words (4 98) per air charge and performances averaged about 120 words per minute A top limit of 11 to 12 syllables was very satisfactory, however, one speaker did achieve an extreme range of 22 syllables

3 The air charge was gulped in about one-half second, a more rapid performance than that of some normal speakers inhaling for an intra-phrasal breath pause

4 The mean frequency (pitch) level is almost exactly one full octave below that of the normal male speaker

5 Contrary to some reports, frequency (pitch) variability is quite comparable to that of normal speech

6 It is, however, very doubtful that the average frequency level can be raised much, if any, above 66-70 cycles per second

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THIRTY-THIRD ANNUAL SPRING CONGRESS OF GILL MEMORIAL EYE, EAR AND THROAT HOSPITAL

The Gill Memorial Eye, Ear and Throat Hospital announces to the profession its Thirty-third Annual Spring Congress in Ophthalmology and Otolaryngology, April 4 through April 9, 1960 A formidable list of speakers will participate

For further information write Superintendent, P. O Box 1789, Roanoke, Va ability can be anticipated for esophageal speakers? The results of the experimental frequency analysis indicated that the superior esophageal group (for both the mean and median frequency) was essentially one whole octave lower than the comparative group of superior normal speakers. This, in fact, represents a very low pitch and may be identified to the ear by striking the second C below middle C on the piano, however, despite the difference in frequency level the distribution of frequency usage showed greater variability for the esophageal speakers than for the normal speakers. The standard deviation values were 7.0 and 4.3 tones per second. Four of the esophageal speakers used the highest frequency which was lower than the median frequency used by the lowest of the superior normal group. The total range of frequencies was 13.2 tones for the superior esophageal speakers compared with 10.5 tones for the superior normal group.

This striking and unexpected finding clearly negates any assumption that the esophageal speaker must be monotonous in pitch. In fact, it supports the contention that esophageal speakers can learn to sing simple tunes at a slow pace. The wide range of the values in this study possibly could be attributed to a much longer reading selection and some improvement in the experimental equipment. It should also be pointed out that these frequency variations are taking place at a very low level in the frequency range.

CONCLUSIONS

The authors feel that this study exploring the rate and frequency characteristics of superior esophageal speech may be useful in establishing realistic norms for those who must learn to speak in this unique way. This study has utilized much longer samples of speech than previous studies, and it is, therefore, believed that the results are highly reliable for these carefully selected superior speakers. All in all, the results are rather conservative, being much more optimistic for reasonably effective speech than earlier studies based on small samples, but far less optimistic than predictions for success in recent popular literature.

1. The esophageal speakers spoke relatively easily and were

of the nose is very important Sometimes a diagnosis is obvious, the nose is externally deviated or depressed One very important point to remember, however, is that edema, especially in children, sets in very early and may fill in any deformity This edema will result in the nose s being smoothly swollen when, in fact, it may be depressed; thus, a false picture may be obtained In adults this edema forms later and may be less marked

The bridge of the nose and bony vaults are next palpated for crepitus One must not make the all too frequent mistake of palpating too vigorously, and causing further damage to the already splintered bones One must also bear in mind that crepitus is not too common in fractured noses, as the fragments quickly become well splinted due to the early edema formation, therefore, a fracture may be present even though there is no palpable crepitation

After the outside of the nose is examined, the inside is anesthetized and shrunken with 4 per cent cocaine packs If the mucous membrane will not shrink down one must ascertain whether the swelling is from the medial or lateral nasal wall If from the medial wall, deviation of the septum and/or submucosal hemorrhage with hematoma must be considered It must be emphasized that a hematoma of the septum has normal mucosal coloring and is not ecchymotic Lateral swelling may be from buckling of the lateral cartilages and/or subchondral hemorrhage

If the mucous membrane does shrink well, tears in the mucous membrane are looked for to show the site of the fracture or displacement of the septum, lateral cartilage or bony walls

Finally X-ray studies are done I strongly believe that entirely too much emphasis is placed on an X-ray report of "no fracture seen" Too often a patient will be told that he does not have a fractured nose because an X-ray did not reveal it, and several years later he will have to have a rhinoplasty or submucous resection, due to resultant deformity. Reports in the literature[1 3] state that on the average only 50 per cent of nasal fractures are seen by X-ray

THE IMPORTANCE OF NASAL FRACTURES

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The importance of nasal fractures lies in the fact that they are usually thought of as an insignificant type of fracture, and as such management is commonly on a "hit or miss" basis, yet, when one realizes that any imperfection in realignment of the nose can be very noticeable cosmetically, then it is understandable why a little more thought should be put into this subject

If the history of a patient who presents himself for rhinoplasty is gone into, it is my impression, based on 50 such patients, that fully one-third of them will give a history of previous traumatic injury to the nose If patients could remember all the traumatic events in their lives, especially in early childhood, probably at least one-half of these patients would give such a history

PRESENT DAY MANAGEMENT OF FRACTURED NASAL BONES

At the present time the treatment of nasal fracture is not stressed very much in residency training programs The usual routine consists of taking X-rays, inserting an elevator into one nares and elevating the nasal bones into a supposedly correct position Asch forceps are also being used Splinting, consisting of adhesive tape or Wood's metal over dental compound, usually is next applied As will be mentioned later, there is a very small percentage of nasal fractures that should be reduced openly for better results

METHODS OF MAKING AN EARLY DIAGNOSIS

One of our first problems is to make an early and correct diagnosis before attempting treatment If the patient is seen early before edema has developed, inspection of the outside

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A Simple Fractures—

1. Green stick—14 patients
2. Linear without displacement—21 patients
3. Linear with either lateral displacement or depression—37 patients

B Comminuted Open Fractures—28 patients

The ages of the patients ranged from 3 years to 74 years, however, if we divided nasal fractures into adults and children, using age 16 as a dividing line, we find that 39 of the 100 patients were children Children had 12 of the greenstick fractures, one of the linear fractures without displacement, 16 of the 37 displaced or depressed fractures, and 11 of the 28 comminuted open fractures

The most common type of nasal fracture in adults is the one with lateral displacement or depression

RESULTS OF TREATMENT

To evaluate the results of treatment, the author compared the pre-treatment X-rays and photographs of patients with the post-treatment X-rays and personal clinical examination A result was considered good if the author could find no post-treatment clinical or X-ray evidence of external or internal nasal deformity A result was considered improved if the position of the displaced or depressed bone or cartilage on post-treatment X-rays had changed from that recorded on pre-treatment X-rays, but deformity was still present on clinical examination In other words, an improved result may still have a resulting deformity, only this deformity is less than would have been if not treated A result was considered poor if the alignment of the fractured bone or septum had not changed in position when the pre- and post-treatment X-rays were compared, and also pre-accident photogrophs were compared with the present clinical condition

The results of the treatment were as follows

A 14 Greenstick fractures—100 per cent good results

At the Massachusetts Eye and Ear Infirmary we are fortunate, indeed, to have a radiologist having 36 years of experience in eye, ear, nose and throat X-ray During this time he has developed a technique that I am sure gives much better than average results His technique consists of three films *1* lateral view with good soft tissue contrast to show either depression of the dorsum of the nose, nasal process of the frontal bone, plus any displacement of the bony arch from before backwards, *2* an exaggerated Water's view to show lateral displacement of the bony arch, *3* his own special view consisting of a dental film inside the mouth with the X-ray beam tilting down from the top of the head in such a way as to miss both the frontal bone and the teeth, but to include the bony nasal arch, and thus pick up the displacement He feels that with this technique most bony fractures are seen[10]

CLASSIFICATION OF NASAL FRACTURES

For the purpose of analysis, the 100 cases of nasal fractures studied by the author at the Massachusetts Eye and Ear Infirmary have been classified into simple and comminuted, with subdivisions modified after Becker[1]

A green stick fracture was one in which there was a bending of the nasal bones and septum, but no actual true fracture

A linear fracture without displacement was a simple fracture that was in good position and could be treated successfully with little skill and care Treatment in all cases consisted of ice and pain medication If either lateral displacement or depression was present, treatment consisted of blunt elevation, with or without splinting and/or intranasal vaseline gauze packing

A comminuted open fracture was one in which the nasal bone was shattered into multiple pieces and there was a mucosal tear exposing bone Treatment was the same as for depressed or displaced lateral fractures

Using the above classification, the 100 cases could be broken down as follows

POSSIBLE FACTORS CONTRIBUTING TO POOR RESULTS

It is my own personal feeling based on the 21 cases in which I did the initial treatment, that one of the most important factors contributing to poor results is that posttraumatic swelling, by filling in the depressions, may obscure a deformity that has not been corrected, also, while the nose may be reduced to the correct position, if it is not held in this position by splinting there is a tendency for it to slip back or fall down again Forman's comparison of the architecture of the nasal pyramid to an arch is a very good one, for when the lateral buttresses and abutmens are broken, the keystone and side walls of the arch will collapse The same thing will happen with a fractured nose The nasal bones and frontal process may be reduced to perfect position, but if there is no provision made to give support artificially either directly in the form of an uplift, as with intranasal packing or indirectly in the form of lateral support to the buttresses or ascending processes in the form of external nasal splinting, or both, then the nasal pyramid may collapse or be in imperfect position

The results at the Massachusetts Eye and Ear Infirmary bear out this fact In the type of fracture where support by splinting is needed and was not given, we have very poor results In those cases where we did not need splinting, we obtained very good results

Another factor contributing to some of the poor results was the fact that Asch or Walsham forceps were not available during this two year period for those cases in which there was locking of displaced fragments If one uses merely a blunt elevator, it is very difficult at times to replace perfectly the septum into the groove of the vomer, to unlock a displaced and depressed ascending process, or to correct overriding nasal bones upon the maxilla

A third reason for our own poor results, in the author's opinion, is that no attention has been paid to damage to the upper and lower lateral cartilages, the osseous vault and sometimes the septum receiving all the attention It should be emphasized that, if a hematoma forms around these cartil-

B 21 Linear fractures without displacement—100 per cent good results

C 37 Linear fractures with

1. Displacement—40 per cent good results, 35 per cent improved results, 25 per cent poor results
2. Depression—20 per cent good results, 25 per cent improved results,, 45 per cent poor results

D 28 Comminuted open fractures—10 per cent good results, 20 per cent improved results, 70 per cent poor results

ANALYSIS OF 100 CASES

During the two years preceding December 31, 1957, there were 162 cases of nasal fractures treated by residents of the Massachusetts Eye and Ear Infirmary The author personally examined 100 of these patients at least two months' post-reduction, and he did the initial fracture reduction in 21 cases The 79 remaining cases were treated by seven other residents The time between the accident and the initial treatment varied from one hour to 52 hours In the majority of patients, the treatment consisted of elevating the depressed nasal bones with a blunt elevator and replacing the displacement by digital pressure toward the midline Asch reduction forceps were available only during the last three months of the study (16 cases) and external and internal splinting was used on only 20 cases

It is interesting to note that a few of the patients were told that their fracture was "reduced the best that could be done without doing plastic surgery"

For the purpose of analysis, the 62 cases on which we have no follow-up, will be disregarded

We have no way of evaluating how the nose looked when the doctors finished reducing the fracture, but, after all, the patient is not interested in this point, only the final result Accordingly, this study evaluated only results from two to 18 months after reduction

Using Asch forceps, the maxillary bones may have to be rotated outwards, after which the nasal bones or the naso-frontal process may be elevated easily Lateral displacement can next be corrected by inward pressure by Asch forceps on the maxillary bones at the base of the nose These procedures may or may not also correct the septum If not, one may either have to replace it in the groove of the vomer by Asch forceps, or, under direct vision, push the upper fragments into position, using a septal elevator through a mucosal tear After the bony and cartilaginous deformities are realigned, the nose should be packed internally along both sides of the septum either with half-inch vaseline gauze strips or half-inch gauze strips soaked with cod liver oil The cod liver oil reduces odor and also makes removal much easier by preventing adhesiveness to the nasal mucosa such as vaseline gauze tends to produce External splinting to prevent "widening" of the nose due to spreading of the ascending processes is next applied, using dental compound over the dorsum and also over the bony arch It is important not to get the compound too pliable by using water that is too hot and also to have the compound "set" quickly by applying ice cubes directly on the compound While "setting," constant digital pressure inward must be applied bilaterally in order that the compound will not relax and thus lose its splinting effect If we do not splint the fractured nasal arch, it may tend to revert to its displaced position, therefore, the corrected position should be maintained by internal packing for five to seven days, and by external splinting for about ten days

This brings us now to the very important subject of when to do an open reduction It is my own personal feeling that one should be as conservative as it is practically possible Naturally, all simple bony fractures can be treated by closed reduction, providing accurate diagnosis by inspection and X-ray is done beforehand If there are any tears in the mucosa, a small probe should be inserted and palpation should be carried out in order to ascertain whether there is any exposed bone If bone is palpated, then the original mucosal tear should be enlarged with a blade to obtain a good exposure, so that a precise reduction under direct vision can be accomplished Hematomas or effusions of the cartilaginous ages and is not corrected, it will lead to curling and fibrosis with resulting deformity

SUGGESTED METHOD OF TREATMENT

There is nothing new that can be added to the treatment of fractured nasal bones that has not been written in the literature many times by many authors [1,2,3,4,5] The prime requisite, however, is a working knowledge of the anatomy and relationships of the bony arch and cartilaginous structures, including the septum

Several points in the anatomy of the bony arch are important enough to be mentioned here First of all, the nasal part of the frontal bone extends downward behind and beneath the nasal bones and thus adds strength and support to the bony arch It follows that a fracture which involves the nasal portion of the frontal bone will require more uplift and support to prevent postreduction depression of the bridge than a fracture that does not While this type of fracture initially may be reduced quite easily, only with proper packing and splinting will postreduction depression be prevented

Second, the lateral cartilages extend upwards on the inner surface of the nasal bones and are intimately fused with them, the clinical significance of this being that the lateral cartilages participate in displacement of the nasal bones and are rarely detached from them during trauma, thus, any damage to them, such as lacerations or hematomas, must also be corrected by suturing, evacuation, etc

One must keep in mind also, that while the nasal bones heal by bony union without scar contraction, the nasal cartilages do not They heal by an ingrowth of connective tissue and not by cartilaginous union Cartilage may also curl and buckle from organization of inflammatory exudate, or chondritis may set in and undergo calcareous degeneration with resulting cosmetic deformity

After going through the previously outlined diagnostic steps, early evacuation of effusions and hematomas must be carried out to prevent cartilaginous damage All locked fragments should be unlocked before any elevation takes place

X-rays are used only to confirm the clinical diagnosis and as a permanent record of the case

Relatively uncomplicated fractures in children may be reduced in the emergency ward, and hospitalization is not necessary A short general anesthesia rather than local is preferred for children A thin layer of cotton moistened in 4 per cent cocaine and vaseline and wrapped around a periostial elevator is introduced into the nares beneath the depressed bone, and the nasal arch is elevated The displaced bone is then replaced to its original position by digital pressure I have found that it is necessary to maintain the corrected position by means of lateral splints as with adults, otherwise the bones tend to revert to their previously displaced position

SUMMARY

In 100 cases of nasal fractures seen at the Massachusetts Eye and Ear Infirmary, the post reduction cosmetic results were not so good when the fracture was of the comminuted open type as they were with greenstick, or linear fractures with or without displacement or depression The reason for this poor result is thought to be due to poor post-reduction splinting and packing The literature is reviewed partially and a composite method for diagnosis and treatment presented

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structures (septum or upper and lower lateral cartilages) must be evacuated by incision rather than needle aspiration, which will fail most of the time because partially clotted blood may be present that could block the aspirating needle Open reduction should be used if old associated fractures are present, or if any other previous defect, such as a hump that the patient may want corrected, is present

NASAL INJURIES IN CHILDREN

Children present a special problem as far as nasal injuries are concerned, because the anatomy of both the osseous and cartilaginous structures of the nose is different There is less ossification and calcification of the nasal, vomer, and ethmoid bones which usually results in a bending or greenstick fracture rather than a complete through-and-through break, therefore, the valuable sign of crepitation may not be present It follows, also, that less force is needed to produce the fracture in children The cartilaginous structures of the nose not only are softer and curl easier, but they seem to form more scar tissue leading to a deformity that increases as the child grows

When a child presents himself with a nasal injury, early reduction is very important because edema sets in very quickly and may obscure the picture by filling in the defects The nose then may have a falsely smooth appearance If edema is already present when the child first presents himself, it is very hard to determine the exact status of the injury without first decreasing the swelling I believe that it is a mistake to tell the parents to apply ice for three or four days while we wait for the swelling to go down Instead, inject the swollen tissue, using a 25 gauge needle, with 1 to 2 cc of hyaluronidase to reduce swelling After injection of the swollen tissue, the inside of the nose is sprayed with 1 per cent ephedrine sulfate, repeated three times, and then packed with long fiber cotton strips that have been dipped in the same solution and then wrung out After a wait of five minutes, the packs are removed, and inspection of the inside of the nose is carried out, looking for tears in the mucosa, open fractures, or dislocation of cartilaginous structures

In patients in whom the mastoid cavity heals, problems exist because of the presence of the mastoid bowl Infections of the lining of the cavity may result The patient should not get water in the mastoid cavity, since infection can occur from this This elminates swimming In children, especially, the care of a modified radical or radical mastoidectomy is a chore A child will not tolerate too much in the way of postoperative care Once the child has been subjected to a session of curettage, with its attendant pain and bleeding, he will rarely cooperate in further treatment

It, therefore, occurred to us that since the cavity tends to close, and since we have been trying to devise various techniques to make the cavity small or to obliterate the cavity, why should we tolerate the radical mastoid bowl at all? It is recommended, therefore, that rather than do an endaural or postauricular radical mastoidectomy, which leaves a wide open mastoid cavity exposed to the outer world, we should carry out a procedure which we call the anterior-posterior technique The posterior part refers to a classical postauricular mastoidectomy and the anterior to the exploration of the middle ear with an ear speculum and the operating microscope In this way, there would be no mastoid cavity to care for, and one would not worry about the problem of granulation tissue and atresia A number of patients have been operated upon in this manner with very good results It is for this reason, that we present the preliminary report on this technique No special claim is made for originality, except that we have combined techniques well known to us, in one operation, and thus avoid some of the distressing problems that occur in the classical endaural mastoid operations

The operation is performed under endotrachial general anesthesia The mastoid portion can be done first, or the middle ear portion first and the mastoidectomy last The patient is prepared in the usual manner The hair from the postauricular area is shaved for a small distance The usual post-auricular incision is made and the mastoid cortex exposed This procedure can be carried out, using the light from the draped Zeiss microscope for illumination and magnification and for completing the various steps of the operation The

ANTERIOR-POSTERIOR TECHNIQUE FOR THE TREATMENT OF CHRONIC OTITIS MEDIA AND MASTOIDITIS

Preliminary Report[*]

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The surgical treatment of chronic mastoiditis with cholesteatoma has been a great problem since the earliest days of otology Ever since the first operations were described, radical mastoid surgery has remained relatively unchanged In the postoperative period, our problems of healing begin The newly created radical mastoid cavity tends to fill with granulation tissue requiring constant curettage in an effort to keep this exuberant granulation tissue under control The granulation tissue often becomes infected Many methods have been tried to clear or control the granulation tissue in an attempt to promote healing These include X-ray therapy, the use of caustic solutions of silver nitrate and trichloracetic acid, curettage, and excision Eventually, a fibroma may form, which completely fills and occludes the mastoid cavity Atresia of the external auditory canal may accompany the filling of the mastoid cavity Secondary operations to excise the fibrous tissue often become necessary, and secondary skin grafting may be needed Numerous methods to control the size of the mastoid bowl and to hasten its healing have been suggested, including the use of fat grafts, cartilage grafts, bone grafts from the tibia, cancellous bone from the illium, muscle flaps, and plastic material of various sorts These agents were all tried in an attempt to control the size of the mastoid cavity During the 1930's an extensive bibliography was built up on methods of obliterating the mastoid cavity This contains articles too numerous to record

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[*] From the Department of Otology Temple University Medical School

Editor s Note This manuscript received at The Laryngoscope Office and accepted for publication Feb 15 1959

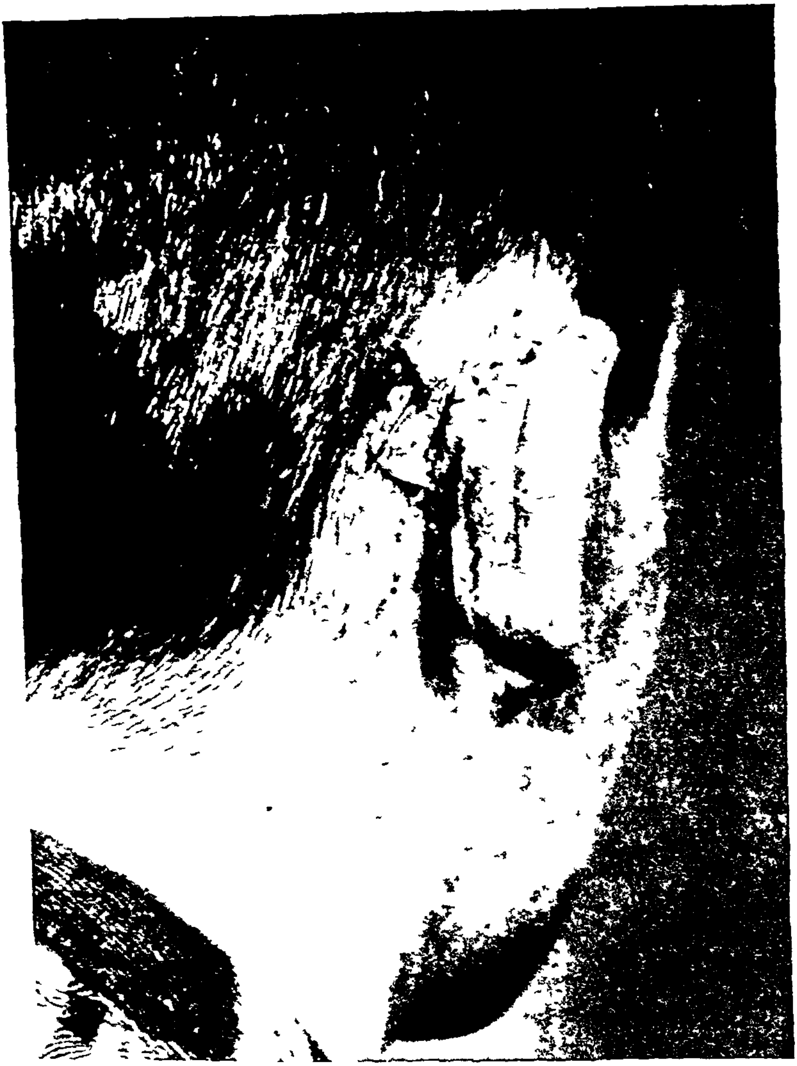

Fig 1 First postoperative dressing Note presence of plastic tube in upper angle of wound This allows a projection of the tube out of the dressing so that the nurses or residents can introduce appropriate antibiotic solution

can be cleansed of infection, granulation tissue, and cholesteatoma The tympanic perforation is handled by cleansing the edge of the perforation and removing the infected granulation tissue Any remaining polypi or granulation tissue can be removed with a small punch and curette Usually, there is a mastoidectomy is carried out with cutting burs and curettes When the mastoidectomy has been completed, the additus is widened and the epitympanic space exposed The incus and malleus are then skeletonized and cleansed of all diseased membrane Any cholesteatoma, granulation, and scar tissue is removed from the ossicles, the epitympanum, and some of the tympanic cavity below the incus When this portion of the operation is completed, the posterior canal wall is thinned but not removed A bridge of bone is allowed to remain over the auditory ossicles to protect the incus and malleus If the ossicles are diseased or partially necrotic, the incus and head of the malleus may be removed This portion of the operation is done under 16X magnification By this approach, the upper portion of the epitympanic recess well into the middle ear, can be explored A small No 190 plastic tube (see Fig 1) is put into the attic region and sutured to the upper angle of the wound A No 8 rubber catheter is placed in the lower portion of the wound and sutured to the lower end of the wound A small piece of tape is placed around the upper catheter so that it cannot slip into the mastoid cavity The post-auricular wound is then closed with interrupted silk sutures in the classical manner

The operation is then switched to the middle ear, and is done under 10X to 16X magnification The canal walls are injected, and through the ear speculum a Shea triangular incision is made This incision can be extended anteriorly, both superiorly and inferiorly to expose the entire hypotympanum The middle ear is then inspected Cholesteatoma matrix and granulation tissue is removed as well as the ossicles, should they be diseased Practically all of the middle ear can be visualized in this manner The stapes is exposed and freed of scar tissue and granulation tissue If the ossicular chain is intact, the drum is allowed to fall back in the usual manner If the ossicles have been removed, a small piece of No 90 plastic tubing is fitted as a couplet over the head of the stapes The posterior superior area of the middle ear cavity is also carefully cleansed, and all of the diseased tissue is removed far posteriorly and superiorly until we meet the place where the debridement was stopped on the mastoid side of the operation, thus, the entire attic and middle ear and manipulative procedures is cut to a minimum, which is pleasing to both patient and otologist

SUMMARY

A preliminary report of a surgical technique is presented We have called this the Anterior-Posterior Technique This includes performing a post-auricular complete mastoidectomy to eliminate the mastoid disease and an operation through an ear speculum and the operating microscope to eliminate middle ear pathology This technique offers rapid healing with a dry ear, without the disability of an open mastoid cavity, granulation tissue, drainage, and long protracted healing There is minimal postoperative care The mastoid cavity wants to heal and close, therefore, we let nature take over The middle ear can be handled by the approach through the ear speculum and with the use of the operating microscope This makes for rapid healing and is known to all otologists Variations of this technique can be applied to include myringoplasty and tympanoplasty, with grafting and plastic prosthesis A dry ear with an improvement in hearing results

Beury Building

THE MOUNT SINAI HOSPITAL

An intensive Postgraduate Course in Rhinoplasty, Reconstructive Surgery of the Nasal Septum and Otoplasty will be given July 16, 1960, through July 29, 1960, by Dr Irving B Goldman and staff at the Mount Sinai Hospital in affiliation with Columbia University

Candidates for the course should apply to Registrar for Postgraduate Medical Instruction, The Mount Sinai Hospital, 5th Avenue and 100th Street, New York 29, N Y fair amount of bleeding, when the incision is first made and the middle ear entered, but as the diseased membrane is removed, the bleeding stops This can be controlled by small gelfoam sponges soaked in epinephrine 1 1000 solution or in thrombin topical solution After this portion of the operation is completed, the drum and skin flap are eased back into place and held with small pieces of gelfoam packing

In the postoperative period, Neomycin or other appropriate antibiotic solution is introduced into the plastic tube in the upper angle of the wound This tube is allowed to project from the dressing The solution is put in three to four times daily while the patient is hospitalized The tube is allowed to remain in place for many weeks, if necessary, and can be covered with a small dressing The drainage tube, which is in the lower angle of the wound, is removed on the fifth to seventh day The tube in the upper angle of the wound is allowed to remain until the ear becomes dry, since this can be used to introduce antibiotic solution or to irrigate the middle ear The tube can be seen in the attic region when the drum is inspected through a speculum As the ear clears up, the perforation in the drum can be seen to close with a layer of scar tissue

This operation has been done on patients with cholesteatoma and attic perforations The perforation is not closed, but when the middle ear becomes dry, it usually seals and heals itself with the formation of scar tissue When the perforation heals, if there is an intact ossicular chain, the hearing is usually at a high level If the ossicles have been removed and a plastic tube is placed on the stapes, a myringostapedopexy is produced and a good hearing level results This same operation can be carried out when a larger perforation is present or even when more of the middle ear has been destroyed The operation can be completed by use of a skin graft or myringoplasty over the promontory, or a myringoplasty-tympanoplasty can be done at a later date when the ear is dry

In our patients, healing was very rapid and occurred in from two to four weeks The number of postoperative visits occurred over the distribution of the auriculo-temporal nerve. The condition has often been called Frey's syndrome or the auriculo-temporal syndrome. In 1932, two patients with this syndrome were reported by Peter Bassoe[5] of Chicago. One patient had a mixed tumor removed, and the other followed drainage of surgical parotitis.

In 1948 Freedberg, et al.,[6] collected only 35 cases from the literature; however, due to the great increase in the number of parotid operations being done, many more cases have been reported in the past few years.

Recently, interest in the subject has been renewed by Laage-Hellman of Gothenberg, Sweden, and by Glaister, et al., of Middlesex Hospital, London.

Laage-Hellman[7] investigated 123 patients who had undergone conservative parotidectomy. He employed the Minor Starch-Iodine test to study sweating. The face and upper neck are painted with a solution of iodine, castor oil and alcohol, and starch powder is sprayed thinly over the area. The patient sucks on a lemon slice. Each functioning sweat gland is seen as a blue spot which may coalesce (see Figs. 1 and 2). In response to a direct inquiry, 76 of the 132 (62 per cent) patients replied that they had symptoms on eating. Twenty-seven reported both sweating and flushing, 32 had sweating only, and 17 had only flushing; however, when he examined the patients and used the starch iodine test, he could demonstrate gustatory sweating in all 123 cases. Ten showed sweating alone, and three showed flushing alone. The sweating was most frequently found over the distribution of both the auriculo-temporal and the great auricular nerves. Sometimes only the auriculo-temporal was involved and rarely only the great auricular. In only 16 cases (13 per cent) were symptoms severe enough to embarrass the patient. He concludes that the condition is an invariable sequel of surgery about the parotid, and that only occasionally are symptoms severe enough to disturb the patient sufficiently to require therapy. He prefers to call the syndrome Gustatory Sweating and Flushing (G.S.F.).

In 1958 Laage-Hellman[8] found an interval of five weeks to

GUSTATORY SWEATING AND FLUSHING[*]

The Auriculo-Temporal Syndrome—Frey's Syndrome

WILLIAM GARTH HEMENWAY,

Chicago, Ill

That sweating and flushing of the cheek may occur after parotid surgery, injury, or infection has been recognized for many years As the phenomenon has not been reported in recent American literature it appears that many physicians are unaware of the condition Patients presenting these symptoms may be misdiagnosed, subjected to needless investigation and consultation, and labelled as psychoneurotic As the frequency of parotid operations has increased during the last decade more cases are being seen

A few weeks or months after an injury, operation or infection in the region of the parotid gland, the patient may notice that an area on the side of his face anterior to the ear begins to flush and sweat when he eats Certain foods, such as lemon and apples are more prone to elicit the symptoms than others Either sweating or flushing may come on first, and occasionally one may be present without the other Symptoms vary considerably in severity, and the condition persists indefinitely

This paper will review some of the literature, present two recent cases and suggest a possible satisfactory treatment

Duphenix[1] is credited with reporting the first case in 1757 Symptoms developed following drainage of a parotid abscess In 1853 Baillarger[2] reported a bilateral case after parotid infection Weber[3] described a case following suppuration of the parotid lymph nodes Lucie Frey,[4] of Poland, reported a case in 1923 The patient had sustained a bullet wound in the parotid area and later developed typhus and typhoid fever She was the first to observe that the sweating and flushing

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[*] From the Section of Otolaryngology of The University of Chicago

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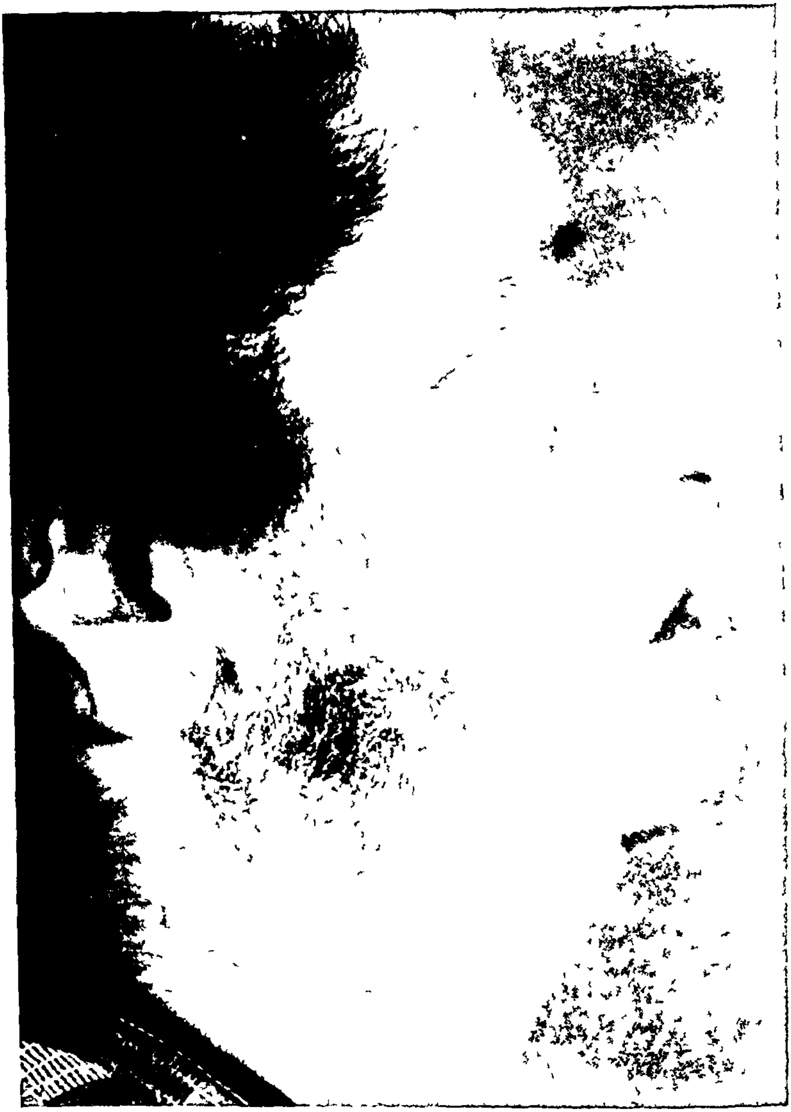

Fig 1 Case 1 Sweating over the distribution of the auriculo-temporal and great auricular nerves when the patient is chewing a lemon The skin has been painted with iodine solution and sprayed with starch

one year between surgery and the onset of symptoms in 58 cases He points out that this latent period strongly favors the theory of misdirected parasympathic nerve regeneration as an explanation for the sweating and flushing

Treatment is discussed by Laage-Hellman[6] in another recent publication Cutting the auriculo-temporal would abolish the condition, but this is difficult to perform in an operated field and may be followed by regeneration and recurrence The otic ganglion is inaccessible to surgery Injection with alcohol would result in mandibular nerve anesthesia for several

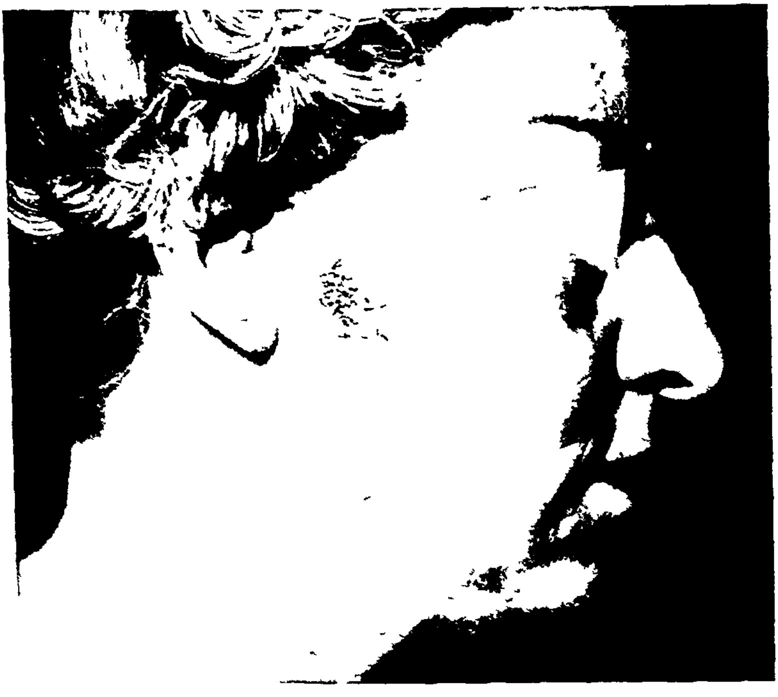

Fig 2 Case 2 Sweating over the distribution of the auriculo-temporal nerve This was abolished by injecting the otic ganglion with local anesthesia

months, which would probably be more uncomfortable than G S F Intracranial division of the glossopharyngeal nerve has been effective but is too extensive a procedure for such a benign condition X-ray therapy is effective in dosages sufficient to destroy the sweat glands This takes about 5000 r which seems too much X-ray to use on a benign process Systemic anticholinergic drugs have not proven practical, due to severe systemic effects He recommends 3 per cent Scopolamine Hydrobromide cream applied locally to the involved area every few days. This satisfactorily controls symptoms in most patients.

Glaister, Hearnshaw, Heffron Pick and Patey[10] state that sweating is the important feature which may or may not be accompanied by flushing. Two patients with the syndrome were very thoroughly investigated. Sweating was observed and recorded by a method described by Thomson and Sugarman.[11] Anesthesia of the mouth reduced but did not abolish the sweating. Local injection of atropine prevented the sweating from appearing. Blocking the auriculo-temporal nerve with a local anesthetic stopped the sweating as did injection of the otic ganglion. No effect was obtained after blocking the sympathetic at the superior cervical ganglion. The authors conclude that the only satisfactory explanation for the phenomenon is aberrant parasympathic nerve regeneration.

CASE REPORTS

Case 1. J. Z., 651219. In April, 1956, this 41 year old white female had undergone a subtotal parotidectomy for a mixed tumor of the right parotid. The postoperative period was uneventful. Eighteen months later she stated that her right cheek began to flush and "the gland began to leak" when she was eating. Examination revealed a well healed incision, normal facial movements and no evidence of recurrent tumor. Upon eating an apple typical flushing and profuse sweating were observed. The Starch iodine test (see Fig. 1) showed sweating distributed over the area of skin supplied by the auriculo temporal and great auricular nerves. On learning that the condition was not serious the patient did not want any treatment.

Case 2. A. S., 708755. This 41 year old housewife was seen at the University of Chicago Clinic on July 15, 1958, by Dr. Dwight Clark. Two and one half years previously she had undergone subtotal parotidectomy elsewhere for a chronic parotid infection. Since the operation she complained of severe intermittent pain and recurrent swelling about the parotid area. Sometime later she noted sweating and flushing of the right cheek. A diagnosis of Frey's syndrome was made and the patient was admitted to the hospital for study. The Starch iodine test (see Fig. 2) revealed gustatory sweating over the distribution of the auriculo temporal nerve and flushing of the skin was noted over the same area. A neurological consultant found no organic basis for the pain and considered it to be functional. There was no evidence of swelling about the operative site at any time while she was under our observation. Novocaine injection of the right otic ganglion by Dr. S. Mullan temporarily abolished the phenomenon. Sectioning of Jacobson's nerve in the middle ear was advised but was refused by the patient.

DISCUSSION

Recent studies have confirmed the thesis that the sweating and flushing were due to aberrant regeneration of parasympathic nerve fibers which were interrupted at surgery Parasympathic secretory fibers to the parotid gland[12,13,14] originate in the inferior salivary nucleus in the medulla They leave the brain with the glossopharyngeal nerve and enter the middle ear as Jacobson's nerve, which emerges on the roof of the petrous pyramid as the lesser superficial petrosal nerve They enter the infratemporal fossa via the cannaliculus innominatus or the foramen ovale The fibers synapse with ganglion cells in the otic ganglion which is just below the base of the skull, medial to the mandibular nerve The post ganglionic fibers reach the parotid via the auriculo-temporal nerve, which also contains sensory fibers to the skin and sympathic fibers to the sweat glands and blood vessels of the skin

After the auriculo-temporal nerve has been cut the parasympathic fibers regenerate along sympathetic pathways in the auriculo-temporal nerve and sometimes in the great auricular nerve After a latent period of between five weeks and one year the normally sympathetic innervated sweat glands and blood vessels of the skin acquire a parasympathic nerve supply Thus, when the patient eats he gets a reflex flushing and sweating instead of a reflex stimulation of parotid salivary flow

Most cases are very mild and require no therapy If symptoms are severe enough to warrant treatment, 3 per cent Scopolamine cream applied locally as recommended by Laage-Hellman[9] seems to be effective I would suggest sectioning Jacobson's nerve in the middle ear as a safe effective treatment The operation was described by Lempert[15] several years ago as a treatment for tinnitus aurium Plugging the bony canal from which the nerve emerges would effectively prevent regeneration, although preganglionic parasympathic fibers do not regenerate as easily as post ganglionic fibers Experiments are now underway in our laboratories to determine the effect of sectioning Jacobson's nerve[16] on parotid secretion

SUMMARY

Recent excellent work on Gustatory Sweating and Flushing is reviewed

Two illustrative cases after subtotal parotidectomy are presented Sectioning Jacobson's nerve on the promontory is feasible and should give permanent relief

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SCIENTIFIC PROGRAM OF THE AMERICAN OTOLOGICAL SOCIETY, INC

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Early Embryology of the Auditory Ossicles and Associated Structures in Relation to Certain Anomalies Observed Clinically

Barry J Anson, Ph D (Med Sc) ,
Jerome S Hanson, M S (by invitation), and
Shatik F Richany, Ph D (by invitation)

Tissue Reaction Following Reconstruction of the Oval Window in Experimental Animals

Richard J Bellucci, M.D , and
Dorothy Wolff, Ph D , (by invitation)

Human Cochlear Responses to Sound Stimuli

John L Bordley, M D.,
Robert J Ruben, A B (by invitation), and
George R Nager, M D (by invitation)

Some Physiological Factors in Inner Ear Deafness

Merle Lawrence, Ph D

Distribution of Hearing Loss in Various Population Centers

Aram Glorig, M D

The Management of Middle Ear Lesions Simulating Otosclerosis

Francis A Sooy, M D (by invitation)

Experiences with the Stapedectomy Technique of Shea for the Surgical Treatment of Otosclerosis

David Myers, M D ,
Woodrow D Schlosser, M D (by invitation), and
Richard A Winchester, Ph D (by invitation)

SUMMARY

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Clair M Kos, M D

Partial Stapedectomy
J V Hough, M D (by invitation)

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Harold Schuknecht, M D
(Open discussion of last four papers)

Acceleration as a Means of Determining the Sensitivity of the Components of the Non-Auditory Membranous Labyrinth
Walter H Johnson, Ph D (by invitation) ,
J Brydon Smith, M D , and
Joseph A Sullivan, M D

Localization of Monaurally Impaired Listeners
Richard Viehweg, M A (by invitation)

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THE
LARYNGOSCOPE.

VOL LXX FEBRUARY, 1960 NO 2

DISCUSSION ON ACOUSTIC NEUROMA*†

DR M R DIX
and
DR C S HALLPIKE,

London, England

THE OTONEUROLOGICAL DIAGNOSIS OF TUMOURS OF THE *VIIITH NERVE*

In the course of the last 12 years it has been possible through the good offices of our colleagues at Queen Square, to examine a large number of subjects suffering from tumours of, or affecting, the VIIIth nerve

Although their total number has amounted to some 300, observations have been confined to a series of 95, in most of which a full range of otoneurological tests was carried out, including tests for loudness recruitment, and in which the presence of a tumour was later confirmed, either at operation or post mortem

It is not the purpose of this communication to present any comprehensive catalogue of these findings Instead, we have confined ourselves to a few familiar and practical themes concerned in the main with certain tests of cochlear and *vestibular* function, which lie very clearly within the province of the otologist and provide results of high diagnostic importance

---

*From the Otological Research Unit Medical Research Council National Hospital Queen Square London W C 1

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Our cases have been divided into the following two groups

Group I—Acoustic neurofibromata The number of subjects in this group was 70

Group II—Other tumours arising within or invading the cerebello-pontine angle, and so involving the VIIIth nerve The number of subjects in this group was 25

COCHLEAR FUNCTION

Hearing Loss—Our first theme is the amount of the hearing loss in subjects with VIIIth nerve tumours A review of the literature, especially some of the earlier papers, leaves the impression that the hearing loss is nearly always severe or complete, and indeed the belief still seems to be held in some quarters that, unless this is the case, the diagnosis of a tumour is doubtful (Cushing, 1917)

TABLE I

Hearing in 95 Subjects with Verified Angle Tumours

	Group I Acoustic Neurofibromata	Group II Other Angle Tumours
Complaint of deafness	64 of 70 subjects (91%)	18 of 25 subjects (72%)
Average pure tone loss for speech frequencies	53 db for 70 subjects	42 db for 25 subjects

More recent publications have made it necessary to modify this view and Ireland (1949), in his series of 92 cases, reported normal audiometric findings in 5 per cent Edwards and Paterson (1951) also reported normal hearing in 2 per cent of their 157 cases

We have considered separately the findings in our two groups of subjects Of the 70 subjects in Group I, with acoustic neurofibromata, there was a complaint of deafness in 91 per cent For the 25 subjects of Group II, however, this figure was only 72 per cent This difference was in agreement with the audiometric findings Here, we have expressed the hearing loss as an average in decibels (db) for the main speech frequencies, 500, 1,000 and 2,000 c/s For Group I the average hearing loss was 53 db For Group II, however, the hearing loss was only 42 db These figures are summarized in Table I

Normal hearing does sometimes occur In all of our 165 cases of verified acoustic neurofibromata, normal audiograms were found in two only In the much smaller number of verified cases of other types of angle tumour, 63 in all, four showed normal hearing It would seem, thus, that the degree of deafness in tumours affecting the VIIIth nerve is of some value in establishing the character of the tumour, the finding of slight or moderate deafness favouring the diagnosis of some variety of angle tumour other than an acoustic neurofibroma

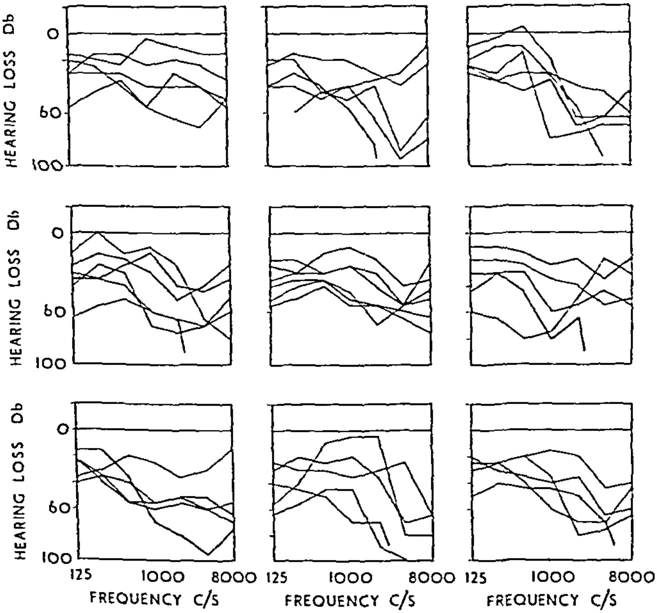

Fig 1 Angle tumours Group I

The 95 subjects included in Groups I and II amount to approximately one-third of our 300 cases Their average hearing loss amounted to 48 decibels and in all 95 a measurable degree of hearing was preserved It may be concluded normal equal loudness line, indicating the absence of loudness recruitment

The number of cases in which full loudness recruitment was present at one or more frequencies was found to amount to some 10 per cent This requires explanation, and it may be recalled that we have explained it as being due to changes in the cochlear end-organ resulting from interference by tumour pressure with the cochlear blood supply (Dix and Hood, 1953) This argument was fortified by the finding in two such cases of the disappearance of loudness recruitment after the removal of the tumour

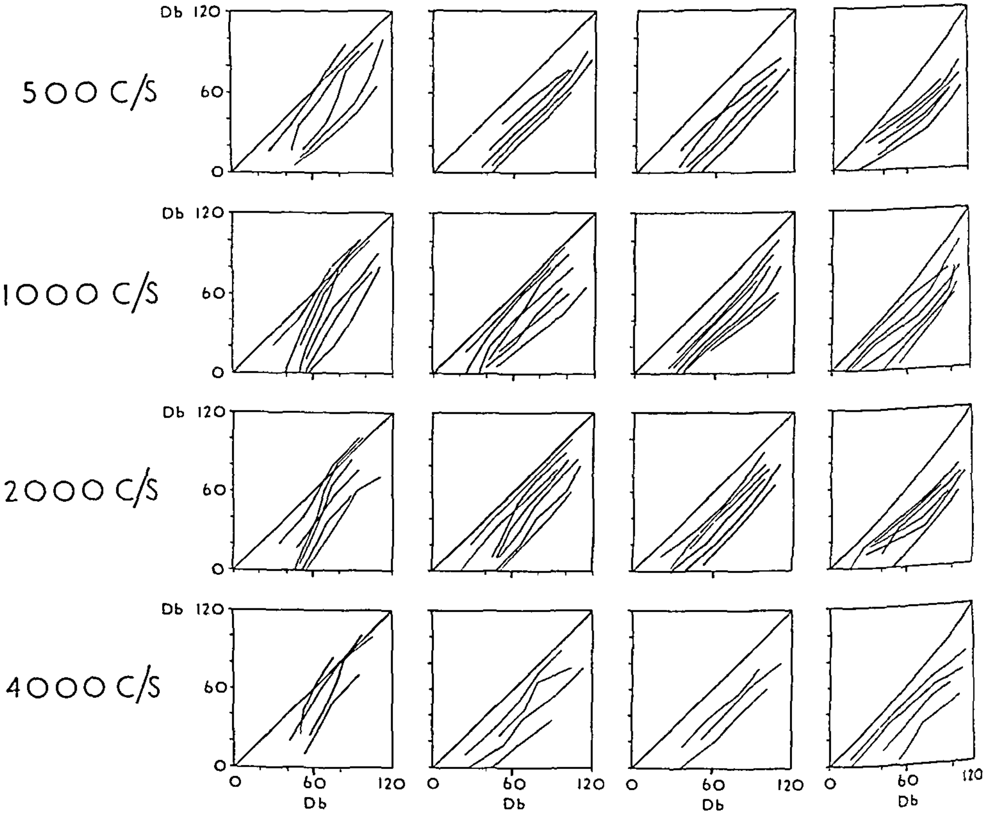

Fig 3 Angle tumours Group I

Before leaving the subject of loudness recruitment, it would seem appropriate to consider the experiences of others From a wide review of publications dealing with the diagnostic value of the tests it seems safe to say that since the demonstration (Dix et al, 1948) that loudness recruitment was pathognomonic of end-organ disease, and was characteristically absent in VIIIth nerve tumours, the loudness recruitment test has continued to provide a most valuable means for the differentiation of cases of perceptive deafness into two classes end-organ deafness and nerve fibre deafness A complete list of other investigators who have substantiated these findings would be a long one, but the following seem particularly de-

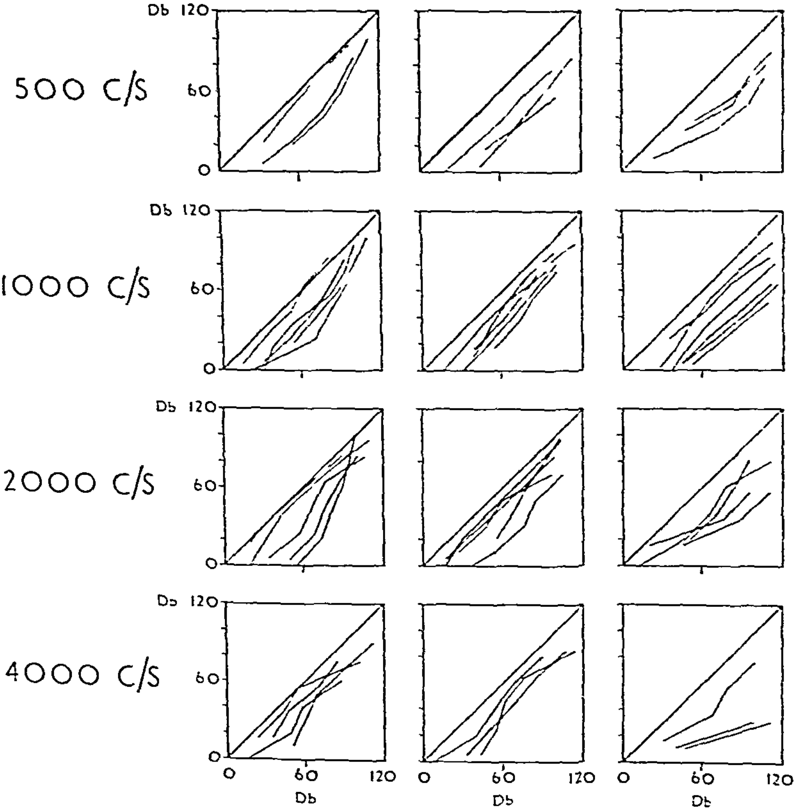

Fig 4 Angle tumours Group II

serving of reference. Lundborg (1952), Moller and Nenzelius (1957) in Sweden, Kristensen and Jepsen (1952) and Thomsen (1955) in Denmark, Luscher (1950) in Switzerland, Hahn (1954) in Italy, Zangemeister (1950), Jatho (1956) and Stenger (1956) in Germany, Ireland (1949) in Canada, and Riesco MacClure (1957) in Chile, and finally, Segal and Hociota (1957) in Roumania. In the United States, too, an increasing and formidable body of evidence has been accumulated by observers including Saltzman (1949), Eby and Williams (1951), Jerger (1952), Schucknecht and Woellner (1955), Yantis (1955) and Goodman (1957). Finally, Simonton (1956), in a paper published from the Mayo Clinic, reports findings very reminiscent of our own, in which he describes the disappearance of loudness recruitment following the removal of a small tumour. He agrees with us in attributing the loudness recruitment to end-organ damage, resulting from interference by the tumour with the cochlear blood supply.

In this country few contributions seem to have been made, and Denny (1955) has described the test as "not giving much assistance," the reason given being that absence of loudness recruitment in cases of VIIIth nerve tumour is not a constant finding. This we consider an unreasonable view. After all, very few diagnostic tests can be relied upon to give 100 per cent results. Yet, in some respects, the diagnostic significance of the loudness recruitment test is as high as this. Thus, our own figures, well supported by others, show that the test is positive in 100 per cent of cases of Ménière's disease, and negative in 90 per cent of cases of VIIIth nerve tumours. A negative result, therefore, gives odds approaching the infinite against Ménière's disease. A positive result gives odds of nine to one against a tumour. There can, we think, be few diagnostic tests which give results more decisive than this.

Bone Conduction.—Another group of tests of great importance in the investigation of cochlear function is that of bone conduction. While the loudness recruitment test is indispensable in differentiating the two great categories of perceptive deafness, end-organ deafness and nerve fibre deafness, the diagnosis of perceptive deafness must first be established, and for this we are still vitally dependent upon the bone conduction tests

Up to some ten years ago, references were to be found in the literature describing the occasional occurrence of true conductive deafness in cases of VIIIth nerve tumour The tests were carried out in the classical manner with tuning forks, and evidence of satisfactory masking procedures was rarely provided

We (Dix and Hallpike, 1950) have described two cases of VIIIth nerve tumours, examined some two years earlier, in which we had encountered evidence of conductive deafness In both of these cases the Rinne test gave what appeared to be true negative results

We endeavoured to explain this conductive deafness in terms of a rather unusual albuminoid coagulum within the labyrinth We used for our masking a hissing noise delivered to the unaffected ear from a speaking tube This, at the time, was the best technique available, and we know now that its masking efficiency is inadequate Since then electronically generated masking noises have been used, and for a number of years we have relied entirely on the narrow-band type of masking noise described by Hood (1957)

Since we have had this invaluable facility at our disposal, we seldom use the tuning forks for the evaluation of hearing by bone conduction, and have never seen a case of VIIIth nerve tumour in which there was any evidence of conductive deafness So far as we know, the deafness of VIIIth nerve tumours is always of the perceptive type We tell this story of our experiences, and of our change of views, since it illustrates in a manner which could not be bettered the extent to which efficient otoneurological diagnosis is dependent upon up-to-date audiological methods

VESTIBULAR FUNCTION

We are in agreement with others that the early vestibular symptoms of VIIIth nerve tumours take the form of a sense of imbalance and unsteadiness of gait, and this was complained of in 73 per cent of our 70 cases Complaints of vertigo were only made in 49 per cent The term "vertigo" is not restricted to the paroxysmal kind of attack typical of Ménière's disease, and this we found to be of rare occurrence

Stance and Gait—Tests for ataxia of stance and gait are important These are carried out with the eyes closed and open Abnormalities were present in 77 per cent of our cases, though it must be added that their localizing value is by no means always clear cut

Spontaneous Nystagmus—Examination for spontaneous nystagmus is of very great importance, and considerable experience is often needed to detect it in its early stages when it is often of high significance The eyes must be observed under good diffuse lighting with the gaze directed straight ahead and deviated to left and right to points just within the limits of binocular vision Spontaneous nystagmus was present in 81 per cent of our cases in Group I as shown in Table II

TABLE II

Spontaneous Vestibular Nystagmus in 95 Subjects with Verified Angle Tumours

Character of Nystagmus	Group I Acoustic Neurofibromata	Group II Other Angle Tumours
To contralateral side only	20 (29%)	9 (36%)
To ipsilateral side only	1 (1%)	0
To both sides	36 (51%)	8 (32%)
Nystagmus absent	13 (19%)	8 (32%)
Total	70	25

The findings have been considered in relation to the stage of development of the lesion, and it is fairly clear that nystagmus to the unaffected side is the first to appear, followed, as the tumour progresses, by nystagmus to the affected side In some subjects we observed the development of this nystagmus over a period When first detected it is of the first degree only, that is to say, it occurs only when the eyes are directed towards the unaffected side Later it becomes of second degree, that is to say, present also in the straight ahead position of gaze At this stage it usually is accompanied by first degree nystagmus towards the affected side The findings seem to indicate that these two kinds of nystagmus, contralateral and ipsilateral, are due to lesions of different anatomical structures, which are affected in a particular sequence The structures whose derangement is responsible for the contralateral nystagmus are probably certain of the vestibular nuclear elements within the brainstem As to the structures which are concerned with the ipsilateral nystagmus, less can be said The evidence, however, would seem to point to certain of the cerebellar elements

Positional Nystagmus—Positional nystagmus is another physical sign of great importance and interest It is, however, a specialized subject which cannot be included in the present contribution

Caloric Tests—Lundborg (1952), in his extensive study of 300 proved acoustic neuromata, described the caloric as the most valuable of the tests of vestibular function He stated that the results were abnormal in 84 2 per cent of his subjects and normal in 15 8 per cent Revilla (1947) also, in his review of 145 cases, found 6 8 per cent of normal caloric results

Our own results have shown the caloric tests to give abnormal results in 100 per cent of cases, and we regard the test, performed as we perform it, as indispensable

The reason for the discrepancy between our findings and those of other workers arises, we feel sure, from differences in technique Lundborg used 10 to 20 cc of water at either 27° C or at 47° C, while Revilla used cold water only We think it essential to use both cold and hot water at 30° C and 44° C, as described by Fitzgerald and Hallpike (1942), and to interpret the results in accordance with certain principles which would seem to us to be reasonable and well founded As explained in a number of publications since 1942, abnormalities in the pattern of the caloric test responses may take one of three possible forms there may be a canal paresis, there may be a directional preponderance to the opposite side, or the two may be present in combination

In Fig 5 are shown these three possibilities, as they may be found in a tumour of the right VIIIth nerve There may be right canal paresis (1), there may be a directional preponderance to the opposite side (2), or the two may be combined (3) The way the combined pattern (3) arises is easy to understand if we introduce into the directional preponderance pattern (2), a right canal paresis (1) The right cold response is then brought back to the point shown by the white triangle and the cold responses are thus made equal and apparently normal (3)

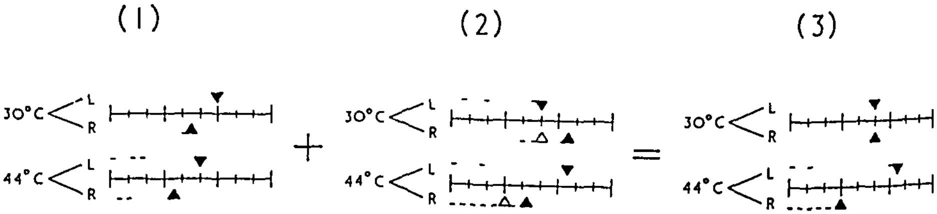

Fig 5 (1) Right canal paresis Lesion of right canal elements (2) Directional preponderance to left Lesion of right tonus elements (3) Combined lesions of right canal and right tonus elements

The right hot response again is brought back to the point shown by the white triangle, and the hot responses are thus widely separated (3) Consideration of these three patterns shows very clearly that cold stimulation alone will give the true answer only in cases of canal paresis (1) If there is a directional preponderance (2), then the cold responses would be misleading, since they would seem to show sensitivity loss of the left or unaffected labyrinth Similarly with the combined pattern (3) the cold responses again give a misleading impression, namely, that the labyrinths react equally and normally

The results of our caloric tests are shown in Table III They are 100 per cent abnormal It may be seen that in 15 of our 95 cases exhibiting directional preponderance or a combined pattern, the results obtained with cold stimulation alone would have appeared entirely normal, or would have appeared to give a better response from the affected labyrinth. This, we feel sure, is the explanation of normal caloric responses reported by Lundborg and others in VIIIth nerve tumours.

DISCUSSION

We come now to certain wider problems connected with the otoneurological diagnosis of VIIIth nerve tumours. These we shall endeavour to outline in a series of questions which from time to time are put to us, directly or indirectly, by neurologists and otologists with practical interests in the clinical value of the modern diagnostic tests which we use.

TABLE III

Abnormalities of Caloric Responses in 95 Subjects with Verified Angle Tumours

	Group I Acoustic Neurofibromata	Group II Other Angle Tumours
Canal paresis (total)	58 (83%)	8 (32%)
Canal paresis (partial)	7 (10%)	7 (28%)
Directional preponderance	0	3 (12%)
Combined	5 (7%)	7 (28%)
Total	70	25

The first question is to what extent is the diagnostic evidence which they provide really indispensable? For instance, is it not true that in the majority of cases the demonstration of deafness will call for no special audiometric equipment, and that the simple observation of spontaneous nystagmus and the results of an ice-water caloric test will provide all we need to know of the vestibular dysfunction? If so, then such tests could be performed in any unspecialized aural clinic, and indeed the neurologist who is generally in charge of the case could well apply them himself and integrate their results with the neurological signs and symptoms which form an essential part of the clinical picture. Where, here, is the need for any highly specialized neuro-otological investigation? The question, arising as it does from prevailing clinical experience of these tumours with advanced deafness, spontaneous nystagmus and other neurological signs, is a perfectly reasonable one, and indeed, nothing has done more to discredit neuro-otology in the past than the unreasonable use in cases of this kind of over-elaborate diagnostic tests, in particular the rotational tests

Our answer is that although these tumours are commonly presented for diagnosis in this advanced stage they should, if we look to the future, be seen much earlier, that is, in their otological stage, when their effects are for the most part limited to the VIIIth nerve elements During this stage they will be detected by means of modern tests and by these alone

The second question arises from what has just been said Are we quite right in concluding that many of the tumours do pass through this purely otological stage? The affirmative answer, which we feel able to give, is based in part upon the evidence of histopathology, and in part upon clinical experience

Let us consider first the neurofibromata of our Group I The prevailing view of their pathogenesis, based chiefly upon the work of Henschen (1910, 1915) is that they arise from the neurilemmal portion of the vestibular nerve within the porus acusticus Our own clinical data, being derived in the main from fairly advanced tumours, do not yield any decisive evidence as to the exact site of origin of the tumours Nevertheless, some useful information is provided Thus, in the 47 subjects whose audiograms are shown in Fig 1, there was, as shown by these audiograms, a substantial preservation of cochlear function In 83 per cent of these, however, the caloric responses were completely abolished, and in another 17 per cent they were severely reduced

Partial loss of trigeminal function and other neurological disturbances were present in most of these cases in varying degrees Nevertheless, the dominant note of the overall clinical picture is quite clearly indicative of a lesion having its maximum effect upon, and therefore originating in, the vestibular elements of the VIIIth nerve We would expect, therefore, upon the evidence of these findings that all these tumours did, in fact, pass through an early otological stage in which their effects would be substantially limited to the VIIIth nerve, particularly its vestibular elements

As regards the other tumours of our Group II, the story is a different one Since the VIIIth nerve is not primarily involved, the VIIIth nerve signs are less in evidence than the neurological signs Thus, good preservation of hearing is more likely to occur with incomplete loss of the caloric responses Indeed, it is our experience of these tumours that some such unusual balance between the otological and neurological abnormalities often provides a useful key to their diagnosis It need hardly be said that here again much depends upon the precise evaluation of cochlear and vestibular function by means of modern tests

The next question is this Granted the value of modern tests for the early diagnosis of these tumours, is the course of the surgical treatment greatly influenced thereby? This question may seem a peculiar one Nevertheless, it is not without practical significance Thus, in some subjects in whom we have made the diagnosis in advance of the appearance of any severe neurological signs such as nystagmus, surgical treatment has been considered inadvisable, the opinion being held that in the absence of urgent indications such as papilloedema, the known post-operative morbidity makes it wiser to leave the tumour undisturbed Now it is not really for us to provide the answer to this contention Nevertheless, we feel bound to point out that it appears to be based upon the experience of the post-operative morbidity in advanced tumours Experience of the post-operative morbidity in small tumours is unfortunately not yet widely available, but from such that we have, that is to say of tumours operated upon before the appearance of any evidence of brain-stem distortion, the post-operative morbidity is little more than that encountered following a section of the VIIIth nerve for Ménière's disease (Elliott and McKissock, 1954) The real difficulty at present is, we feel sure, that not enough of these cases are presented for diagnosis in their early, or otological stage

Having decided that the early diagnosis of these tumours is an essential factor in their effective surgical treatment and that this early diagnosis depends largely upon modern oto-neurological tests, we return finally to the question "What exactly are these tests, and what special technique and equipment do they require?" The subject needs no more than a swift review

The Tests of Vestibular Function—Here, the caloric tests are of prime importance Of their principles and practice no more need be said

The Tests of Cochlear Function—For the establishment of the perceptive character of the deafness, reliable bone conduction tests are essential These are impossible in many cases unless the correct masking procedure is employed This involves the use of insert receivers and narrow-band masking noise, as described by Denes and Naunton (1952) and by Littler, et al (1952), and more recently developed by Hood

The Loudness Recruitment Test—Here, again, special apparatus is required, of which the principles and their applications have been specified and fully described in a number of publications (Dix, et al, 1948, Dix and Hallpike, 1949, Hood, 1950, Dix and Hood, 1953, Hood, 1954)

Test Equipment—No serious difficulty would seem to arise in the case of the caloric tests We have for years used a simple douche can and thermometer, but thermostatically controlled tanks make the work easier These are readily obtainable and we warmly recommend their use

More serious problems arise in connection with the equipment needed for special tests of hearing In our clinic practically all of the equipment we use has been designed to accord with certain theoretical principles, and has been constructed accordingly in our laboratories In our hands it gives excellent results, and these provide the basis of a number of contributions published in the course of the last ten years relating to a variety of problems of cochlear and vestibular function in health and disease

On the whole, our results have been substantiated, latterly, to an increasing extent by other workers There is, however, no doubt that difficulties have arisen and it seems reasonable to suggest that they are attributable to some extent to the lack of the special equipment which we know to be such a vital necessity Certainly we can say of any commercially available audiometers that in none is there yet provided any narrow-band masking for the performance of bone conduction tests, while facilities for loudness recruitment tests, when available at all, are nearly always quite inadequate

Although the stress which we lay upon these various technical difficulties may seem very strong, we nevertheless consider it fully justified If the otologist is to play his part, and play it well, in what we regard as an important and expanding field of work, he must have at his disposal up-to-date and efficient electro-acoustic and other equipment There is no difficulty in specifying what this equipment should be, or its performance, and we hope that before long it will be properly standardized and made generally available

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OTOLOGICAL FUNCTIONAL MICRO-SURGERY POST GRADUATE COURSE

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SOME PRELIMINARY OBSERVATIONS ON THE EFFECT OF GALVANIC CURRENT ON TINNITUS AURIUM[*]

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INTRODUCTION

Galvanic stimulation has now been used for many years as a supplementary test for vestibular function The effects of this type of current on auditory activity are less well known It has been reported that galvanic stimulation can produce tinnitus in the ear on the same side as the cathode, but there seems to be no detailed study on this phenomenon

Our interest in this problem arose when it was found that, in some cases, during routine galvanic stimulation tests, tinnitus could be increased or decreased in intensity according to the polarity of the current. Our interest was further stimulated by the results of Tasaki and Fernandez[9] on the effects of DC currents on the cochlear microphonics and action potentials recorded from guinea pigs In view of the fact that their results suggest that the currents they used acted on the sensory receptors themselves rather than on the nerve or more centrally, we wondered whether galvanic stimulation may be able to distinguish tinnitus due to peripheral causes from that due to central causes The extent to which this question has been answered so far is reported in this paper

METHODS

Thirty-three patients were studied All of these patients

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RESULTS

Fifteen patients reported changes in the intensity of the tinnitus accompanying changes in the current strength. Group A (see Table I) shows those patients whose tinnitus was affected by the current, while Group B (see Table I) consists of those in whom no effect was produced. In those patients who had unilateral tinnitus, the anode on the side of the tinnitus would produce a decrease in the intensity of the tinnitus, in some cases total suppression, as the strength of the current was increased. This suppression would last throughout the duration of the stimulus and, as the current was gradually reduced to zero, the tinnitus would gradually return to its former intensity.

TABLE I

Results of Galvanic Stimulation Effect on Tinnitus Aurium in 33 Cases Classified as to Hearing Loss

Diagnosis	Conductive	Hydrops	Post Infect	Skull Fract	Noise Trauma	Presbycusis	Unknown	Normal
Group No 1*—(affected)	2	2	2	1	1	1	6	0
Group No 2†—(no effect)	1	0	0	0	3	10	2	2

*Group No. 1—Subjects whose tinnitus was affected by the galvanic stimulation.

†Group No. 2—Subjects in whom no effect was produced.

The current strength at which the total suppression of the tinnitus was reported varied from 1.5 mA in one patient to 8.8 mA in another. It was a common finding, however, that once the subject had reported that the noise was diminishing in intensity, it disappeared entirely within 1.0 mA. Conversely, with the cathode on the tinnitus side, the opposite effect was obtained, *i.e.* the tinnitus would increase in loudness as the current was increased and would return to its former level as the current was reduced gradually to zero. Threshold values of the change in the tinnitus were the same complained of "noises in the ear (s) " Each patient had complete ear, nose and throat examination that included a detailed audiometric survey in addition to gross neurological and static labyrinth investigation

The test was carried out in a sound treated room so that ambient noise would not detract from the patient's ability to hear his own tinnitus The subject was first asked to match his tinnitus for both frequency and intensity by presenting various sample tones and mixed noises in the sound field The electrodes, which consist of a round 1″ diameter electrode and a 2″ pad type, were then applied to the following eight positions

1 Anode on right zygomatic arch, cathode in left hand

2 Reverse of No 1

3 Anode on left zygomatic arch, cathode in right hand

4 Reverse of No 3

5 Anode on right zygomatic arch, cathode on left zygomatic arch

6 Reverse of No 5

7 Anode on zygomatic arch, cathode on ipsilateral mastoid

8 Reverse of No 7
(Smaller electrodes ⅛″ and ¼″ diameter are used for No 7 and No 8)

Each patient in this series had already experienced the galvanic test for labyrinthine function, so that he was aware ot the sensations of vertigo, tingling and copper taste caused by the current In no case was any suggestion made as to the possible effects of the current, but the patients were asked to report any change in their tinnitus they might experience Repeat measurements were made, not only during the test, but also when the patients returned to the clinic several weeks or months later Only those patients whose responses were definite and repeatable were included in the group denoted as giving positive results

results, namely, *1* that we are uncertain of the site at which the current may exert its effect, *2* that tinnitus aurium can be due apparently to a number of separate factors

If we could assume that the current produced its effect mainly at the cochlea, then our findings could be explained on the basis of those reported by Tasaki and Fernandez[9] and Tasaki, Davis and Eldredge[10] These authors showed that a DC polarizing current, with the positive pole in the scala vestibuli and the negative pole in the scala tympani, increased the amplitude of the cochlear microphonics relative to the amplitude of the responses when no current was applied When the polarities were reversed, however, the amplitudes of the cochlear microphonics were depressed The amplitudes of the action potentials of the VIIIth nerve—judged by the heights of the N1 responses—were similarly affected by the polarizing current Tasaki and Fernandez point out that their observations are similar to those from experiments on other sensory receptors and that the applied current affects the process of initiation of the propagated nerve impulses in the sensory nerve fibers and not the processes of conduction We might, therefore, speculate that in some cases of tinnitus aurium of peripheral origin, the pathological process may have caused the resting membrane potential to be raised to critical levels for firing propagated nerve impulses In these cases, the anodal current could conceivably reduce the value of the resting membrane potentials to levels below those which trigger the propagated impulses The fact that the current appeared to affect only the tinnitus rather than auditory acuity for all frequencies tends to support the above idea, thus, those cases in which no result could be obtained, would suggest that the current did not reach those parts of the cochlea concerned with the tinnitus, or if it did reach the cochlea, it was not of sufficient strength to change the membrane potential effectively It is also probable that in some of these cases (presbycusis) the tinnitus was not of peripheral origin

This thesis, however, must be tempered by the fact that there is some evidence that galvanic stimulation may exert its effect centrally Dohlman[3] has stated that the absence of for both the increase and decrease of the current In those patients with bilateral tinnitus the results were of the same pattern if the anode were on the right zygomatic arch and the cathode on the left, tinnitus in the right ear would fade, while that in the left ear would become relatively louder as the current strength was increased There were four patients who had had occasional tinnitus, but who had no tinnitus at the time of the examination In these four cases, it was possible to produce tinnitus in the same ear by cathodal stimulation Each subject reported that the noise so produced was the same as that previously experienced

The cases summarized below illustrate our results

Case 1 L B, a colored female, 22, CNS lues Tinnitus in right ear ringing, steam escaping Tinnitus in left ear, motor noise synchronous with pulse beat Audiogram shows speech reception threshold (SRT) of 95 db in both ears, discrimination score, AU, 0 per cent Caloric tests show hypocative labyrinth Hearing loss has been progressive since 1957 Anodal current decreased the tinnitus while cathodal current increased the tinnitus in respective sides

Case 2 V S, white female, 48 Normal hearing until skull fracture in 1941 Tinnitus, low pitched roar, AD Audiogram shows no serviceable hearing in right ear while left ear is normal Caloric tests reveal marked hypocative right labyrinth with normal left side Galvanic test tinnitus abolished with anode on right zygomatic arch (Positions 1, 5, 7), marked increase in the tinnitus with cathode on right zygomatic arch (Positions 2, 6)

Case 3 L C, white male, 51 Endolymphatic hydrops (Meniere's) Tinnitus in left ear described as whistle Left ear has flat nerve loss of 60 db, discrimination score of 14 per cent, complete loudness recruitment, marked tone decay and diplacusis Right ear normal Caloric tests show hypocative left labyrinth and normal right labyrinth Galvanic test anode on left zygomatic arch, (Position 3) produced change in the tinnitus, cathode on left zygomatic arch (Position 4) produced marked increase in the whistle When anode was placed on left zygomatic arch and the cathode on the right zygomatic arch (Position 6) the whistle was heard in both ears

Case 4 H C, white female 48 Bilateral otosclerosis with nerve involvement in left ear Tinnitus bilateral low pitched roar heard more in left ear Audiogram shows a 35 db conductive loss with 98 per cent discrimination in right ear, a 70 db mixed loss, discrimination score 88 per cent in left ear Caloric tests were normal bilaterally Galvanic test with anode on left zygomatic arch (Position 2, 6) the tinnitus in left ear was abolished, with cathode on left zygomatic arch (Position 4, 5) the tinnitus in left ear was increased There was no effect on the tinnitus in right ear from any electrode positioning

DISCUSSION

There are two main difficulties in the interpretation of the tinnitus could be affected by the galvanic current had severe hearing losses

Davis[1] has maintained that "there must be some regulatory mechanism to adjust the excitability of the nerve fiber and the strength of the resting current to levels very close to the threshold of stimulation Disturbance of this 'homeostatic' mechanism in one direction would cause loss of sensitivity Disturbance in the opposite direction would give tinnitus" Our results would agree with this idea for most of the cases which we studied, for we feel that the galvanic current must be affecting this regulatory mechanism, whatever it may be We do not, of course, claim any therapeutic value for these cases, but we do feel that this method provides a useful physiological tool for the study of a condition for which experimental animals cannot be used

SUMMARY

1 The effects of galvanic stimulation on tinnitus aurium in 33 patients are discussed

2 Fifteen patients reported changes in the intensity of the tinnitus according to the polarity of the current If the anode were placed on the zygomatic arch on the side of the tinnitus, the intensity would decrease as the current was increased conversely, with the cathode on the affected side an increase in the current strength would produce an increase in the tinnitus intensity

3 Eighteen patients reported no change in the tinnitus from galvanic stimulation

4 These results are discussed with reference to tinnitus of possible peripheral and central origins and also in relation to recent experimental cochlear studies by others

We thank Professor David Myers for his kind encouragement, and Professor G Dohlman for his helpful discussion

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the galvanic response is due to a degeneration of cells in the ganglia vestibulare and their central connections. More recently, Dix, Hallpike and Harrison,[2] and Ingelstedt and Walander[4] found that if the peripheral vestibular apparatus was damaged by streptomycin, the galvanic current could exert its effect on the vestibular nuclei. On the other hand, other data have been presented by Mackenzie,[7] and Gloria and Fowler[5] which indicate that the galvanic current is effective only peripherally. If, as seems likely, high intensities of current reach the central structures, it would be difficult to see how the current could be so specific in its action according to its polarity. It may be, of course, that an anodal current will decrease the values of the membrane potentials of the neurons in the central nucleus which are responsible for the tinnitus, in the same way as was suggested for the peripheral action of the current. On the other hand, the current may in some way be stimulating efferent fibers and modifying the sensory input to the central nervous system, an idea that appears plausible from the anatomical findings of Rasmussen,[8] and the physiological studies reported by Galambos.[1]

Whatever the mechanism may be, we agree with Dohlman's suggestion (personal communication) that electrophysiological studies should be carried out to determine the patterns of discharge of single neurones in central structures in response to DC polarization of the cochlea.

It does not seem probable that the effects reported could be due to anodal block of the nerve fibers, for the current strengths do not seem to be high enough to produce such results. This fact is emphasized by Tasaki and Fernandez in their studies.

It is interesting to note that in Table I but one out of 11 patients with presbycusis reported change in his tinnitus when the current was applied. This, perhaps, may be explained on the basis that tinnitus accompanying presbycusis is of central rather than peripheral origin. This interpretation, of course, cannot be substantiated at the present time. Another interesting and, perhaps significant, observation in this series was the fact that the majority of patients whose

THE IMPORTANCE OF HEARING AT 3 KC FOR UNDERSTANDING SPEEDED SPEECH

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For some time we have been bothered by a discrepancy between the relatively low frequency cut-off which allows low-pass filtered speech signals to be understood well by the normal ear, and the somewhat higher frequency region at which individuals seem to need to hear in order to operate well in everyday life

In the classic paper on speech intelligibility by French and Steinberg[1] it is shown that for one set of typical talkers and listeners a syllable articulation of 95 per cent correct was achieved when all frequencies above about 4,500 cps were eliminated, however, when the effect of words, especially polysyllabic words is added to the situation, or even when these words are put into sentences with all the mnemonic, redundant, symbolic, and evocative associations and rules of human language, the frequency effect seems to change

The effect of low-pass filtering on speech sounds has been attacked by Hirsh, et al ,[2] who showed that eliminating all frequencies above 1,800 cps reduced nonsense syllable articulation to 73 per cent, but polysyllabic words in isolation remained high at about 97 per cent, indeed, polysyllabic words in isolation remained at about 95 per cent even when all frequencies above 800 cps were eliminated!

Some data on sentence intelligibility using the same filters were summarized by these same workers It is stated that sentence intelligibility is similar to polysyllabic words in isola-

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WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

The West Virginia Academy of Ophthalmology and Otolaryngology will hold its annual meeting at the Greenbrier Hotel, White Sulphur Springs, W Va, from April 10 to 12, 1960

Among the guest speakers will be Dr Harold G Scheie, Professor of Ophthalmology, Pennsylvania University School of Medicine, Philadelphia, Pa, Dr Charles E Iliff, Associate Professor of Ophthalmology, John Hopkins University School of Medicine, Baltimore, Md, and Dr Oscar T Becker, Clinical Associate Professor of Otolaryngology, University of Illinois, Chicago, Ill

For any additional information please contact the secretary, Dr Albert C Esposito, First Huntingon National Bank Building, Huntington 1, W Va

The relation of sentence intelligibility to syllable- and letter-articulation was specified by Fletcher (see p 266) From these data one can predict, though with considerable error, sentence intelligibility from a knowledge of syllable- or letter-articulation as derived from nonsense syllables The Bell laboratories in general used this conversion in their thinking, and have used nonsense syllables for the most part in assessing communications equipment There were two reasons for this first, as noted above, the testing time is much shorter, second, the sentence tests work well only in a rather narrow region of distortion, unless a system distorts rather badly, all sentences come through at or near 100 per cent They obviously cannot be used to rank order an ordinary group of communications systems, or ears, all of which perform equally well

A minor difficulty with these particular sentences is that they often demand quite good intelligence or special knowledge, of politics, current events, New York City geography, etc

Lists of sentences have been built up from time to time by the Harvard Psycho-Acoustic Laboratory[4] and others for special purposes, such as the testing of communications in noise, but no list has as yet been published to meet the several demands put by our immediate requirements We thus felt called upon to construct our own

One hundred sentences of colloquial difficulty were written, about five to 15 words each, constructed of key words and ideas much as the Fletcher lists, together with a group of four words for each sentence in a multiple-choice response

The rules for construction were

a Three key word-ideas per sentence

b Of the four responses offered on the answer sheet, only one could conceivably be correct if the whole sentence was understood

c If any one of the key words was missed, at least one and tion, in that scores of nearly 100 per cent are obtained with 800 cps low-pass filtering

Such astounding results are repeatable in the laboratory, but they do not agree with clinical experience An ear with an audiometric pattern sloping sharply off at 800 cps—or even an octave or more higher for that matter—can perhaps handle clearly spoken speech suitably amplified in the absence of noise or other distortion, but no one will question that such an ear will in many other situations operate well below normal Every hearing clinic is filled with such individuals seeking help

We have sought to resolve, or at least throw light on the discrepancy by presenting not clear but distorted speech to ears with audiometric patterns which simulate low-pass filtering We fixed upon a speedup in words per minute as a convenient and illuminating type of temporal distortion For this work we used sentences rather than words or syllables

There are several reasons for preferring sentence intelligibility over word-, syllable-, or phoneme-articulation first, especially important when working with patients rather than with trained listeners, the phonetic alphabet need not be used, second, with sentences it is easier to preserve the many natural transitions in connected discourse from one phoneme to another This aspect of speech has recently been emphasized by several laboratories The major reason for utilizing material other than sentences for assessing general speech recognition is a matter of time Finally, with speeded speech the measurement of the speech unit per unit time is much more convenient

Fletcher[3] made these points many years ago, and provided one transfer from sentence intelligibility to articulation as measured by syllables and letters He had his laboratory construct 49 lists of 50 sentences each Sentences were five to 12 words in length, each containing four or five “thought” words all essential to the meaning of the sentence The response to a sentence (each was cast in an interrogative or imperative form) could be scored right or wrong An attempt was made to reduce intellection

Forms 1 and 2 for neither A nor B is significant at the 05 level

In summary, we had shown that even with what is considered extremely rapid speech one can expect normal listeners to do quite well so long as the talker maintains rather crisp articulation (As a matter of fact, it is a question whether the drop from around 100 per cent to 82 4 per cent intelligibility is entirely due to the increased speed Some, if not most, of the decrement is certainly due to an occasionally slurred or partially elided word or syllable) Further, we

TABLE I

Frequency Distributions of Scores on Speeded Speech

Tests A and B (N 25)

	Per Cent Intelligibility Attained							
	100-98	97-95	94-92	91-89	88-86	85-83	Mean	Range
A Form 1	3	3	7	6	4	2	80 8	70 98
A Form 2	2	10	5	3	5		84 0	72 98
B Form 1	6	11	8				88 4	84 98
B Form 2	9	9	6	1			89 6	82 98

had tapes covering the range 280-345 words per minute, each with an alternate comparable form

With these tapes and this information we were ready to test the hypothesis in the clinic that audiograms defective in the higher frequencies would do relatively poorly on these tapes, in the absence of central damage

Patients were collected, one at a time, who possessed the audiometric patterns necessary and who gave no evidence whatever of central damage and were far from senile Each was given a complete oto-audiologic workup and then given Test A (either Form) at a level about 40 db over his 50 per cent threshold for the PB lists

In a group of six men (eight ears) with no audiometric loss greater than 15 db through 3 kc, but with losses of 20 to 85 db at 4 kc, no disturbance of speeded-speech reception was seen The average per cent correct for Test A was 84 5 per cent, range 76 to 94 This is indistinguishable from the usually two or three of the three other responses would offer a reasonable answer

There were, of course, numerous exceptions to these very difficult response item requirements, but in the main the rules were followed Mr Robert Pettie and Mr William Atkinson composed sentences and responses Their sentences were dovetailed to compose the full list (see Appendix A)

A speaker with general American dialect, who habitually talks rather rapidly but with high intelligibility, and who was able to attain much faster speeds was chosen He read these hundred sentences repeatedly into a Western Electric 640AA microphone connected to the Western Electro-Acoustic Company condenser microphone complement and thence directly to an Ampex 300 tape recorder About five seconds elapsed between sentences On several readings the speaker read just as fast as he could, while still articulating each phoneme approximately correctly On several other readings he slowed a trifle, then more, and so on These tapes were then analyzed on the Sound Apparatus Company sound level recorder, words per minute computed (neglecting, of course, pauses between sentences) and a selection made representing a distribution of speeds of 345, 330, 324, 316, 300, and 280 words per minute, Tests A-F respectively

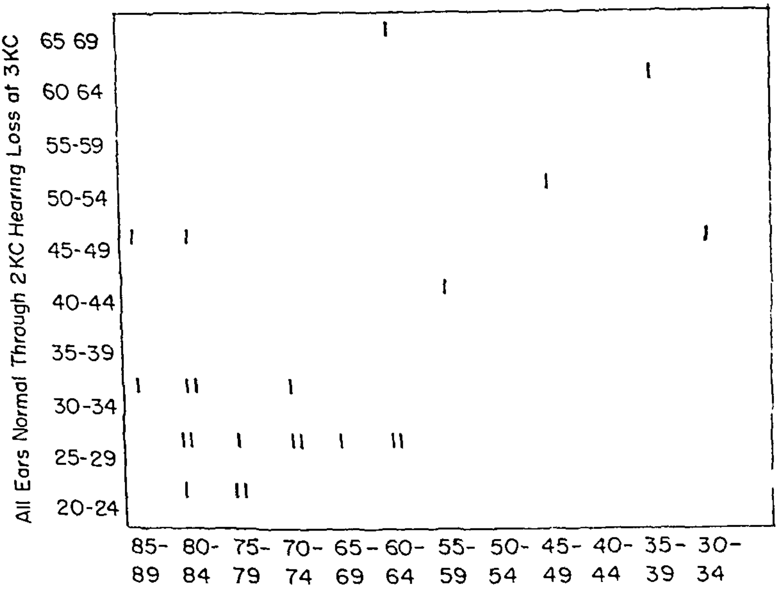
It was ascertained, by administering certain of these tapes to large groups of young men with normal hearing and with an IQ of 100 or better, that test-retest reliability was as good with 50 of these sentences as with 100 Accordingly each tape was split into Forms 1 and 2 of 50 sentences each, so that at each speed an alternate form was available

A group of 25 men as described above was given Forms 1 and 2 of the two fastest speeds No audiometric loss existed greater than 15 db in the 500-3000 cps range The tapes were played 40 db over threshold for the normal ear Table I shows a distribution of per cent intelligibility and other information on these tests

The mean difference between Tests A and B is significant at less than the 01 level, while the mean difference between

10 per cent or more lower than the worst man in the normal-hearing group

It is noteworthy also that in this group of 22 ears only seven scored 80 per cent or better, the highest score yielded being 88 per cent correct, whereas in 36 per cent of the normal-hearing group the scores were 90 per cent correct or better There is very little overlap between these two groups The difference between means is, of course, highly significant



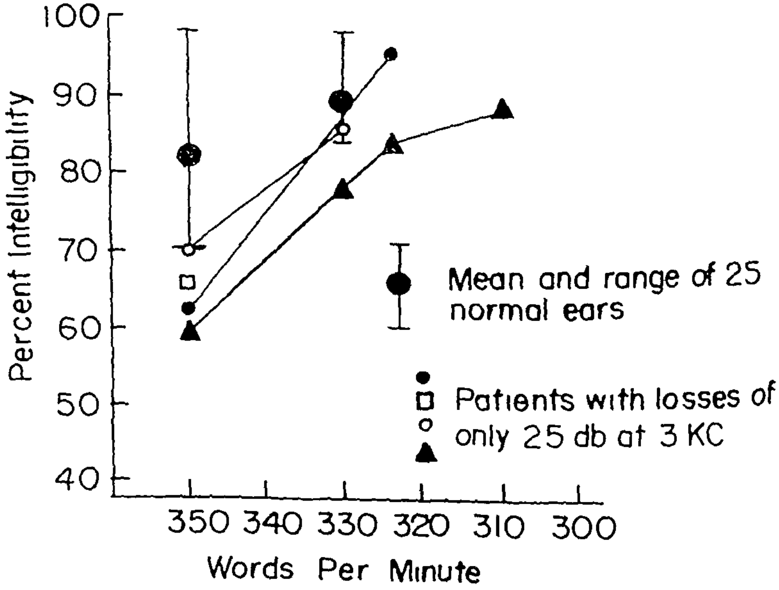
Relation Between Hearing at 3KC and Speeded Speech Reception

Fig 2

The conclusion seems obvious that a normal or near-normal audiogram at 3 kc is essential for high sentence intelligibility if stress is put upon the speech signal through a speed-up

If hearing at 3 kc is essential for full efficiency on speeded speech, hearing at 2 kc should be much more so On another group of 13 men (17 ears) with no loss greater than 15 db at 1 kc, but with losses of at least 20 db at 2 kc, an even more results in Table I with perfectly normal ears and indicates beyond real doubt the irrelevance of 4 kc in this connection

In a group of 19 men (22 ears) with no loss greater than 15 db through 2 kc, but with losses of 20 to 65 db at 3 kc, some real disturbances occurred Mean score was 69 4 per cent correct Low scores were found in four ears even though the loss at 3 kc was only 25 db These four cases are graphed

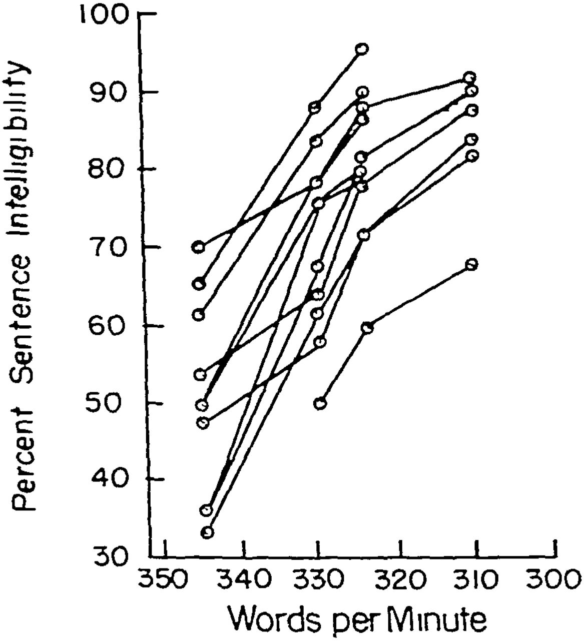


Deteriorating Effect of Speed With Only Minor Loss at 3 KC

Fig 1

in Fig 1 together with the means and ranges for the normal-hearing group These men all scored at least 10 per cent below the mean on Test A, and all below the lowest man on Test A in the normal-hearing group

In Fig 2 is graphed the relation between audiometric loss at 3 kc and per cent correct on the fastest speech tests Of seven ears with losses of 40 db or more, five yielded scores speed. Here, where each ear is its own experimental control, we see that the audiometric pattern, rather than such factors as lowered intelligence, poor language background, etc., can account for the relatively very poor performance on the faster lists.

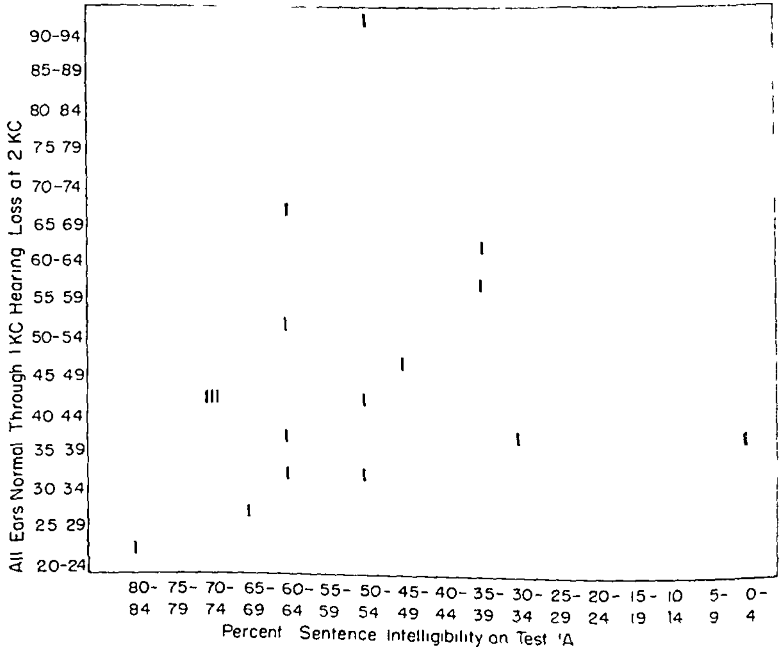


Increase in Intelligibility for Slower Speech for 11 ears Normal through 1 KC but with 20 db or More Loss at 2 KC.

Fig. 4

The mechanism of the decrement for intelligibility in consequence of an audiometric loss at 3 kc can be stated with some confidence. We reject the notion that frequency distortion *per se* is responsible, and accept the simpler explanation that about 15 per cent (84 minus 69.4) of the cues for sentence intelligibility are frequency-dependent upon the 3 kc direct relationship is found Fig 3 shows the per cent intelligibility for these ears as it varies with loss at 2 kc All but one of these ears (and it with least loss) are far below normal performance on Test A

Yet, in spite of rather steep losses at 2 kc (note in Fig 3 that ten of the ears had drops of 40 db or more), these ears

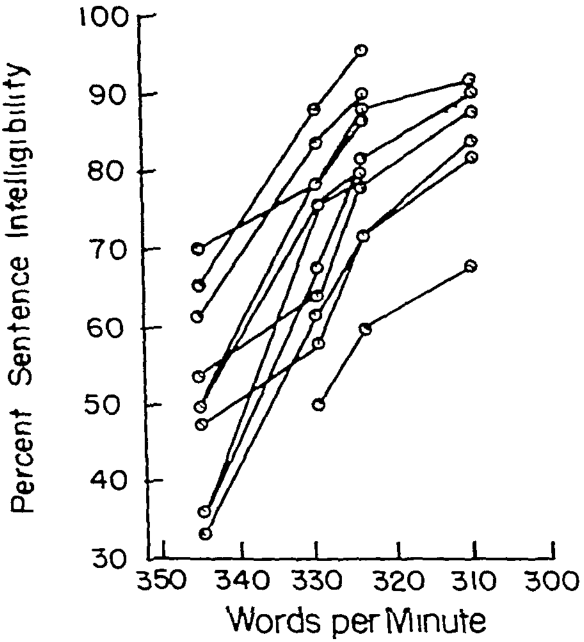


Relation Between Hearing at 2 KC and Speeded Speech Reception

Fig 3

can handle ordinary speech quite well Only four showed a loss of more than 15 db on the PB lists, and all reached a PB maximum of well over 80 per cent rather quickly, furthermore, time was taken to examine 11 of these ears with two or three speeds slower than Test A, and in nearly every case a quick rise to an intelligibility approaching the normal case was found

Fig 4 shows the rapid increase of intelligibility with slower speed. Here, where each ear is its own experimental control, we see that the audiometric pattern, rather than such factors as lowered intelligence, poor language background, etc., can account for the relatively very poor performance on the faster lists.



Increase in Intelligibility for Slower Speech for 11 ears Normal through 1 KC but with 20 db or More Loss at 2 KC.

Fig. 4

The mechanism of the decrement for intelligibility in consequence of an audiometric loss at 3 kc can be stated with some confidence. We reject the notion that frequency distortion *per se* is responsible, and accept the simpler explanation that about 15 per cent (84 minus 69.4) of the cues for sentence intelligibility are frequency-dependent upon the 3 kc region. With slower inputs these lost cues can be compensated for by the heavy frequency-redundancy in speech, but at the word-rate of Test A the speech input is too rapid for this redundancy to operate very efficiently. By eliminating or reducing the possibility of using the redundancy in the speech signal, one can demonstrate the necessity of hearing in the 3 kc region for connected discourse.

Such a result brings connected discourse into approximately the position of phonemes as reported by Fletcher (p. 282 ff). With normal ears and filtering that eliminates all frequencies above 2500 c/s, the following individual speech sounds dropped to 85 per cent articulation or worse: t, k, n, z, ch, s, f, and th. Yet these represent some of our commonest phonemes, often keys to word intelligibility and consequently (though less often) to sentence intelligibility. It does not seem far-fetched to believe that sentence intelligibility should decline 15 per cent when these sounds are missed or confounded, providing one markedly reduces the opportunity for redundancy to operate.

Our two groups with sharp drops at 3 kc and 2 kc, suffice to show the special importance of the higher frequencies when speed stress is put upon the speech signal. For the rather redundant spondees in quiet, Glorig and his associates have shown that the contribution of even the 2 kc region in hearing can pretty well be ignored, while Harris[6] has shown that while 2 kc contributes significantly to PB intelligibility, 3 kc does not. The conclusions of these workers should probably now be modified to re-emphasize the higher frequencies in thinking about the relation of audiometric pattern to speech reception in audiological counseling, compensation formulae, and hearing aid fitting, to name three areas.

To take only one of these areas by way of example, we may inquire whether the mathematical equations relating audiometric loss to the speech reception threshold should not be re-worked, using a variety of distortions in the speech signal, much as found in daily life. We are accustomed to listening to speech through very low-fi systems such as intercoms, public address systems, small radios, etc. We are accustomed to speakers whose initial quality, including speed, is very debilitating We are accustomed to speech in public buildings where noise-masking and reverberation are extreme It would seem wise to include some examples of distortions such as these in any speech reception test purporting to simulate everyday conditions. Speeded speech may serve as a paradigm of these distortions It is more than likely that our explanation for the present data, given above in terms of a reduction in the possibility of utilizing redundancy, can be generalized to the case of an actual loss of redundancy, as in reverberated (by masking) or interrupted speech For all these conditions, the higher frequencies should perhaps receive a tilt upward in our reflections on the matter

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APPENDIX A

Form 1

1 A grass roots movement is likely to start
where there is wind with the soil very short with people

2 What drops from trees in the fall?
rain leaves birds woods

3 The game of horseshoes can be won with
steel hoofs ringers ties

4 What shines in the sky at night?
sun polish stars darkness

5 Ice formed at the edge of the roof by dripping water is called
heavy frozen icicles sheets

6 How much is two times three?
two three five six

7 In the fall the most popular team sport in America is
wrestling New York Yankees **football** October

8 Which coin is half of a dime?
nickel dime half-dollar dollar bill

9 A man's routine walk may be considered
concrete sidewalk **a constitutional** the dog

10 Which is a city in New England?
Trenton **Boston** London Maine

11 A plane lighting on the deck of a carrier is most likely to be
a pigeon a pilot **a Navy plane** brilliant

12 Which man was an American general?
Grant Wilson Napoleon Joan of Arc

13 Some inexpensively bound books issued periodically are called
authors **magazines** news print

14 What color is the grass in the summer?
mown brown hot **green**

15 Cars that travel directly between mountains use
waterways high altitude high octane gasoline **cables**

16 The country north of the United States is
Canada Mexico North Dakota Norway

17 A pencil sharpener is very useful in
turning **the office** shavings pointed

18 What do we look at on a watch to tell time?
eyes clocks **hands** ears

19 A woman wears pumps
by hand water **on her feet** clothing

20 Which is a yellow citrus fruit?
apple lime **lemon** pear

21 Glass lenses worn over the eyes are called
cameras clear windows **spectacles**

22 What does a secretary use to type letters?
typewriter envelope alphabet notebook

23 A very harsh solution called lye
on the bed is to tell falsehoods **will burn the skin** sleeping dog

24 Bright is to dim as loud is to
light **soft** sound hard

25 A square ring encloses
impossible geometry wedding **boxers**

26 How much is five minus two?
two **three** five ten

27 One of the races to which people belong is
running **Yellow** horses contest

28 If ears are for hearing, the nose is for
seeing hearing tasting **smelling**

29 The largest thing living in the ocean is a
ocean liner **whale** fish iceberg

30 The name of a European country is
France Paris Berlin Uruguay

31 A coat worn by a man is likely to be made of
oil mink paint **tweed**

32 How much is one plus three?
one two three **four**

33 A bear which lives near the North Pole is
black penguin **polar** Arctic

34 Name a green leafy vegetable
tree corn **spinach** grass

35 A large stringed instrument in an orchestra is a
bass drum tennis racket tuba **bass violin**

36 If cats have kittens what do dogs have?
bark meow **puppies** fleas

37 To break which kind of record brings fame
testimony phonographic files **athletic**

38 What do we wear on our feet when it rains?
socks mittens **rubbers** raincoats

39 To sign a letter is to add
a plus Zodiac **one's name** billboard

40 Which farm animal is a bird?
horse **chicken** dog eagle

41 Some heavenly stars may be seen better
in old movies with five points **with a telescope** quality

42 What has four wheels and a motor?
bicycle wagon **automobile** motorboat

43 A forward pass is made by throwing a
between mountains not bidding proceed **football**

44 Complete the rhyme One two, button my
shirt three **shoe** lip

45 A new broom sweeps
floor **clean** money straw

46 Trains run on railroad
model **tracks** locomotive stations

47 Many voices singing together make a
choir song party duet

48 What green vegetable goes with carrots?
rabbits blue butter **peas**

49 A new pipe must be broken in
by the smoker steel tobacco water

50 Underline the largest farm animal
elephant pig **horse** whale

Form 2

1 A very expensive American made car is
the Ford Detroit money **Cadillac**

2 What month rhymes with remember?
September July ember Mother's Day

7 In the fall the most popular team sport in America is
wrestling   New York Yankees   **football**   October

8 Which coin is half of a dime?
nickel   dime   half dollar   dollar bill

9 A man's routine walk may be considered
concrete   sidewalk   **a constitutional**   the dog

10 Which is a city in New England?
Trenton   **Boston**   London   Maine

11 A plane lighting on the deck of a carrier is most likely to be
a pigeon   a pilot   **a Navy plane**   brilliant

12 Which man was an American general?
Grant   Wilson   Napoleon   Joan of Arc

13 Some inexpensively bound books issued periodically are called
authors   **magazines**   news   print

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mown   brown   hot   **green**

15 Cars that travel directly between mountains use
waterways   high altitude   high octane gasoline   **cables**

16 The country north of the United States is
Canada   Mexico   North Dakota   Norway

17 A pencil sharpener is very useful in
turning   **the office**   shavings   pointed

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eyes   clocks   **hands**   ears

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by hand   water   **on her feet**   clothing

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25 A square ring encloses
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two   **three**   five   ten

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45 A new broom sweeps
floor **clean** money straw

46 Trains run on railroad
model **tracks** locomotive stations

47 Many voices singing together make a
choir song party duet

48 What green vegetable goes with carrots?
rabbits blue butter **peas**

49 A new pipe must be broken in
by the smoker steel tobacco water

50 Underline the largest farm animal
elephant pig **horse** whale

Form 2

1 A very expensive American made car is
the Ford Detroit money **Cadillac**

2 What month rhymes with remember?
September July ember Mother's Day

3 Ground in which plants are grown is called
soil grain electrical circuit support

4 Which animal walks on two legs?
centipede **chimpanzee** dog tulip

5 The larger body of Congress is called
House of Representatives politics legislation grave

6 Name a river in Africa that flows north
Rhine **Nile** Mississippi Congo

7 The mouth organ is known otherwise as
anatomy teeth **harmonica** disease

8 A penny saved is a penny
spent pinched **earned** bank

9 Five large inland lakes are called
Great Lakes continental Hudson Bay Great Salt Lake

10 Name a European capital city
Washington **Paris** Hartford Venice

11 Count by twos Two, four, six
four number count **eight**

12 What is the Gulf of Mexico?
body of water Spanish speaking country game gasoline

13 A club carried by a policeman while on duty is a
social Policeman's Benevolent Association pistol **nightstick**

14 If I mix beans and corn I get
pizza Sukiyake **succotash** Mexican corn

15 Precipitation which on striking the ground may bounce is called
chemical snow ball **hail**

16 A stitch in time saves
thread **nine** clothes clock

17 The kind of pitch which involves a catcher is to be found in
evergreen trees tar **baseball** slope

18 If you had trouble seeing you would wear
a hat **eyeglasses** better light hearing aid

19 The term Silent Service applied to military forces means
quiet Army **Submarine Force** defense

20 Up is to down as high is to
sky kite elevator **low**

21 Bill is the name for a statement of
bird's beak a nickname poster **money owed**

22 A white fluid that you drink is
snow **milk** kerosene coffee

23 The flag of the United States contains red and white
stars **stripes** forty eight thirteen

24 Some one a year older than twelve is
eleven **thirteen** fourteen twelve

25 Which country has a female monarch
Queen Juliana **England** never King

26 People who lived in ancient Rome spoke
Greek Italian **Latin** Sanscrit

27 Quarters which are small and made of metal are called
small pieces of meat **25-cent pieces** one-fourth rooms

28 Pedals are used when riding a
flower **bicycle** horse rose

29 The type of match most likely to be carried by a person is
a book-match wrestling identical items marriage

30 Name a cold country in the northern hemisphere
Iceland Antartica Spain Hawaii

31 Hands which travel in circles are found
wrists helping **on a clock** shaking

32 How many inches in a foot?
three shoes **twelve** socks

33 Mail delivered to your door is handled by
boy babies chain open **postman**

34 How many two cent stamps in a dozen?
two **twelve** twenty-fours thirty-six

35 A large statue in New York harbor is
Statue of Liberty the Empire State Building ocean liner law

36 Bible is the name of a
book religion noise water

37 China is a country in the Far
away **East** large enemy

38 Underline the insect that flies and stings
ant robin moth **wasp**

39 A 3 dimensional object with equal sides is
geometry in inches **a cube** barrel

40 What meal is one likely to eat at seven in the morning?
shave **breakfast** television dinner

41 A slight wavy motion on the surface of water is called
hair do gesturing surf **ripple**

42 The last car in a freight train is called
locomotive convertible box car **caboose**

43 Material used to break up rock is called high
cliff **explosives** ball jump

44 Which man is a Navy officer?
colonel **admiral** Wave policeman

45 Men employed by a city to put out fires are called
firebugs commissioners smoking **firemen**

46 What holiday falls on a Thursday in November?
Easter Labor Day **Thanksgiving** Monday

47 A figure with four equal sides is a
equilateral triangle straight line geometry **square**

48 How much is four divided by two?
one **two** three four

49 The number of zeros in one thousand is
three two thousand fraction money

50 Which liquid is not a beverage?
milk gasoline lemonade beer

U S Naval Medical Research Laboratory, New London, Conn

14 Broad Street, New London, Conn

INTERNATIONAL POSTGRADUATE COURSE IN RECONSTRUCTIVE SURGERY OF THE EXTERNAL NASAL PYRAMID AND NASAL SEPTUM

An international course in Reconstructive Surgery of the External Nasal Pyramid and Nasal Septum will be given in Mexico City July 4-15, 1960, under the auspices of the University of Mexico Medical School and its Postgraduate Department, in cooperation with the American Rhinologic Society, and under the direction of Dr Andres Bustamante Gurria, of Mexico City The guest professor will be Dr Maurice H Cottle, Chicago Medical School He will be assisted by a faculty of otorhinolaryngologists from the United States and Mexico

Class membership is limited Apply for registration information to Dr Robert M Hansen, Secretary, American Rhinologic Society, 1735 North Wheeler Ave, Portland 17, Ore

FIBROUS DYSPLASIA WITH MONSTER-LIKE DEFORMITY*

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and
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Review of the literature on fibrous dysplasia (Lichtenstein and Jaffe,[1] Weinmann and Sicher,[2] Brunner,[3] and Hoboeck[4]) leaves one in the dark as to the true nature of the disease Numerous etiological factors have been suggested congenital anomaly of the bone forming mesenchyme, aberration of ossification in membranous bones of the skull, metabolic disturbance, trauma with abnormal local response, disturbed vascularization, hypothalmic lesion, endocrine dysfunction, but none appears to have solid, factual basis Some authors (Stamm,[5] Davis[6]) include under "fibrous dysplasia" such conditions as osteoma, osteoid fibroma, ossifying fibroma, but others (Thoma,[7] Weinman and Sicher[2]) oppose strongly this view and consider fibrous dysplasia a distinct entity

The salient clinical features of the disease are onset in childhood, slowly progressive course, and standstill on reaching maturity One bone (monostotic form) or many bones (polyostotic form) may be affected, usually on one side of the body Albright's disease, in which skeletal lesions are associated with cutaneous hyperpigmentation and precocious puberty, possibly represents a more severe form of the illness

Lack of unanimous opinion as to the pathologic criteria by which fibrous dysplasia may be labelled as such, makes it rather difficult to ascertain the incidence of the disease and of its various forms Involvement of a single bone, however, seems to be much rarer than that of multiple bones

During the past three years we have observed six cases of fibrous dysplasia three were monostotic, all affecting the maxilla, two were polyostotic, affecting skull and facial bones on the same side (one of these cases was described previously by Rosen, et al[8]) The sixth case was one of Albright's disease

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*From the Department of Otolaryngology Beilinson Hospital General Federation of Labor in Israel Sick Fund Petah Tikva Israel

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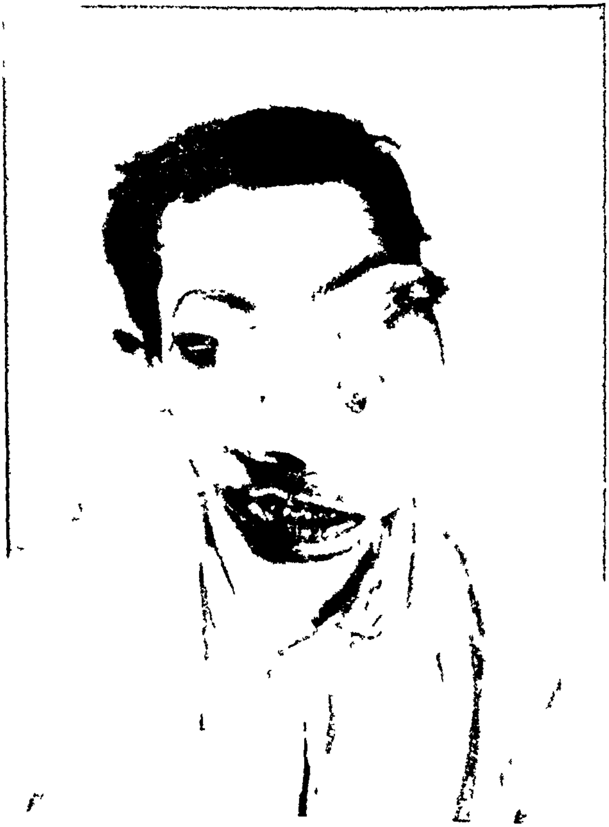

Fig 1 A monstrous deformity resulting from fibrous dysplasia

A rather unusual example of the monosteotic variant affecting the left maxilla will be described

Case 1 A male patient, aged 25 born in Iran and residing in Israel for the past five months, was seen for the first time in May, 1956 His family history was essentially negative

At the age of 14, a small prominence appeared over the left nasal wall The swelling increased gradually in size, expanding the face and dis placing the eyes sideways Diplopia and nasal obstruction supervened and the patient had to keep his mouth constantly open The mass ceased to grow at the age of 20 and remained stationary since A year prior to admission the patient noticed a superficial sore over the prominent part of the tumor which failed to heal

The monstrous appearance excluded the patient from social contacts, and he had to stop school attendance, nevertheless he succeeded in learning to read and write (both his sisters are illiterate) Since his arrival in Israel he was confined to a back room going out only in the evening the left half of his face covered with a shawl

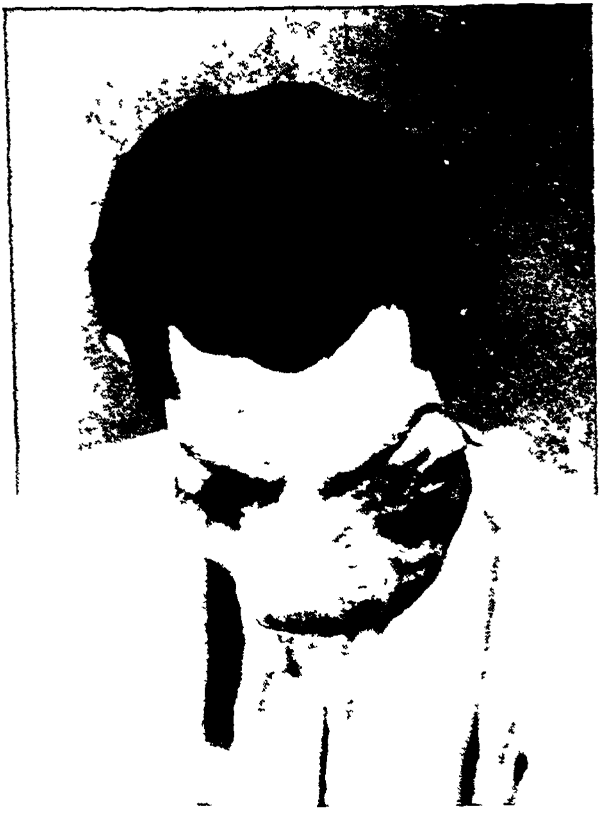

Fig 2 A monstrous deformity resulting from fibrous dysplasia

On seeing the patient for the first time the reaction was mostly one of shock repulsion and unbelief The patient's condition was poor, but except for the head he was normally and proportionately developed The head was monstrous (see Figs 1 2, and 3), the eyes were pushed widely apart looked sideways and bulged as in a lizard Papillary distance was 12 cm Eyeball motility acuity of vision and eyegrounds were normal The nose was displaced to the right, and the left nostril was occluded A pressure sore was observed over the left side of the nose Upper dentition was distorted, and hard and soft palates were depressed markedly, leaving hardly any room for the tongue Respiration was disturbed, and stertorous sounds were produced during sleep The nasopharynx was occluded, and examination of the larynx was not feasible Examination of the neck chest, abdomen and extremities showed no pathological findings

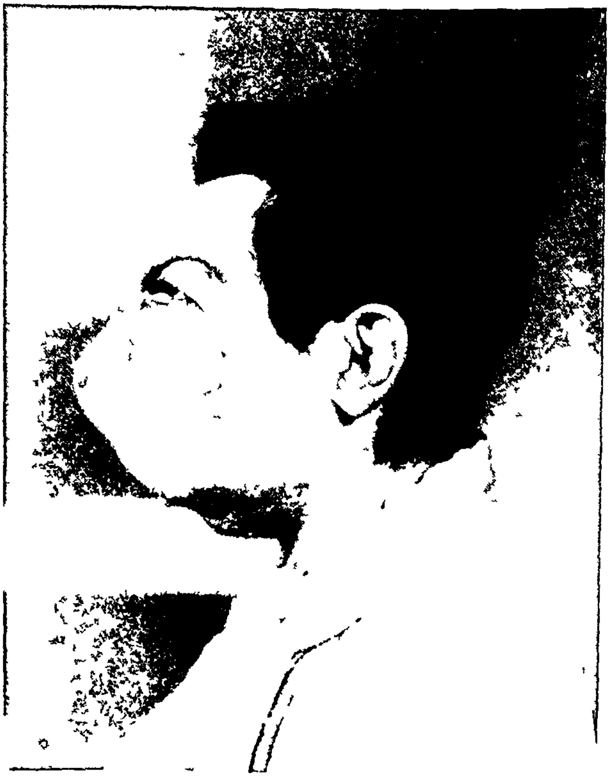

Fig 3 A monstrous deformity resulting from fibrous dysplasia

Laboratory studies blood count and morphology E S R and urinanalysis were normal The Kahn Wassermann reaction was negative Blood chemistry urea glucose electrolytes total proteins albumin and globulin, cholesterol calcium phosphorus, potassium alkaline phosphatase as well as urinary excretion of calcium on a regular hospital diet, all were within the normal limits I^{131} uptake in the thyroid gland was elevated (38 per cent after two hours and 60 per cent after 24 hours), urinary excretion was 27 per cent after 24 hours

Radiological studies of the chest the gastrointestinal and the urinary tracts revealed no pathology X ray examination of the skull showed a well circumscribed amorphous mass the size of a fist replacing the normal structure in the midpart of the facial skeleton (see Figs 4 and 5) The orbital roofs were pushed upward and thinned out It appeared almost certain that the left maxilla was the only bone involved

We decided to perform an extirpation of the diseased maxilla, hoping that when followed by a plasty, the patient's condition would become bearable The operation was planned as follows

1 Tracheotomy under local anesthesia (intubation was not feasible)

2 General anesthesia through an endotracheal tube

3 Ligation of the left external carotid artery

4 Excision of the badly nourished thinned out skin over the left maxilla with preservation of as much cutis as deemed necessary for a lining of the resulting cavity

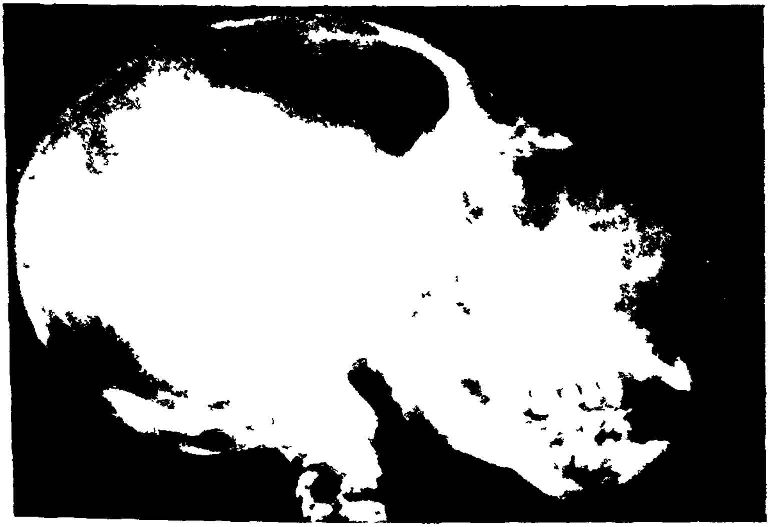

Fig 4 Amorphous mass in the midpart of the facial skeleton

5 Maxillectomy, total or partial

6 Operation cavity to be lined with adjacent skin or with a skin graft

7 The fate of the left eye to be decided at the end of the operation

The operation did not proceed accordingly The usual premedication exerted a marked depressive effect patient's breathing became deep, slow and irregular and his complexion livid and cyanotic An emergency tracheotomy was done and oxygen and blood were given A mixture of nitrous oxide and oxygen was then administered through an endotracheal tube The left external carotid artery was ligated

The surgical approach was through a modified Moore's incision simultaneously excising the sore and the adjacent atrophic skin A thin bony shell was exposed and brisk bleeding from periostal vessels appeared On removing the bony shell a huge cavity was opened and found to be filled with blood and a soft spongy substance Bleeding was uncontrollable, and tablespoons were used to evacuate the contents as rapidly as possible After a few minutes the whitish bony wall of the cavity could be seen Hot saline sponges were put in and this partly controlled the bleeding, however, the blood pressure dropped suddenly, and respiration and cardiac beat stopped Artificial respiration, massive blood transfusion and intravenous noradrenaline were of no avail

On postmortem examination a large cystic structure was found with a

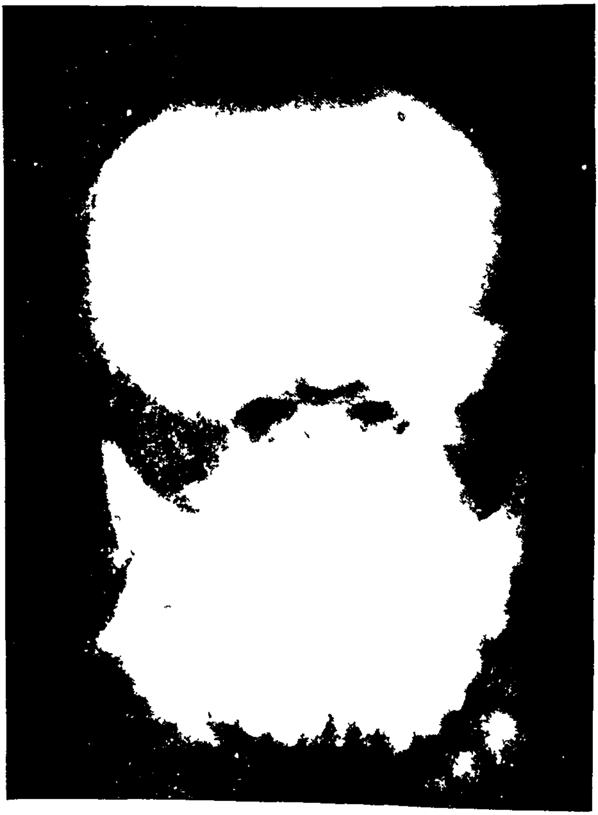

Fig 5 Amorphous mass in the midpart of the facial skeleton

bony wall discrete from the surrounding bone (see Fig 6) The cyst occupied a paracentral position, its center situated approximately where the left maxillary sinus should have been The diameter of the cavity was about 10 cm It displaced the paranasal sinuses and the nasopharynx, reaching posteriorly the bodies of the cervical vertebrae The left orbital roof was displaced superiorly so that an impression was made in the inferior aspect of the left temporal lobe In no place did the mass actually penetrate into the cranial cavity, but the floor of the anterior fossa was paper thin in some places The lungs were soft and airy The heart was drop shaped and showed a few petechial hemorrhages in the auricular pericardium The gastrointestinal tract, liver, gallbladder, bile ducts, spleen, pancreas, adrenals, kidneys, urinary passages and abdominal vessels were normal There were no other bony deformities

Microscopic examination showed areas of fibrous connective tissue with foci of newly formed, primitive bone tubercula Osteoblasts were seen in the vicinity of the primitive bone

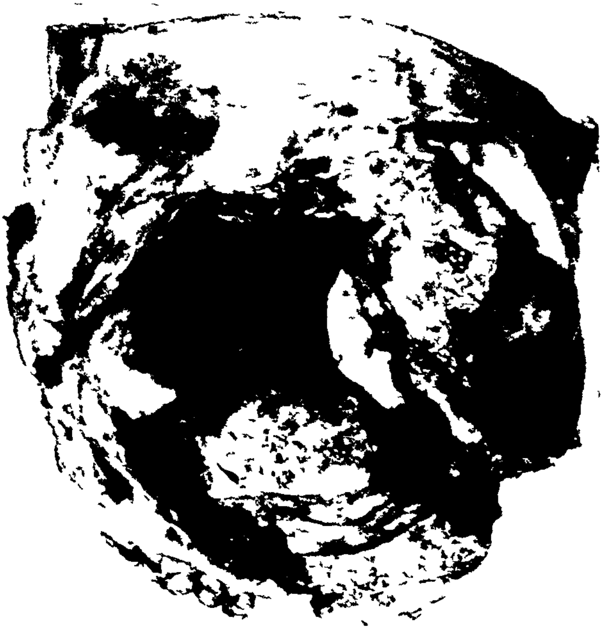

Fig 6 Large cystic cavity displacing the surrounding structures

COMMENT AND SUMMARY

An unusual example of the monostotic form of fibrous dysplasia in a 25-year-old male is described It involved the left maxilla, resulting in a monstrous external deformity No other lesions were found in the body, and laboratory studies revealed normal values except for an elevated uptake of I^{131} At operation respiratory depression and hemorrhage resulted in irreversible cardiac arrest

Retrospectively it seems to have been a mistake to continue the operation after completing tracheotomy The cause of the respiratory depression should have been more carefully investigated and the intervention postponed or perhaps abandoned

It appears plausible that deformities like the one described here served as inspiration for the mythological concept of a monster, as well as for some works of art by Hieronymus Bosch and by Pablo Picasso, among others

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POSTGRADUATE COURSE

The next postgraduate course in Laryngology and Broncho-esophagology to be given by the University of Illinois College of Medicine is scheduled for the period April 4 to 16, 1960 The course is under the direction of Dr Paul H Holinger

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Ill

A MODIFIED TECHNIQUE OF RHINOMETRY

With a Preliminary Note on the Effect of Nasal Decongestants Administered Orally [*]

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New Orleans, La

Determination of the size of the effective nasal airway has long presented a challenge to rhinologists It presents a particular problem to modern rhinologists because of the popularity of decongestants for oral administration, which are now used without any real knowledge of the degree of their effect on the nasal mucosa

Rhinometric methods previously used are open to the criticism that they frequently cause distortion of the size of the nares as well as reflex mucosal congestion Indirect methods include measurements of the gross strength of the respiration through one or both nares, or an analysis of the shape and size of spots made by breathing on a mirror, as recommended by Zwaardemaker [1] These methods have the advantage of simplicity but lack the accuracy of more direct methods, which include *1* Courtade's[2] water manometer, an appliance attached externally and unilaterally to the nares, *2* the mask introduced by Reverchon and his associates,[3] to prevent deformity of the external nares in the course of the examination, *3* the mask devised by Uddstromer[4] for the exact, separate measurements of the air flow through the nasal and oral compartments

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None of these methods meets the obvious need for a technique of rhinometry which will be both accurate and practical in the evaluation of the effects of surgical measures and medications on the the nasal airway We began our attempt to devise such a method by a careful review of the numerous procedures previously recommended for this purpose We also bore in mind the principles of nasal permeability worked out by Uddstromer[4] and Proetz[5] We agree with such observers as Stoksted and Nielsen,[6] and Seebohm and Hamilton[7] that the electronic devices available in medicine today permit more convenient and more accurate objective measurements of the nasal airway than any of the devices previously utilized We have found the pressure transducer (strain gauge) and the direct writing recorder especially useful

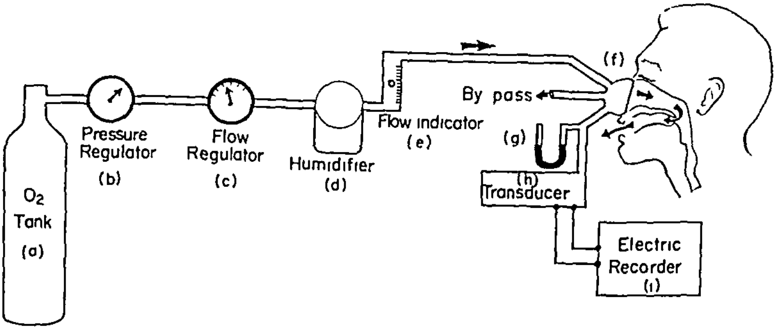

Fig 1 Equipment for rhinometry A—Oxygen source at 10 liters per minute B—Pressure regulator (National cylinder style 6484 C—Flow regular 0 - 15 liters per minute (National Cylinder Gas Model 211-1 D—Humidifier (National Cylinder Gas Model 24703) E—Flow meter (National Cylinder Gas Model 24980) F—Nasal oxygen mask (Boothby-Lovelace-Bulbulian manufactured by Linde Air Products Company) and improvised by-pass valve with lumen calibrated in centimeters H O G—Water Manometer (simple U-tube) H—Strain gauge (Stathan No P23B) I—Electric recorder Weston Model 6701 1 millivolt calibrated in centimeters of water (the 10-inch scale equivalent to 10 cm H O) The following should be noted on wiring diagram Standardizing control (3000 ohm 2 watt potentiometer) strain gauge control (3000 ohm 2 watt potentiometer) 6 volt battery to energize strain gauge 1 5 volt battery to standardize control

DESCRIPTION OF TECHNIQUE

The technique of anterior rhinometry which we employ is a modification of the posterior rhinometric technique devised by Seebohm and Hamilton[7] By their method, a double-lumen

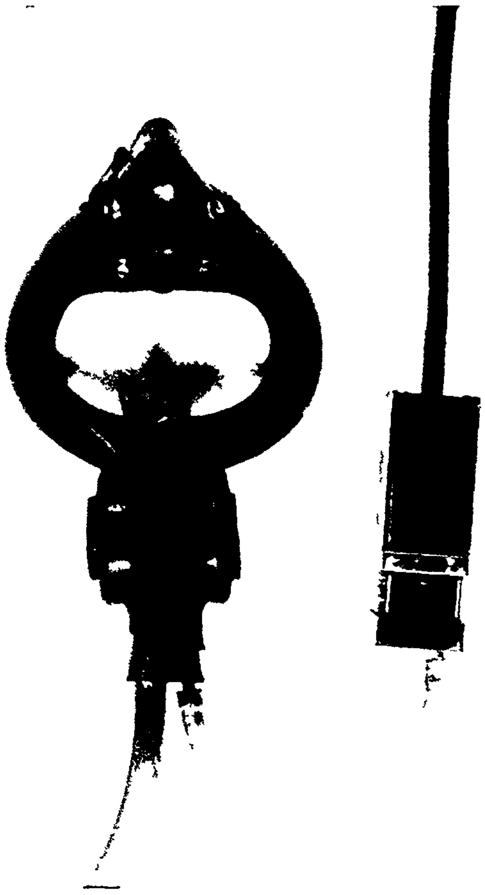

Fig 2 Mask and transducer used in rhinometry

tube is introduced into the mouth, and a measured flow of oxygen is passed into one tube The oxygen flows on into the oropharynx, whence it can alternately pass *1* into the nasopharynx, or *2* out of the by-pass oral tube to a transducer The pressure measurement thus secured is a relative inverse index of the size of the nasal airway This recording method involves feeding the electrical output of the transducer into an amplifier and direct writing recorder In testing this method, we found an undesirable feature, that the mouthpiece used very frequently stimulates the gag reflex, therefore, we modified this method by replacing the mouthpiece with the Boothby-Lovelace-Bulbulian nasal oxygen mask, to which a pressure transducer is connected. The mask is a satisfactory substitute for the mouthpiece, since we do not use intranasal topical applications. With this alteration, an efficient method of anterior rhinometry becomes available. The apparatus necessary is shown in Figs. 1 and 2, and the wiring diagram in Fig. 3.

Warm, humidified oxygen is fed into the mask at 10 liters per minute; it passes into the nose and nasopharynx and then out through the mouth. The fall in pressure within the mask, and, therefore, within the nose, is transmitted to the pressure transducer connected to the mask. The output of the transducer is fed to the recorder, and the number obtained in millivolts is readily converted to centimeters of water pressure. The final value is the index of obstruction of the nasal airway.

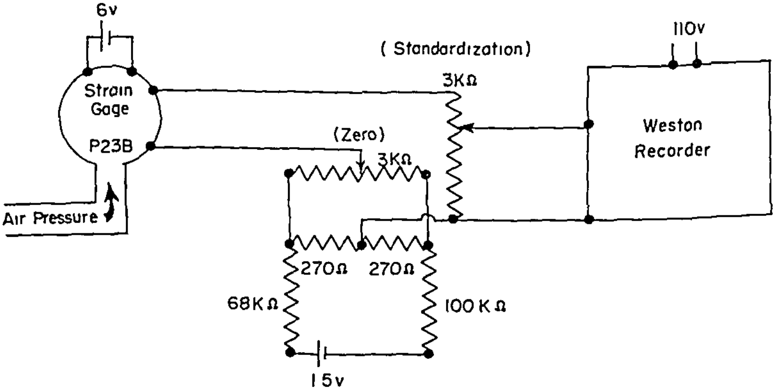

Fig. 3. Wiring diagram of equipment.

EXPLANATION OF OPERATION

Since air is a compressible fluid, the electrical analogue of the fluid system can be used for the explanation of this method (see Fig. 4).

The circuit which we employ is a constant current system, that is, it is a system in which, if the internal source voltage and resistance remain constant, the current in the circuit also remains constant in spite of changes exterior to the source

This is evident in Fig 5, in which the current in the circuit is

$$I\ (\text{Amps}) = \frac{\text{Eb (volts)}}{\text{Rs plus Rn (ohms)}}$$

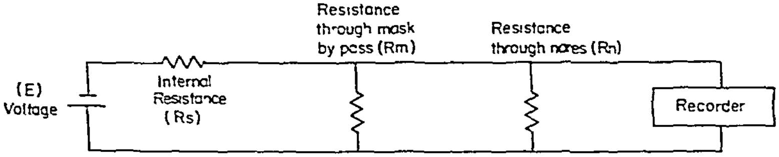
Resistance through mask by pass (Rm)
Resistance through nares (Rn)
(E) Voltage
Internal Resistance (Rs)
Recorder

Fig 4 Electrical analogue of Fig 1

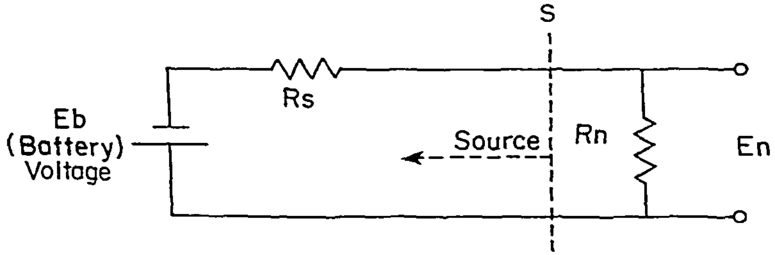

Fig 5 Demonstration of voltage En as equal to Ik Rn Rs—internal resistance S—line dividing circuit from source Rn—resistance equivalent to nares En—pressure drop equivalent to drop in nares

If Rs is very much greater than Rn, then Fig 5 is reduced to

Ik (a constant) $= \frac{\text{Eb}}{\text{Rs}}$ (assuming Eb plus Rs are held constant)

Our fluid analogy replaces Rs by an oxygen flow regulator, Rn by the nasal resistance to flow, and Eb by high pressure oxygen It will be observed in Fig 5 that the voltage En is equal to Ik Rn Since this is true, any change in Rn will cause a change in En, which in analogy is a pressure drop from the mask to the nasopharynx This pressure drop is transmitted to a strain gauge or pressure transducer, the output of which, when applied to the electric recorder, gives

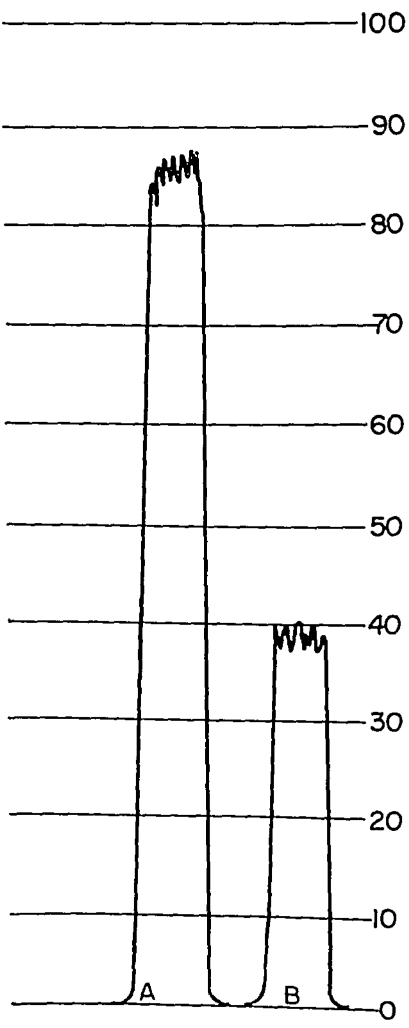

Fig. 6. Example of before and after recording. A represents 8.6 cms of water pressure. B represents (3.8 cms water) improved nasal airway.

a deflection (in millivolts) that represents nasal resistance (when converted to centimeters of water).

Because the fluid system is a non-linear device, the pressure obtained is also a non-linear function of the nares opening. Stoksted and Nielsen[illegible] have amply illustrated the factors involved in deriving the non-linear relationship. The system which we are describing does not require the refinements necessary in their system, because of the method of measurement Inspiration with the mouth closed would be analogous to another voltage source, the lungs, in series with a constant current source It was, therefore, found simpler to have the patient close the glottis, if he could, or breathe gently through the mouth, neither of which actions would significantly complicate the nasal air-flow system The resulting negative and positive pressure on quiet respiration through the mouth in

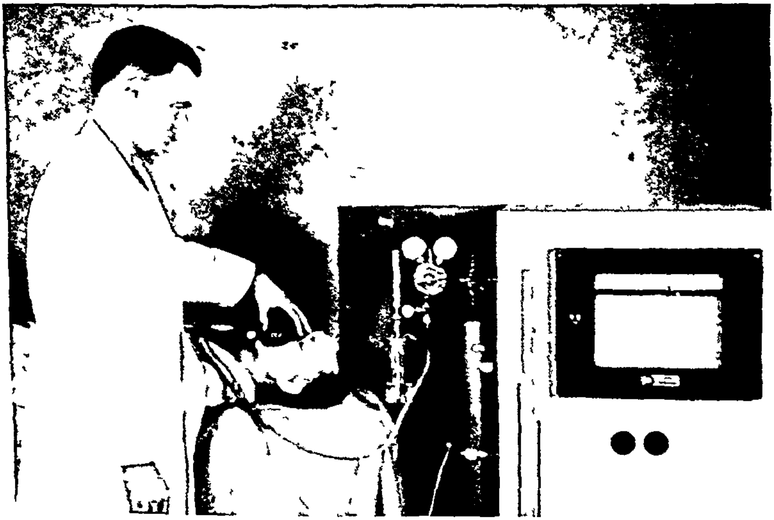

Fig 7 Position of patient and operator during test

patients unable to hold their breath produces no undulations at all, or a minimum too slight to need consideration Any undulations from respiration shown on the graph (see Fig 6) are averaged as a straight line, and the reading is taken accordingly

A by-pass opening is necessary on the oxygen mask If it is not provided when the nose is completely obstructed, the flow rate at the source would be altered, and the recorder would stop at full scale on the paper The nasal mask used (see Fig 2) has provisions for using any one of three different calibrated holes to allow the oxygen to by-pass a completely obstructed nose

CLINICAL APPLICATION (SEE FIG 7)

The patient, whether his nasal obstruction is of infectious, allergic, vasomotor, endocrine or psychic origin, lies recumbent for ten minutes before the test, so that he will be fully relaxed, and maximum passive mucosal congestion will occur He is shown how to open his mouth and allow the oxygen from the nasopharynx to escape into the oropharynx as he breathes quietly through his mouth

With the oxygen input shut off, the recorder is set with the strain gauge control at zero, this setting corresponds with the largest diameter of the nares The flow regulator is turned on to 10 liters of oxygen per minute The nasal outlet of the mask is obstructed, to stimulate a completely congested and obstructed nose, the oxygen in the mask must, therefore, escape through the by-pass valve The standardization control is now adjusted until the recorder reads 1 mv, that is, full scale As this takes place, a U water tube in parallel with the transducer air hose (see Fig 1) will be noted to be 10 cm in height, as the diameter of the by-pass orifice has been made to produce such a pressure

During the tests to determine the effectiveness of whatever medication is used on a partly open nose, the physician closes the by-pass with his finger, thus causing all oxygen to pass through the nose As the calibration of the recorder is known, and full scale on the recorder is equivalent to a given pressure in centimeters of water, any reading less than full scale is that ratio or percentage of full scale pressure calibration (see Fig 6)

PRELIMINARY RESULTS

To test the accuracy of the apparatus, ten patients, all presumably normal, were tested unilaterally, the other side of the nose being taped to simulate obstruction Ten tests were conducted on each patient, and in all instances repeated trials on the same patient produced consistent readings

To date, and preliminary to the study of a later, larger series of tests, we have investigated the effect on 12 patients, of each of four sympathomimetic agents commonly used in compounded pharmaceutical preparations (see Table I) A baseline reading was secured on each patient before the medications were given, always on an empty stomach, and a second reading was secured an hour later At this time we are reporting only the gross objective and subjective results In a later report, the variations in response will be presented As Table I shows, the number of cases in which objective

TABLE I

Objective (Rhinometric) and Subjective Results of Nasal Decongestants Administered Orally to 12 Patients

Drugs	Rhinometric Objective Results Airway			Subjective Results		
	Increased	Decreased	Unchanged	Better	Worse	Same
Ephedrine (Ephedrine Sulfate)	9	1	2	5	3	4
Sudafed (Pseudoephedrine HCl)	4	5	3	2		10
Neosynephrine (Phenylephrine HCl)	10		2	6	1	5
Propadrine (Phenylpropanolamine HCl)	8	3	1	4	3	5
Total patients tests	31	9	8	17	7	24

improvement occurred is considerably larger than the number in which the gross effects were great enough to impress the patient, though the figures are too small to be of any statistical significance

COMMENT

In addition to its simplicity of application, the technique described has been found to have a number of other advantages

1 It provides a permanent record of the original state of the nasal airways and of the results of medical or surgical treatment

2 The technique is an improvement over the method of which it is a modification, and in which a gag-stimulating air stream is directed against the palate and oropharynx Reflex closing of the naso-oropharyngeal communication is infrequent in the new technique The oxygen in the nose can, therefore, escape through the nasopharynx and into the mouth, and a valid estimate of the nasal airway can be obtained on the recorder

3 The 10-inch deflection of the recorder provides a sufficiently large scale for measuring the slightest changes in the caliber of the nasal passages

4 Pulmonary influences are relatively unimportant

5 This is a relatively inexpensive clinical test, which permits the testing of large numbers of patients in short periods of time The nasal air mask input is tolerated by adults and by older children, many of whom were inconvenienced by the mouthpiece used in the original test and would not cooperate in it Young children, of course, do not cooperate well in any test

6 Reflex vascular changes are minimized by the use of warm humidified oxygen

7 Since quiet respiration through the mouth does not alter the results, patients who have difficulty holding their breath while maintaining a relaxed soft palate can be permitted to breathe through the mouth

SUMMARY

A method of anterior rhinometry is described in which the improvement in the nasal airway can be conveniently measured before and after treatment with orally administered nasal decongestants The method is simple to use, causes minimal discomfort to the patient, and provides a permanent record of changes in the airway before and after medication or surgery

Preliminary tests show that the oral administration of nasal decongestants does increase the size of the nasal airway, although the effects may not be sufficient for the patient to realize it grossly

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PROCEDURE OF "INTERPOSITION" FOR OTOSCLEROTIC DEAFNESS

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Surgery for deafness caused by ankylosis of the stapes has been steadily improving Although procedures such as stapes mobilization, stapedectomy, and fenestration of the labyrinth, have been done since the end of the 19th Century, it is really due to chemotherapy and the antibiotics that these operations have been successful in recent years

Sourdille[19] worked on the fenestration operation for many years and in 1927 was able to demonstrate patients operated upon successfully Lempert[10] refined the procedure and described his one-stage operation The extraordinary development of the operation, thanks to the American school, is well known

Although fenestration of the posterior labyrinth may still be used as a palliative procedure, it is crude compared to the latest possibilities offered by surgery for deafness The patient, after fenestration, is left with a cavity which needs constant cleaning and care The ossicular chain is interrupted, the posterior labyrinth is opened, and the hearing cannot be expected ever to be normal again

In 1952, Rosen[16] revived stapes mobilization, a procedure widely used in the 19th Century by the French school, particularly by Boucheron[3] in 1888, Miot[11] in 1890, and E J Moure[12] in 1890, and later discontinued because of the risk of infection Since 1952, however, there have been innumerable publications throughout the world, dealing with thousands of cases attesting to the interest aroused by Rosen's publication

Although stapes mobilization is a more physiological procedure than fenestration of the posterior labyrinth, it still is far from perfect There are many cases in which the anky-

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losis is too far advanced to permit efficient mobilization This difficulty has in the past been overcome by various procedures directed at the footplate, thereby avoiding fracture of the crura For example, Goodhill[6] uses a vibrator, Heermann[8] small chisels and hammers designed to attack the sclerosed articular junction In 1956, Portmann and Claverie[13] described a microdrill which has since been adopted by others,

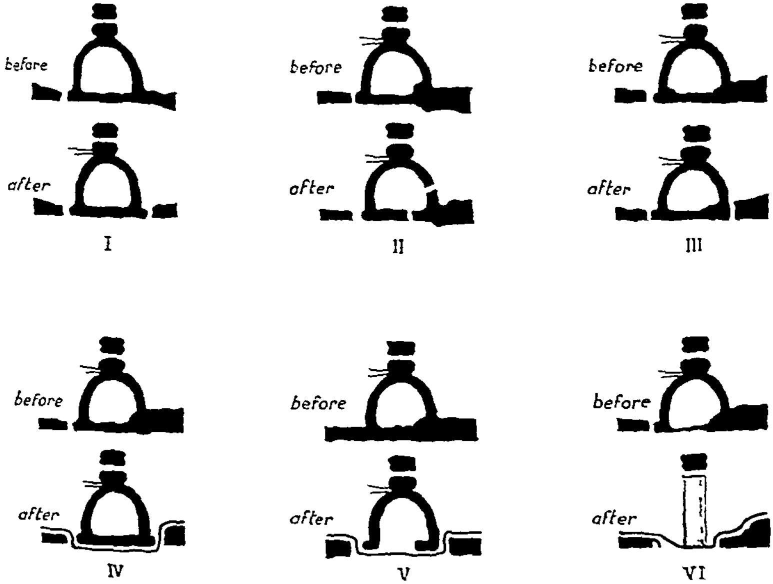

Fig 1 Some stapes surgery procedures I—Simple mobilization II—Anterior crurotomy III—Stapedo-vestibular osteotomy IV—Interposition Type I V—Interposition Type II VI—Fenestration-graft and plastic stapes

notably, John Shea Jr[18] This instrument made possible a true stapedio-vestibular osteotomy, also called "stapedioclasy" by Fr Antoli Candela[1]

Fowler, Jr,[5] in 1954, described his procedure of anterior crurotomy in which he separated the diseased anterior end of the footplate from the disease-free posterior part, which was mobilized Many more techniques might be mentioned, none, however, can be considered the ideal for stapes surgery The successful results of stapes mobilization by simple manipulation at the head or neck was about 35 per cent, whereas the immediate success by newer techniques was quoted by various authors as being 50 per cent, 60 per cent, and even 80 per cent Most of these authors acknowledge that after six months the number is greatly lessened, and after several years the success rate is 25 per cent One might, indeed, logically suppose that manipulation around the footplate may stimulate bone growth and re-ankylosis This was suggested in one case in which I had the opportunity of mobilizing the stapes three times when the patient refused fenestration After the first operation, the excellent postoperative gain was lost in eight months, after the second, in three months, and after the third operation I found the stapes completely fixed It seemed that manipulation stimulated further fixation on each occasion

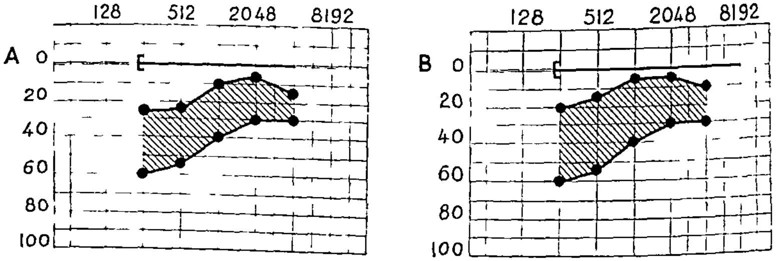

Fig 2 Average of air-bone gap gain with interposition O—bone level before operation A—average of all successful cases 95 per cent of all patients operated by interposition procedure B—average of the best results 75 per cent of all patients operated by interposition procedure

The frequency of refixation after mobilization led to other procedures which were more harmful to the labyrinth Kessel,[9] in 1877, recommended stapedectomy, and Cornelli,[4] in 1949, did the same operation in a larger number of patients Rosen[17] more recently has advocated making a small fenestra in the footplate when he has failed to mobilize it, and others, *i e*, H Guillon,[7] do a wider "platinotomie" None of these procedures, however, seems logical, because first, there is no columellar effect, second, the fenestra thus made tends to close rapidly, third, there is always risk of infection and labyrinthitis while the fenestra is open

It was to avoid this risk of infection that led M Pruvot[15]

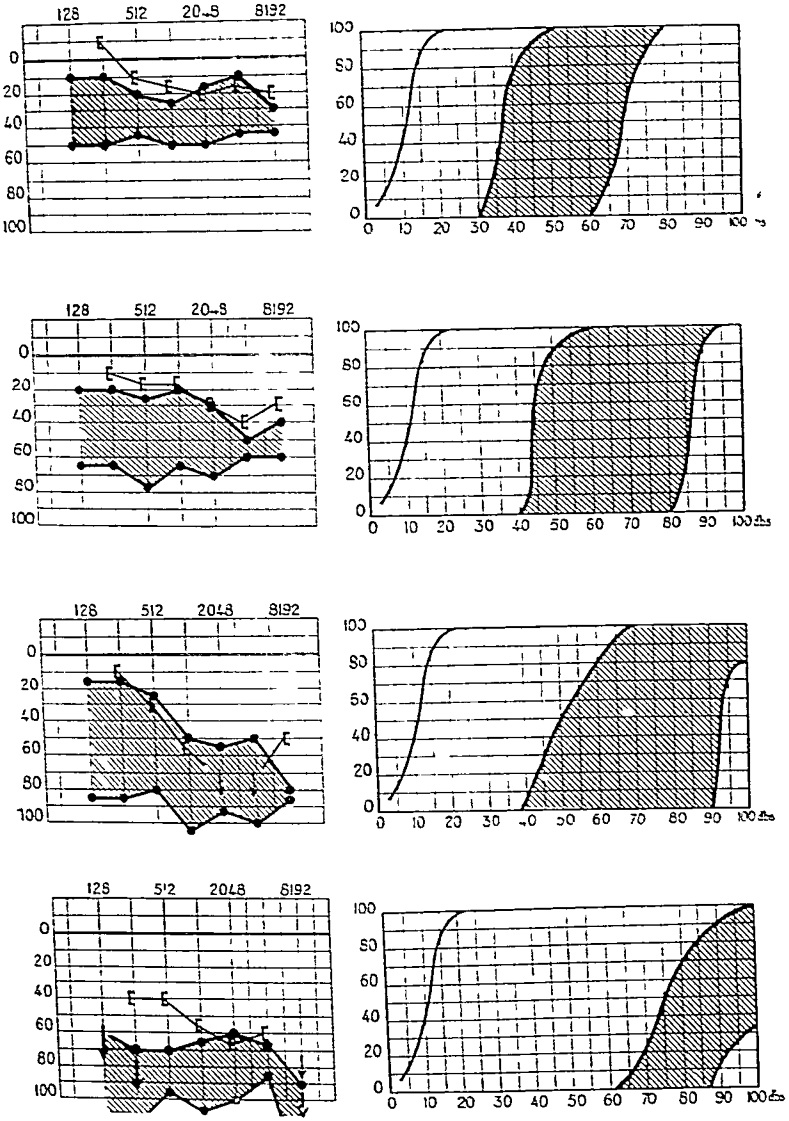

Fig 3 Some cases of interposition results after one year On the left tonal test on the right speech test From up to down different types of troubles III II IV IV

to suggest closing the oval window with a mucus membrane graft. Shea,[15] after removing the stapes, uses a vein graft to cover the oval window and re-establishes a columella effect by using a polyethylene tube, in one end of which the lenticular process of the incus is inserted, while the other end, cut to a point, is placed on the graft over the oval window.

Although his procedure seems logical, and his results are excellent, it seemed to us a pity to use a prosthesis of plastic when the patient's own stapes could more rationally be used. We,[11] therefore, described a procedure (1957) which we called "interposition" which seems to us to be the most physiological surgical procedure at the present time. The objectives of the operation are: *1.* To isolate the stapes, or part of it, from the edge of the oval window; *2.* To insert a graft between the free stapes or a portion of it, and the oval window. The graft has two aims: first, to close the vestibule and protect it from possible infection; second, to interpose tissue between the bony frame of the oval window and the bony footplate, and thus prevent the ankylosis.

STAPEDIO-VESTIBULAR INTERPOSITION

As this operation has been described in previous articles we shall here mention only a few particular points of technique.

Approach

This is almost always the transmeatal approach described by Rosen. It is important to remove enough of the canal to obtain a complete view of the stapedial footplate.

Examination of the Lesions

It is necessary to analyze the lesions causing ankylosis in order to judge what might be accomplished surgically and to estimate the possibility of preserving the stapes. Lesions are classified into four fundamental types (Bellucci[2]).

Type I. The joint space is still visible, but the annular ligament is beginning to ossify in places, mostly in its anterior-superior part.

Type II The otosclerotic surface has overgrown the anterior-superior part of the joint space and is already in the footplate In Types II and III a compensating atrophy of the remainder of the footplate may be seen This appears bluish and transparent The crura may be involved by otosclerosis, or on the other hand, may be atrophic

Type IV The whole footplate is involved by otosclerotic change and is completely fixed and thickened

Types I and II may respond to a simple mobilization procedure, and there were none of these types in the 135 cases reported here[*]

Materials for Interposition

Various materials have been tried, such as animal membrane, sheets of polyvinyl, conjunctiva, aponeurosis, bucal mucous membrane, and vein The last seems to be the best suited for the purpose The operation, described fully elsewhere, consists of (a) disarticulation of the incudostapedio joint, (b) removal of the mucosa around the oval window, (c) careful hemostasis, (d) ablation of the stapes from the window, (e) insertion of the graft and repositioning of the stapes under the incus and on the graft

Results

One-hundred-thirty-five cases were done by this method Of these, 96 have excellent results, *i e*, almost complete closure of the air-bone gap and even improvement of bone conduction Thirty-two have a good result, *i e*, an improvement of 10 to 30 decibels but without close of the air-bone gap Seven have no improvement

Lesions

Of the 96 excellent results
22 were Type II
10 were Type II/III
13 were Type III
20 were Type II/IV

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[*] Types I/IV or II/IV resemble Type I or II but with complete fixation of the remaining footplate

10 were Type III/IV
21 were Type IV

Of the 32 good results

6 were Type II
4 were Type III
7 were Type I/IV
5 were Type III/IV
10 were Type IV

Thus, of 135 cases in which postoperative audiograms were obtained more than three months later, there were 128 (94 8 per cent) good or excellent results Of these, 73 cases had complete fixation of the footplate and were not amenable to simple mobilization

Audiometric Types According to Shambaugh's Classification

Of the 96 excellent results, that is nearly total closures of the air bone gap, we note that

18 cases were Shambaugh Type I
50 cases were Type II
20 cases were Type III
13 cases were Type IV

Shambaugh's Type I case is favorable to fenestration of the semi-circular canal, Shambaugh's Type III has very little chance of success from fenestration according to his classification, and Type IV cases are not suitable for fenestration

Of the 32 good results

4 cases fell into Shambaugh's Type I
14 cases fell into Shambaugh's Type II
10 were Shambaugh's Type III
4 were Shambaugh's Type IV

This makes a total of 17 cases Type I 66 cases Type II, 30 cases Type III, and 17 cases of Type IV

We feel, therefore, that interposition is already a procedure worthy of further research In cases not suitable for fenestration and in which simple mobilization is likely to be completely unsuccessful, it may give a result in which the air-bone gap is completely closed In cases with good bone conduction it may allow the patient to recover absolutely normal hearing even in the face of complete fixation of the footplate

We realize that this series of cases is too small to be completely significant, and too recently done Five or six years must lapse before we can say anything definite about results, however, we believe that research should continue along the lines of stapes surgery, the interposition being only one aspect of the problem, and possibly only one stage of it

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ANNOUNCEMENT

The Annual Spring Meeting of the Alumni Association of the New York Eye and Ear Infirmary will take place April 4-7, 1960

The meeting will be oriented towards pediatric otolaryngology Symposia will be offered on Nystagmus as a Pediatric Problem, Diagnosis and Rehabilitation of Hearing Problems in Children, and Ankylosis of the Stapes There will also be a closed circuit television demonstration of surgical procedures In addition, courses will be offered in Allergy and Immunology in Children, X-ray Diagnosis in Ear and Sinus Problems in Children, Pediatric Laryngeal Problems, and Secretory Otitis Media in Children

Additional information may be obtained by writing to Dr John R Finlay, Secretary, Alumni Association, 218 Second avenue, New York 3, N Y

METHODS OF SHRINKING THE INFERIOR TURBINATES TO IMPROVE THE AIRWAY[*]

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One of the commonest complaints with which the otolaryngologist has to deal is chronic rhinitis with nasal obstruction. Perhaps this is not surprising when we consider the abuse to which the nasal mucosa is exposed. Repeated acute and chronic infections, allergy and endocrine disturbances all tend to engorge and often hypertrophy the mucous membrane, especially over the inferior turbinates. The inhalation of dust, smoke, and exhaust fumes, as well as the lack of humidity in dwellings during the winter months, act as irritants to certain persons. Changeable temperatures in the Spring and Fall with extremes of heat and cold also play their part.

In improving the airway through the nose the normal air currents should be considered. These have been described by various authors since the end of the last Century, but there is still some controversy as to the definite pathways. According to Proetz, "their pattern is determined by three structural elements, the direction of the anterior nares, the essential configuration of the nasal chamber and the relative sizes of the anterior and posterior nares."[1]

In the average Caucasian nose with a slit-like horizontal opening at the anterior nares, the inspired air arches high up into the nose in a parabolic curve before descending into the larger opening in the nasopharynx. This arrangement does not apply to the same extent in the Negro. In the widely dilated and more circular nostrils of the Negro, the inspired air takes a more direct transverse course over the inferior turbinates. This was thought by Thomson and Buxton[2] to be more suitable in a hot, moist climate where

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humidification was less important and there was little or no hindrance to the inspired air In contrast, the transverse elliptical opening of the North American and Esquimo directs the air upward over a larger area This is a more efficient mechanism for warming and moistening the air and more suitable for those in a colder climate

This can easily be demonstrated by means of a sagittal section of a human head fixed in a shallow square box showing the three turbinates, the septum being replaced by a piece of window glass About a foot of rubber tubing is fitted into the nostril and a cigarette placed in the open end of the tube Another tube about double the size representing the trachea, is fitted into the nasopharynx The glass and rubber tubes are hermetically sealed so that the smoke cannot escape A plaster model in place of an actual head is a suitable substitute On lighting the cigarette and breathing smoke through the specimen one can see, in a normal nose without any marked obvious obstruction, how the inspired air arches high up anteriorly before reaching the nasopharynx

Although most of the air is directed upward when the opening in the anterior naris is horizontal, some of the inspired air curves over the inferior turbinate, where it is warmed and moistened by the cavernous tissue before entering the lungs

If a model is used with a rubber or latex external nose which can be manipulated, the direction of the air currents can be changed By tilting the nostril upward, as one does in using a nasal speculum and dilating it with a larger round rubber tubing, the cigarette smoke tends to pass over the inferior turbinate in a similar pattern to that seen in the Negro

This agrees with the suggestion of Van Dishoek,[3] who considered that the inspired air in man was not influenced by the turbinates but took the path of least resistance

In his most recent and fascinating book, Negus[4] graphically showed air currents through the nose in various mammals He quoted these lines which John Hunter wrote nearly a hundred years ago

"Quadrupeds seldom breathe through their mouths, almost always through their noses, so that it may be said they are always on the scent, for the nose seems to make up for the deficiency of the eye, and they chiefly receive information by the nose For this purpose, too, the nose is more free from mucus in other animals than the human, so that the glands are not so numerous I believe that herbivorous animals always breathe through the nose, and this constancy fits the nose better for choosing the food "[5]

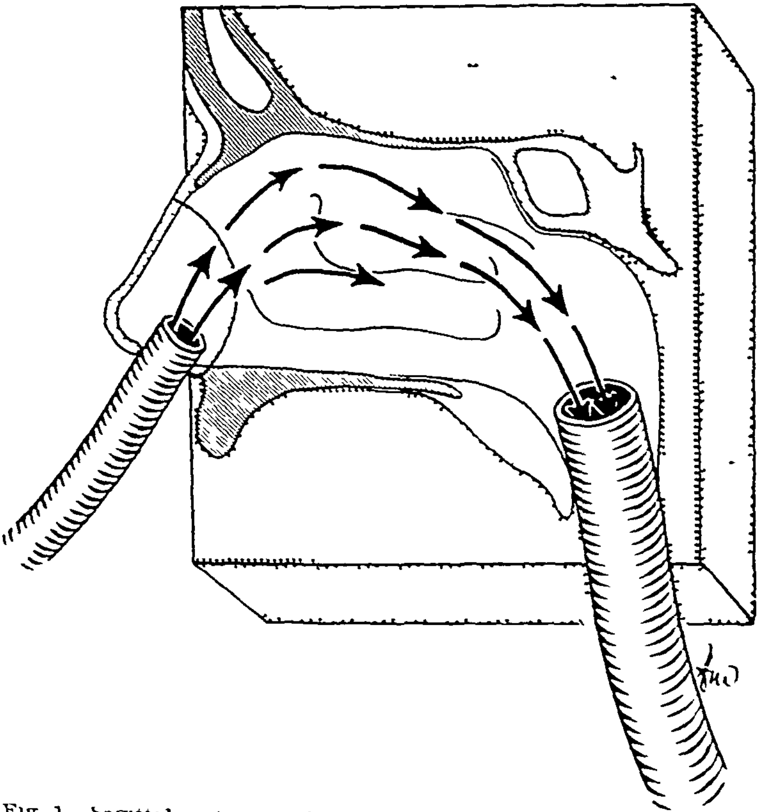

Fig 1 Sagittal section of skull covered with glass to show the normal inspiratory air currents

In general it might be said that long-nosed animals, such as deer or antelopes, have a keener sense of smell than those with shorter noses, as for instance monkeys and chimpanzees In the former the inspired air is directed upward to the olfactory area and warns the animal of impending danger, whereas in the latter the main air current is through the lower half of the nose below the olfactory area As a result, animals with relatively short noses depend more on sight than on smell, and this applies also to man

From a clinical point of view, air currents through the nose in humans assume great importance Although in general, inspired air curves high up in the nose as mentioned, currents also traverse the lower half as well This can frequently be demonstrated in patients who complain of chronic nasal obstruction Every rhinologist has had the experience of improving the airway temporarily in patients with chronic nasal obstruction by simply touching the anterior end of each inferior turbinate with 5 per cent cocaine Occasionally patients also complain of partial blockage of the nose after a deflected septum has been corrected by a submucous resection From the rhinologist's point of view the operation was successful, as the septal obstruction was all removed, but sometimes the patient is disappointed with the result If the inferior turbinates were treated at the same time in well selected cases the result would often mean the difference between success and failure

Assuming there is no redundant tissue to be removed, various agents have been tried to shrink the mucous membrane of the inferior turbinates The electric cautery, silver nitrate, trichloracetic acid, phenol,[6] fuming nitric acid,[7] chromic acid, etc, have all been used to streak the turbinates and cause shrinkage

In recent years, submucous injections of sclerosing agents such as sodium morrhuate or sodium psylliate have been employed on the assumption that they contract the mucosa of the turbinates without injuring the cilia on the surface[8] Submucous electrocoagulation[9] has also been favored by many, as it destroys some of the large cavernous spaces and has the advantage over galvanic cauterization, because there is little or no crusting and a minimum of reaction

The success of these methods of treatment depends upon the proper selection of patients, being most beneficial in those who have no allergic background but have a chronic swelling of the mucous membrane of the inferior turbinates, which shrinks well under the action of vasoconstrictors

If there are fixed tissue changes of the turbinates which are irreversible, these structures will not respond to conservative measures This applies also to the posterior tips of the inferior turbinates If they do not shrink with cocaine or adrenaline, they can be removed with an electrocoagulation snare, or the electrocoagulation needle can be applied[10] in the usual manner, thus avoiding the danger of severe bleeding

When the inferior turbinate bone itself is hypertrophied none of these measures is entirely successful From the foregoing it is apparent that there are a great many patients with vasomotor rhinitis or chronic enlargement of the inferior turbinates who have no evidence of sinusitis, and are not benefited by antiallergic therapy Many of these have tried medical or other conservative forms of treatment to no avail and still seek relief from their persistent nasal obstruction Submucous resection of the inferior turbinates in these cases seems to offer the best chance of a permanent result

A number of incisions have been described by various authors to remove the anterior enlarged third of the bone

In 1930, Odeneal[11] recommended submucous resection of the middle turbinate to improve the airway, in cases of marked hypertrophy Without going into detail in his short article, he stated "It is a simple procedure, by means of a sharp elevator, to separate the mucous membrane and submucous tissues from the underlying bone and remove as much of the bone as is desired, afterwards replacing the flap in position and holding it there for 24 hours by means of a petrolatum-soaked pledget of cotton It will be found necessary to clip off a small portion of the flap before replacing it in position, as there will be a certain amount of redundant tissue The incision is made along the inferior external border of the turbinate, and the dissection carried out internally, it is not necessary to separate it externally as this bone is always left" He concluded by saying, "I have never been called on to reduce the inferior turbinate in this manner, but I do not see why this cannot be done just as easily when indicated"

A few years later (1936) Harris[12] reported an "improved" technique for submucous turbinectomy of the inferior turbinate bone Without illustrations of any kind he said "Under local anesthesia I make an elliptical incision over the most prominent area of the inferior turbinate and resect this portion of the mucous membrane As much of the bone is then excised under direct vision as is necessary" He concluded by saying he had done this operation in three cases with marked improvement in the "breathing space"

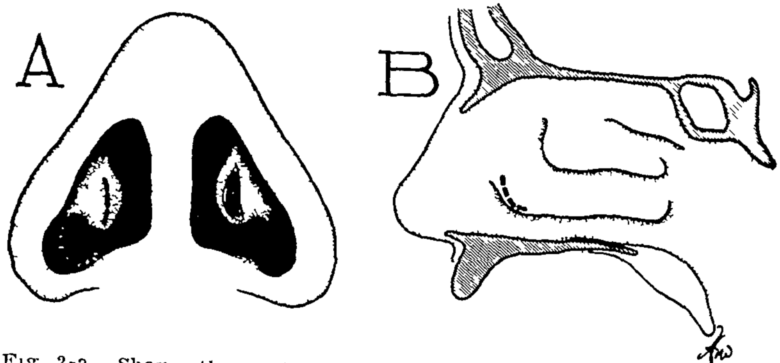

Fig 2-a Shows the vertical incision on the left and the exposed inferior turbinate on the right after elevating the mucous membrane
Fig 2-b The dotted line indicates the incision on the right inferior turbinate bone

According to House,[13] "Submucous resection of the anterior portion of the inferior turbinal bone is of most value in perennial nasal obstruction due to turbinal enlargement, either unilateral or bilateral, which cannot be satisfactorily controlled by more conservative general management Included in this group are vasomotor rhinitis, allergic rhinitis, compensatory unilateral hypertrophy secondary to septal deviation, and irreversible hypertrophy secondary to prolonged irritation This underlying irritation may be due to infection, occupational environment, excessive smoking, etc" He stated that it was not a new procedure and that Yankhauer had advocated it in his student lectures but had left no published reports on the subject

A vertical incision is made over the anterior inferior leading edge of the turbinate He stressed the fact that the anterior portion of the inferior turbinate bone has a rough, pitted surface, and as a result the mucoperiosteum cannot be readily elevated with a blunt septal instrument A right and left angulated Freer knife is useful in separating the periosteum by sharp dissection from the surfaces of the bone The mucoperiosteum is lifted from both the lateral and medial surfaces of the inferior turbinate bone for a distance of about 1 5 cm Any fibers that are adherent are cut away by means of small turbinotomy scissors The anterior third of the bone is grasped with Takahashi forceps rotated gently until it is fractured and then carefully removed without tearing the mucous membrane Usually the posterior two-thirds of the bone is very thin and can be left in place after pressing it to the lateral wall Sutures are unnecessary, as the edges of the incision fall together and are held in place by finger cots

For a number of years Furstenberg[14] has been performing this operation and has been very enthusiastic about the results He does not agree with the statement that the thicker the inferior turbinate bone itself the better the result Some authors have suggested that when the bone is thick the result after submucous resection is good, but if the bone is thin and the mucous membrane hypertrophied, the result is not likely to be beneficial In his opinion he has had equal success with thin bones as well as very thick ones He does "not believe that the thickness of the osseous structure of the turbinate plays any very significant part in the prognosis" His approach is somewhat different He begins by making two or three injections of 1 per cent procaine with adrenalin into the inferior turbinate starting posteriorly and coming forward This assures satisfactory anesthesia He then makes a deep horizontal incision commencing "about two centimeters anterior to the posterior end of the inferior turbinate" In this way he avoids the "rather profuse blood supply which comes in from the posterior end of the turbinate The in-necessary to separate it externally as this bone is always left" He concluded by saying, "I have never been called on to reduce the inferior turbinate in this manner, but I do not see why this cannot be done just as easily when indicated"

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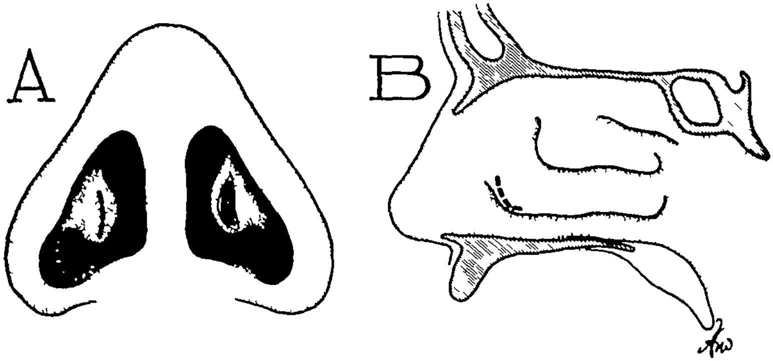

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A vertical incision is made over the anterior inferior leading edge of the turbinate He stressed the fact that the anterior portion of the inferior turbinate bone has a rough, pitted surface, and as a result the mucoperiosteum cannot be readily elevated with a blunt septal instrument A right and left angulated Freer knife is useful in separating the periosteum by sharp dissection from the surfaces of the bone The mucoperiosteum is lifted from both the lateral and medial surfaces of the inferior turbinate bone for a distance of about 1 5 cm Any fibers that are adherent are cut away by means of small turbinotomy scissors The anterior third of the bone is grasped with Takahashi forceps rotated gently until it is fractured and then carefully removed without tearing the mucous membrane Usually the posterior two-thirds of the bone is very thin and can be left in place after pressing it to the lateral wall Sutures are unnecessary, as the edges of the incision fall together and are held in place by finger cots

For a number of years Furstenberg[14] has been performing this operation and has been very enthusiastic about the results He does not agree with the statement that the thicker the inferior turbinate bone itself the better the result Some authors have suggested that when the bone is thick the result after submucous resection is good, but if the bone is thin and the mucous membrane hypertrophied, the result is not likely to be beneficial In his opinion he has had equal success with thin bones as well as very thick ones He does "not believe that the thickness of the osseous structure of the turbinate plays any very significant part in the prognosis" His approach is somewhat different He begins by making two or three injections of 1 per cent procaine with adrenalin into the inferior turbinate starting posteriorly and coming forward This assures satisfactory anesthesia He then makes a deep horizontal incision commencing "about two centimeters anterior to the posterior end of the inferior turbinate" In this way he avoids the "rather profuse blood supply which comes in from the posterior end of the turbinate The incision is then made forward to the extreme anterior end of the inferior turbinate" The mucous membrane is separated from the bone by means of a submucous elevator The flap is first lifted upward and then the lower flap is dissected downward until the free margin of the anterior part of the osseous structure is identified The bone anteriorly and interiorly is removed by means of a narrow, blunt-ended biting forceps All of the osseous portion is carefully taken away Then gauze vaseline covered packs are inserted and left for 48 hours One is placed under the inferior turbinate and one above to bring the flaps together Two other packs are usually used on each side and sedation given to relieve the discomfort "By carefully packing the nose in this manner, one finds later only a small line of scar bisecting the turbinate in its

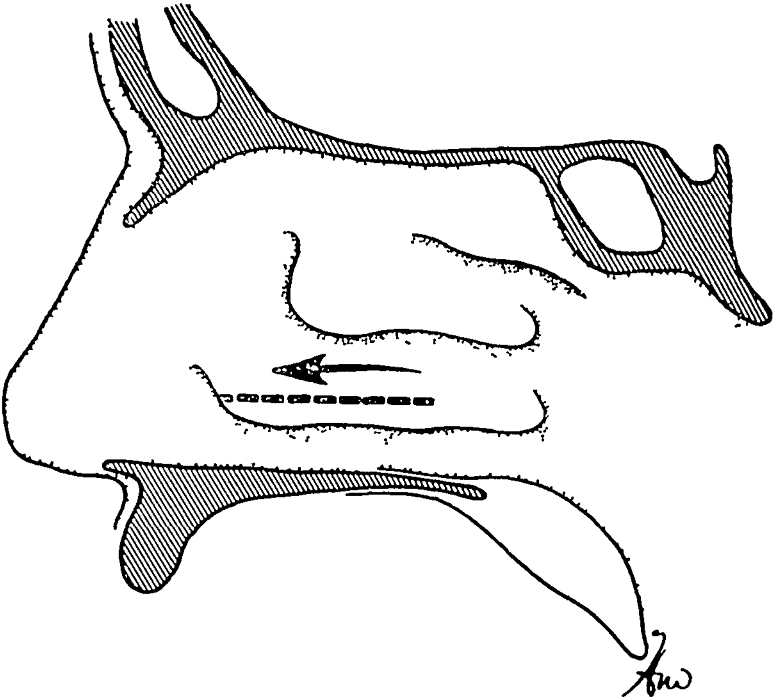


Fig 3 The horizontal dotted line indicates the incision It is about 6 mm above the lower edge of the inferior turbinate bone

anterior-posterior plane with a little dimpling along this line, due to fibrous connective tissue contraction In no way is the physiologic function of the inferior turbinate jeopardized when this is accomplished"

I have quoted freely from notes given me recently by Dr Furstenberg In his hands the results are very successful with permanent improvement in the breathing, however besides this method, the technique I have used when indicated is very similar to that described by Yankhauer and House with a few modifications In most cases it is done in conjunction with a septal resection when this is found necessary After the usual premedication, absorbent dipped in pontocaine is applied to the inferior turbinates This does not constrict the vessels or blanch the mucous membrane Two injections of 1 per cent procaine with adrenaline are made into each turbinate beginning posteriorly and coming forward Another injection is made into the anterior prominent portion and one above at the attachment of the turbinate This balloons up the tissue and makes separation from the bone easier as well as helping to control the bleeding

A semicircular incision is made with a septal or No 15 Bard Parker knife on a long handle, starting above on the lateral wall at the attachment of the turbinate and curving downward over the anterior tip The incision is carried through the mucosa and periosteum to the bone The angulated Freer knife is used to cut the adherent fibers of periosteum first on the lateral and then on the medial side of the bone If the slit-like opening in the head of the turbinate is too small and the vision restricted, the incision can be extended downward along the lower edge Once the denuded bone is exposed it is not difficult to remove the hypertrophied anterior third or half by means of a Jansen-Middleton biting forceps In most cases this is all that is required, as the turbinate usually tapers off and becomes smaller posteriorly unless the posterior tips are enlarged The biting forceps removes the bone cleanly and lessens the danger of tearing the mucous membrane

When the hypertrophied portion is removed the edges of the incision fall together and heal without the necessity of sutures As a rule it is helpful to fracture the remaining posterior one-third of the turbinate toward the lateral wall by means of a long-bladed nasal speculum or a septal elevator Every millimeter gained in this way improves the nasal airway A finger cot lightly packed with folded $\frac{1}{4}$-inch gauze and covered with vaseline is placed between each inferior turbinate and the septum These are removed in 48 hours There is very little bleeding, and the discomfort is similar to that experienced after a resection of the nasal septum When shrinking occurs and the incision well healed, there is little or no scar

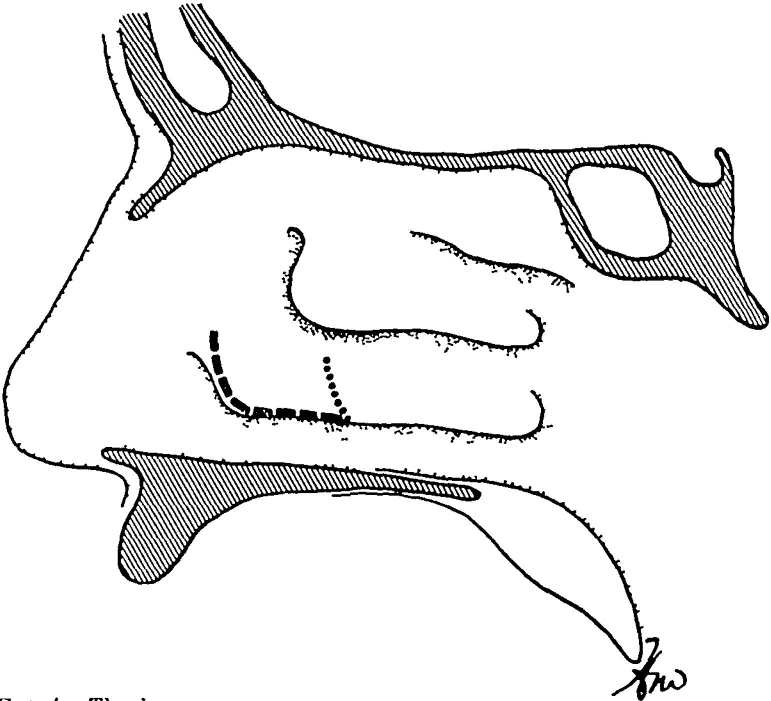


Fig 4 The heavy dotted line shows the incision beginning high up at the attachment of the inferior turbinate and extending down along the lower edge This gives a wider exposure The dotted line posteriorly marks the line of incision when only part of the anterior portion of the inferior turbinate is to be removed

Within a few weeks the inferior turbinate is smaller and thinner as the supporting bony structure has been removed This, of course, gives permanent improvement even in patients with allergy of unknown origin As the naso-lacrimal duct is not disturbed tearing is not a problem

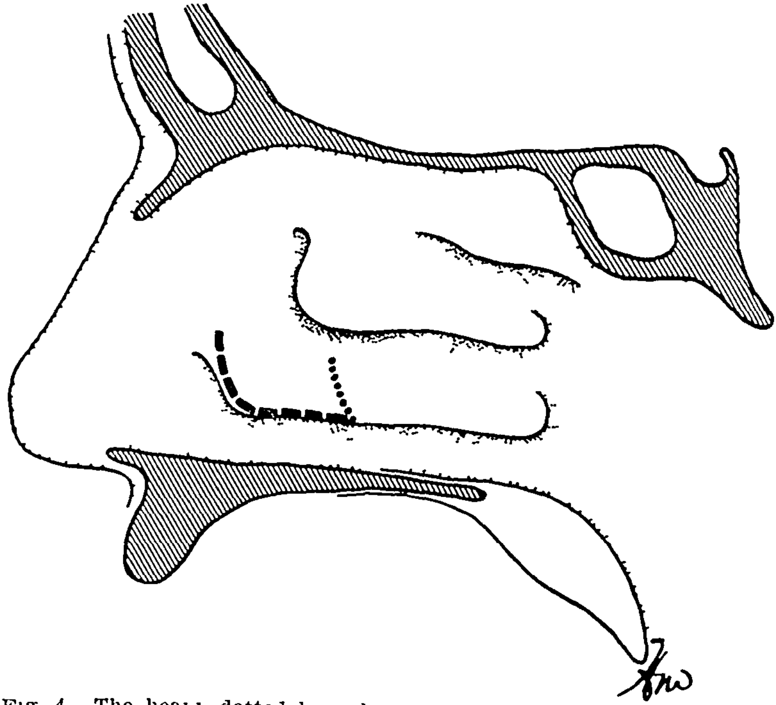
Although no disagreeable postoperative results have been noted, such as atrophy or crusting, some might object to the fact that all the supporting structure anteriorly has been removed If it is deemed necessary to leave part of the inferior turbinate bone, this is a simple procedure

The semicircular or bowed incision is carried down over the anterior tip and along the inferior edge for a distance of about 2 centimeters A second parallel incision to the bone about 2 centimeters posteriorly extends from above at the attachment of the turbinate down to meet the first incision at the lower edge of the turbinate The mucous membrane is elevated from below upward very much like the flap of an envelope When the bone is exposed, as much is removed as is necessary to improve the airway, by means of a Freer gouge and mallet The flap is then replaced and held in position with a vaseline-covered finger cot

Many patients operated upon in this manner have been observed over a period of years None has shown any untoward results Clinically the incisions have all healed by first intention, as there has been no loss of mucosal tissue From the functional point of view the drainage of mucus is still downward and backward as the normal ciliary streams have not been disturbed

Although the airway is greatly improved and the patients in general are enthusiastic about the results, occasionally one is disappointed This is more likely to occur in children about the age of puberty In view of this it would seem wiser to delay the operation until the age of 17 or 18

In conclusion, the relief of nasal obstruction following this combined type of operation (septal resection and submucous resection of the inferior turbinates) is dramatic in the vast majority of cases When indicated, the results are far superior to those obtained when the septum alone is corrected the incision fall together and heal without the necessity of sutures As a rule it is helpful to fracture the remaining posterior one-third of the turbinate toward the lateral wall by means of a long-bladed nasal speculum or a septal elevator Every millimeter gained in this way improves the nasal air way A finger cot lightly packed with folded $\frac{1}{4}$-inch gauze



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AGENESIS OF THE LUNG[*]

Report of a Case

STEVEN BORSANYI, M D ,

Baltimore, Md

Agenesis of the lung is not a common condition The first case was reported by Haberlein[1] in 1787 The number of the authentic cases is about 150 at present and most of them were discovered at autopsy In the series reviewed by Hochberg and Nachlerio[2] in 1955 (100 cases), the diagnosis was made during life in 27 per cent of the cases The incidence of this anomaly's discovery during life has increased during recent years

The etiology is unknown Schwalbe[3] states that the condition is due to a developmental error of endogenous origin, and that this maldevelopment is the result of a faulty germ plasm Ferguson and Neuhauser[4] explain the genesis of this condition on the basis of a defect in the germ plasm Gruenfeld and Gray[5] feel that "developmental anomaly of the lung is favored by the phylogenetic newness of this organ and the enormous growth it must undergo in the embryo before its functional form is achieved " Oyamada, et al ,[6] emphasize the importance of the history of rubella during the first trimester

Schneider[7] classifies the deficiencies of the lung into three groups depending upon the development of the bronchus and lung tissue In type I, one lung is absent In type II, the bronchus ends in a blind pouch, and there is a complete absence of lung tissue In type III, the bronchus is poorly developed and ends in rudimentary lung tissue This classification is of no particular value, has no answer to the question of embryology, nor has it any influence on the prognosis or diagnosis It is even questionable whether type III can be called *agenesis* instead of *hypoplasia*

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[*] From the Department of Otolaryngology University of Maryland School of Medicine

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1390 Sherbrooke street West

THE UNIVERSITY OF MICHIGAN MEDICAL CENTER

The annual Otolaryngology Conference at The University of Michigan will be held April 21, 22, and 23, 1960, under the direction of Dr James H Maxwell, Chairman of the Department of Otolaryngology

Applications may be addressed to the Department of Postgraduate Medicine, University Hospital, Ann Arbor, Mich

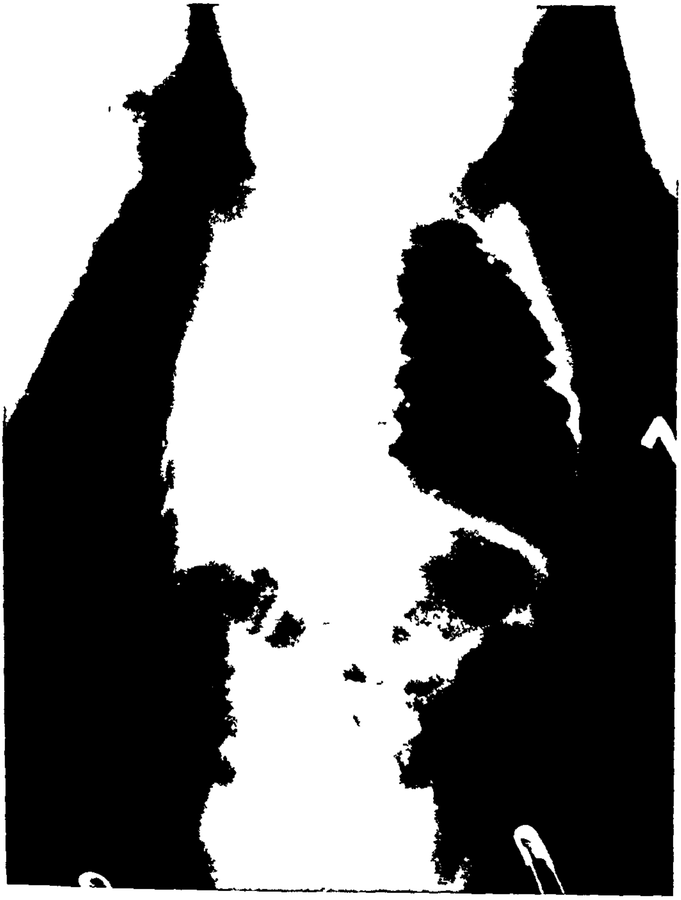


Fig 1 The right side of chest shows a homogenous density The trachea heart and mediastinum are displaced to the right

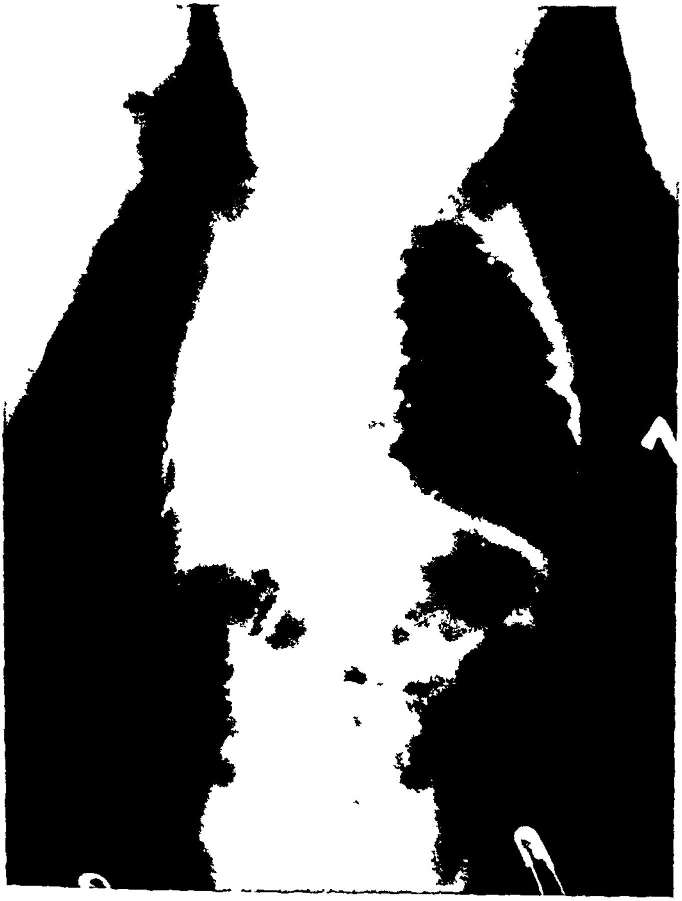
flattening of the involved side If the right lung is missing, the heart sounds will be found on the right side simulating dextrocardia

The characteristic X-ray findings are dense, homogenous shadow with elevation of the diaphragm and narrowing of the intercostal spaces on the affected side There is a displacement of the trachea, heart and mediastinum to the involved side It is difficult sometimes to outline the heart shadow The remaining lung may pass the midline

Sixty per cent of the patients have other developmental defects with pulmonary agenesis The incidence of associated cardiovascular anomalies is 25 per cent[8] Wexels[9] lists some of the associated anomalies with this condition as patent ductus arteriosus, patent foramen ovale, atresia of bowels, atresia ani, atresia of esophagus, ventricular defect, horseshoe kidney, hare lip, cleft palate, malformations of vertebrae, absence of radius, spina bifida, accessory thumb, pulmonary vein entering the azygos vein Wilson's[10] case had a unilateral mandibular dysostosis with agenesis of the lung In our case a rudimentary right thumb was present, and also hydramnios was noted at delivery An anomalous course of the great vessels like the pulmonary artery or the aorta, could be responsible for respiratory difficulty and even cause death due to the compression of the trachea, as in the case reported by Maier and Gould[11] This is unusual, but apparently did occur in this case The usual cause of death in case of vascular compression is strain and ultimately heart failure, if it is arterial, or congestion of the head and neck and selected viscera if it involves the veins Morbidity and mortality depend more upon distortion of the airways than of the vascular channels[8] Supernumerary lobe in the unaffected lung may be an associated finding with pulmonary agenesis, as in the case reported by Lelong, et al[12]

The symptoms are variable, or the patient may be completely asymptomatic Dyspnea, cyanosis and tachycardia are the symptoms encountered in the younger age group The dyspnea is mild, intermittent and related to activity Cyanosis and tachycardia do occur when this condition is aggravated by upper respiratory infection Few cases have wheezing and stridor

The clinical diagnosis is suspected on the basis of the physical and X-ray findings Absence of breath sounds, dullness of percussion, and diminished respiratory excursion are frequent findings, however, the breath sounds could be transmitted from the remaining lung This is best heard at the apex The chest, as a rule, is symmetrical in infants, but lag is uniformly present on the affected side As the child grows older there might be some asymmetry of the chest with



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Hurwitz and Stephens[13] claim priority for advocating bronchoscopy as a rational method for clinical diagnosis. Ferguson and Neuhauser,[4] reporting the first comparatively larger series of congenital anomalies of the lung where the diagnosis was made during life, emphasize the importance of the bronchogram. Ingram, Hudson, and Davis[14] advocate first angiocardiography for demonstration of anomalous pulmonary circulation. Angiocardiography reveals the absence or rudimentary development of the blood supply to the affected lung and, at the same time, reveals congenital malformations of the heart and possibly the kidney. The three last mentioned diagnostic procedures should be undertaken

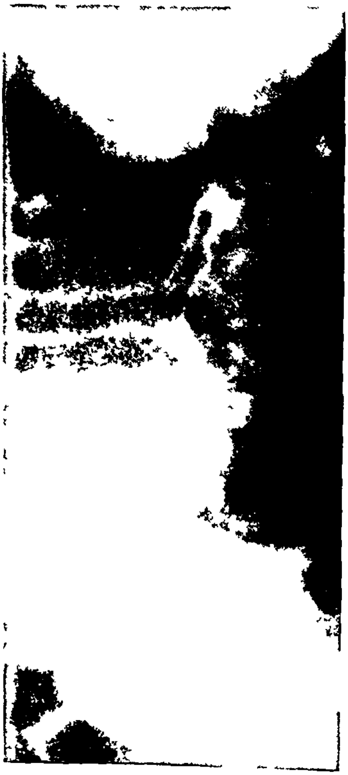

Fig. 2. Barium swallow reveals the displacement of the esophagus toward the side of the affected lung.

after due clinical consideration of each individual case, however, bronchoscopy without anesthesia is now an accepted diagnostic procedure in unusual respiratory conditions in infants

Agenesis of the lung is compatible with life In Hochberg and Nachlerio's[2] series, 27 per cent of the 96 were known to have lived to ages between 15 and 72 years According to the literature this anomaly occurs more commonly in males, and is noted more often on the left side The prognosis depends upon the associated anomalies and also upon which lung is absent Prognosis is considerably better with agenesis of the left than of the right lung Statistics in the ten-year-old age group show a 58 per cent survival when the left side is affected, compared with 30 per cent when the right side is involved[8] The greater shift of the trachea, the main stem bronchus and the great vessels is responsible for the difference in mortality Schaffer and Rider[8] suggested the introduction of some non-absorbable and non-irritating material into the affected hemithorax to prevent the mediastinal shift I was unable to find any reference in the literature that this has ever been carried out

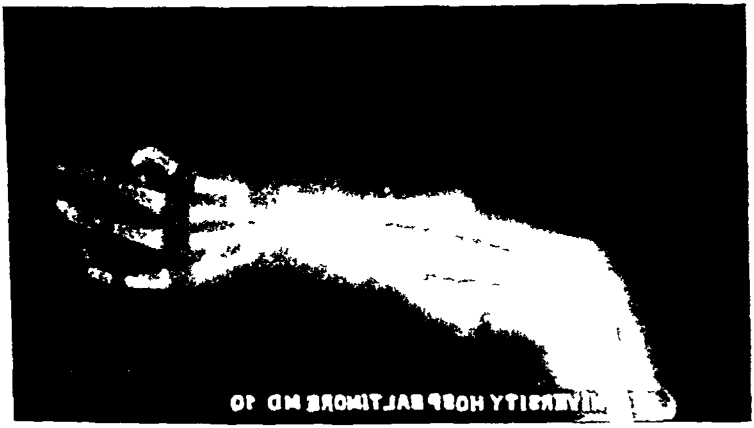

Fig 3 X-ray from the hands shows the hypoplasia of the first metacarpal bone on the right hand

The lack of surgical approach might lie in the fact that

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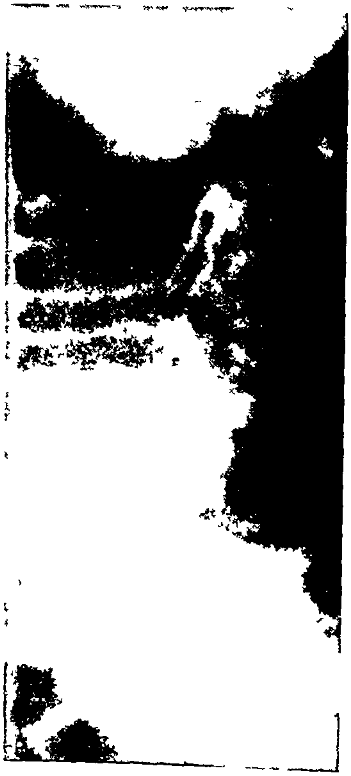

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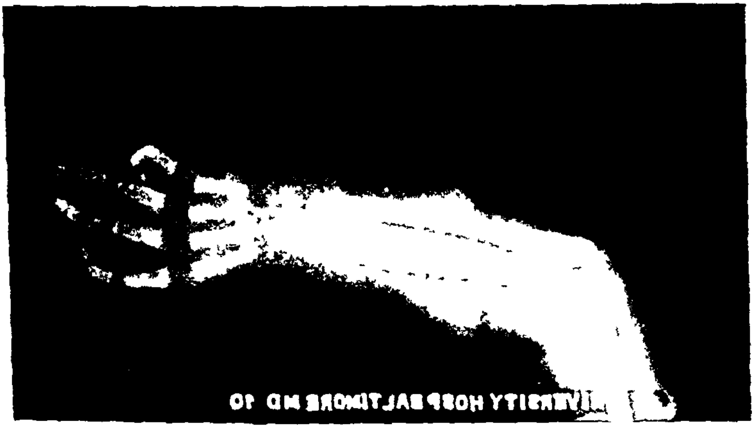

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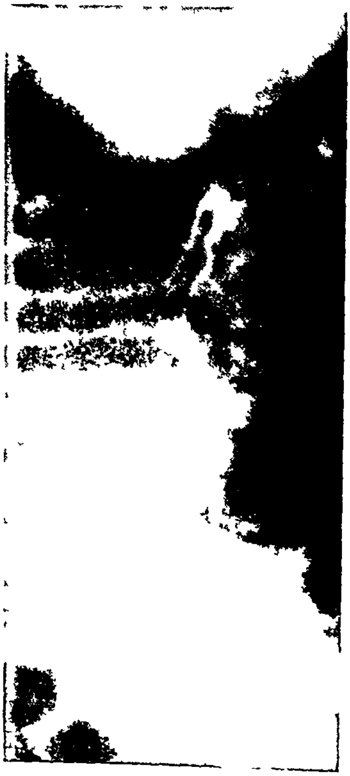

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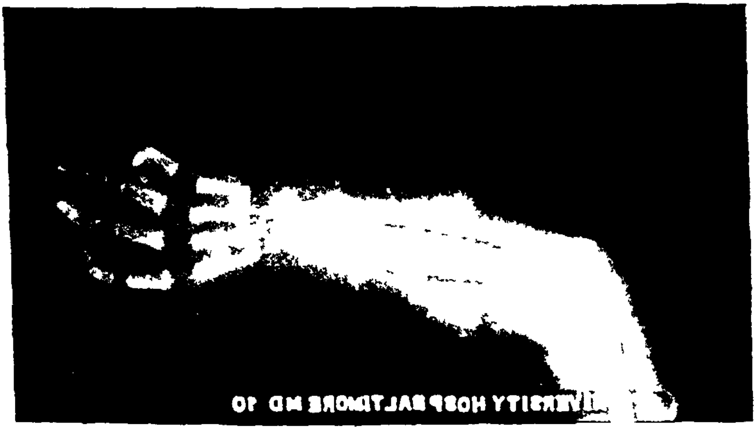

Fig 3 X-ray from the hands shows the hypoplasia of the first metacarpal bone on the right hand

The lack of surgical approach might lie in the fact that

most surgeons believe that the patient with pulmonary agenesis dies of cardiac failure rather than from the effects of the agenesis. In the series reviewed by Oyamada, et al.,[6] associated cardiac defects were discovered in only one-fourth of the cases. This gives rise to the thought that a certain per cent of the patients might benefit from some type of surgical procedure, which would prevent the mediastinal shift and the consequent distortion of the tracheobronchial tree and the great vessels. It is a known fact that the morbidity and the mortality is higher when the right lung is missing and the distortion is relatively greater.

REPORT OF A CASE

A girl, aged seven weeks, was born at University Hospital on August 1, 1959, two weeks before term. The mother, bigravida and bipara with one normal living child, was in good health throughout the pregnancy. When the membranes ruptured, a large amount of fluid was obtained, and the diagnosis of hydramnios was made. The premature female child was delivered by breech extraction with Piper forceps to the aftercoming head. The baby needed resuscitation but picked up rapidly. She weighed 2291 grams.

The child did not show respiratory distress on the following days, but on routine examination dullness was found at the right side of the chest. The breath sounds were heard well in the apex and front of the chest, but they were poorly heard posteriorly mostly over the lower part of the chest. The heart sounds were best heard over the right hemithorax. X-ray of the chest revealed a homogenous density over the right hemithorax. The mediastinum and the heart shadow were displaced to this density. The heart could not be outlined. On physical examination a rudimentary right thumb was also noted. X-ray of the hands revealed hypoplasia of the first metacarpal bone on the right hand. Suction through endotracheal catheter was performed but the X-ray failed to show any change in the previously reported density of the right hemithorax. Barium swallow did not reveal intrinsic abnormalities in the esophagus. There was a moderate shift of the esophagus to the right along the entire mediastinum. The child did well, had normal temperature, no respiratory distress and gained weight at a normal rate. The patient was stabilized, and the clinical and the X-ray findings strongly supported the diagnosis of pulmonary agenesis. She was discharged from the hospital in good condition at the end of the second week. She was brought back to the Otolaryngology Department four weeks later for bronchoscopy to confirm the clinical diagnosis. The bronchoscopy revealed the larynx, trachea and left bronchial tree to be normal. The trachea was continuous with the left main bronchus without evidence of the carina or opening of the right main bronchus.

SUMMARY

A case of agenesis of the right lung, in a seven-week-old female infant is reported. The associated findings were hypoplasia of the first metacarpal bone on the right hand and hydramnios at birth The review of the literature shows that the number of cases diagnosed during life are increasing The difference in morbidity and mortality, according to the side involved, is discussed

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SIXTH INTERNATIONAL CONGRESS ON DISEASES OF THE CHEST

The Sixth International Congress on Diseases of the Chest will be held at the University of Vienna from August 29 to September 1, 1960

NASAL NECROSIS ASSOCIATED WITH POSTNASAL PACKING FOR PROTRACTED EPISTAXIS

JAMES T KING, M D,

Atlanta, Ga

A white male dental technician, 58 years of age, was seen on July 11, 1959, at 4 30 p m at St Joseph's Infirmary because of severe epistaxis of 18 days' duration He was referred by a colleague who was leaving on vacation The patient stated that he had had a similar attack of nosebleed 14 years before, while living in Florida, and had been given up for dead two or three times

The present illness had begun the day of admission (June 23, 1959), and most of the bleeding was from the right nasal chamber, but the left was thought to have bled also On that date packing was inserted into both sides of his nose anteriorly which seemed to control the situation fairly well, however, one week later, after the packing was removed and he was thought to be ready for discharge, profuse bleeding ensued Anterior packs were reinserted, as well as a Fox balloon which was introduced posteriorly In a few hours bleeding recurred, even more profusely

Due to the patient's extreme apprehension and pain, he was anesthetized with sodium pentothal administered intravenously, and two postnasal packs were cinched up tightly one into each choana, which, combined with tight anterior packing controlled the bleeding Five days later the posterior packs were removed The anterior packing was then removed a little at a time over the next four days, when the bleeding recurred, and his nose packed again anteriorly

The first time I saw the patient he was bleeding freely from the right side around the many cotton pledgets with which his nose was packed Upon removal of these, the bleeding stopped The mucous membrane was missing from the posterior inferior portion of the nasal cavity bilaterally, the floor in these areas consisting of bare rough bone—sans periosteum The approximate necrotic area was 1x2 cms on either side and corresponded to the horizontal processes of the palatine bones The posterior septum was freely movable, and the posterior hard palate was rather tender when palpated through the mouth Biopsy specimens taken from the raw edges of the ulcerated areas showed only necrotic inflamed nasal mucosa The nose was not repacked

During this hospitalization the patient was also treated concurrently by an internist urologist and psychiatrist because of mild hypertension, bladder irritation and toxic delusions He had been or was being administered the following drugs Adrestat aspirin B & W No 3 and No 4, Benadryl, Carbrital, Chloromycetin Compazine Darvon Doriden, morphine Koagamin paraldehyde, penicillin phenobarbital Premarin, Pyr

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Editor's Note This manuscript received in The Laryngoscope Office and accepted for publication Aug 17 1959

dium, Rautrax Sparine Stelazine Thorazine Vesprin Vistaril and Fleet's Phosphosoda.* Despite these he continued to bleed intermittently

The episodes of epistaxis, while violent, were of short duration and would not occur more than twice in each 24 hour period in fact, the bleeding would always seem to stop at the time I reached the patient's bedside thus preventing my visualizing the precise bleeding point Daily transfusions were required to keep his hemoglobin at the 9 to 10 gm level The necrotic areas began slowly to cover over with granulation tissue, but a purulent rhinitis was present

On July 17 he was transferred to a more accessible hospital (Georgia Baptist) and most medication was discontinued With the next bleeding episode, the bleeding point was discovered beneath the right inferior turbinate at the junction of the hard and soft palate just behind the necrotic area Blood was spurting into and across the nasopharynx giving the illusion that the left side also was bleeding

The next day, under local anesthesia the right external carotid artery was ligated When last seen on August 11th the patient had had no further show of blood the necrotic areas were healing and the purulent rhinitis was improving

SUMMARY

Presented is a case report of severe epistaxis of 25 days' duration which was finally controlled by ligation of the homolateral external carotid artery Necrosis of the nasal mucosa in the posterior floor of the nasal chamber, probably due to tight and prolonged postnasal packing, was a complication

516 Baptist Professional Bldg

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* Multi Medici Regem Sustulerunt Hadrian 138 AD
from his nose and to ask for "something to make me rest"

THIRTY-THIRD ANNUAL SPRING CONGRESS OF GILL MEMORIAL EYE, EAR AND THROAT HOSPITAL

The Gill Memorial Eye, Ear and Throat Hospital announces to the profession its Thirty-third Annual Spring Congress in Ophthalmology and Otolaryngology, April 4 through April 9, 1960 A formidable list of speakers will participate

For further information write Superintendent, P O Box 1789, Roanoke, Va

A NEW FOREIGN BODY FORCEPS FOR SPHERICAL OBJECTS IN THE EAR AND NOSE

MERRILL LINEBACK, M D ,

College Park, Ga

The problem of round objects within the recesses of the nose and ear has long vexed otolaryngologists At best they are removed with difficulty under general anesthesia Particularly is this true if the foreign bodies are hard without purchase area Usually a ring curet or cerumen scoop has to be utilized and the object rolled out much as a ball and paddle

It was with these difficulties in mind that the author designed an "obstetrics"-type forceps (see Fig 1) with a curved perforated grasping head that allowed the object to fall easily into the two *hands* of the forceps Corrugation of the purchase lips is added for increased effectiveness The width of the distal end in the original forceps is 4 mm , which has proved satisfactory so far, but they are being redesigned to a 3 mm width which is sufficient for the infant's and young child's canal

The idea for such forceps came from a chance jesting re-remark in Surgery, when the author remarked to the nurses that removing a round object from the external canal was much the same procedure as delivering the head in obstetrics Since June of 1959, I have encountered seven round, hard, spherical foreign bodies, five in the nose, and two in the ear, that have been very satisfactorily removed with the aid of this instrument One of the main advantages is the absence of bleeding that usually accompanies removal with a ring curet As an added precaution with posterior foreign bodies near the nasal choanae, the author passes one of his Bardek adenoid fossa balloons into the opposite nasal passageway into the nasopharynx, inflates it, and then allows anesthesia to pro-

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*This instrument manufactured by Storz Instrument Co , St Louis Mo

Editor's Note This manuscript received in The Laryngoscope Office and accepted for publication Jan 4 1960

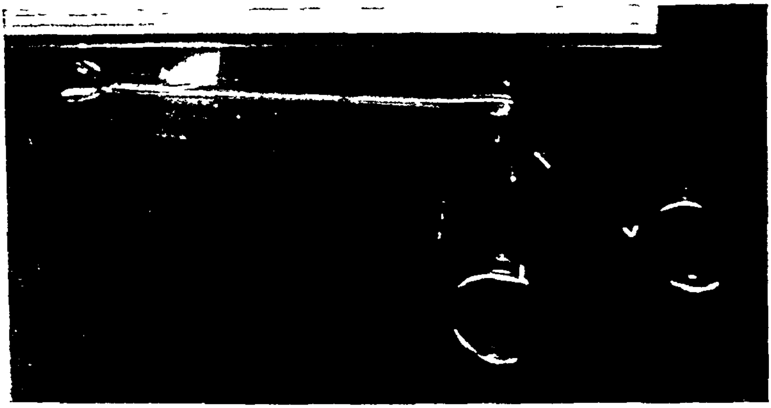

Fig 1 The 'obstetrics'-type forceps showing length and the opening corrugated jaws

ceed This added safety factor prevents aspiration of the foreign body into the bronchi as happened once, prior to using the nasal balloon

SUMMARY

A new foreign body forceps for use with hard, spherical foreign bodies in the ear canal and nose is presented and discussed The added advantage of using an adenoid balloon to prevent aspiration of nasal foreign bodies is emphasized Lack of damage to the meatus of the external auditory canal is an added feature of the new forceps

124 West Princeton Avenue

CARDIAC REFLEX UPON LARYNX

To The Editor

The nerve of T Drobnik is known in Europe It connects the areas of the larynx, especially those of the inferior constrictor, with the heart, lungs, pericardium, and other viscera[1]

Clinicians should take records of the voice of the patient, and some reflex, in tone, and in expression, will be evident, even some reflex causing shortness of breath, on excretion, as in forced speech

THOMAS HORACE EVANS, M D ,
350 South Main St , Freeport, L I N Y

REFERENCE

1 EVANS, T H "Pretracheal" Nerve *Anesthesiology*, 20 2, March April, 1959

AMERICAN ASSOCIATION FOR CLEFT PALATE REHABILITATION

The American Association for Cleft Palate Rehabilitation will hold its Eighteenth Annual Convention at the Brown Palace Hotel, Denver, Colo , Thursday, Friday and Saturday, May 12, 13 and 14, 1960

This Association is composed of medical, dental and paramedical specialists who are interested in the rehabilitation of persons with cleft lips and palates

SCIENTIFIC PROGRAM OF THE AMERICAN OTOLOGICAL SOCIETY, INC

The Deauville Hotel, Miami Beach, Fla, March 13-14, 1960

Early Embryology of the Auditory Ossicles and Associated Structures in Relation to Certain Anomalies Observed Clinically

Barry J Anson, Ph D (Med Sc)
Jerome S Hanson, M S (by invitation), and
Shatik F Richany, Ph D (by invitation)

Tissue Reaction Following Reconstruction of the Oval Window in Experimental Animals

Richard J Bellucci, M D, and
Dorothy Wolff, Ph D, (by invitation)

Human Cochlear Responses to Sound Stimuli

John L Bordley, M D.,
Robert J Ruben, A B (by invitation), and
George R Nager, M D (by invitation).

Some Physiological Factors in Inner Ear Deafness

Merle Lawrence, Ph D

Distribution of Hearing Loss in Various Population Centers

Aram Glorig, M D.

The Management of Middle Ear Lesions Simulating Otosclerosis

Francis A Sooy, M D (by invitation)

Experiences with the Stapedectomy Technique of Shea for the Surgical Treatment of Otosclerosis

David Myers, M D,
Woodrow D Schlosser, M D (by invitation), and
Richard A Winchester, Ph D (by invitation)

Vein Plug Stapedoplasty
Clair M Kos, M D

Partial Stapedectomy
J V Hough, M D (by invitation)

Experimental and Clinical Experiences in Oval Window Surgery
Harold Schuknecht, M D
(Open discussion of last four papers)

Acceleration as a Means of Determining the Sensitivity of the Components of the Non-Auditory Membranous Labyrinth
Walter H Johnson, Ph D (by invitation),
J Brydon Smith, M D , and
Joseph A Sullivan, M D

Localization of Monaurally Impaired Listeners
Richard Viehweg, M A (by invitation)

Deafness Due to Mumps, Inner Ear Pathology
John R Lindsay, M D , and
Patricia R Davey, FRCS (Ed) (by invitation)

Action of Enzymes on Effusions Obtained from the Middl Ear
Carl F Gessert, Ph D (by invitation),
Elizabeth S Baumann (by invitation), and
Ben Senturia, M D

Tumors of the Temporal Bone in Their Reciprocal Behavior
George Kelemen, M D

Labyrinthectomy
Terence Cawthorne, FRCS

The Lurking Latent Cholesteatoma
Victor Goodhill, M D

PROGRAM FOR THE SCIENTIFIC SESSION OF THE AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC

Miami Beach Fla

March 15, 16, 17, 1960

"The Influence of Deafness on the Creative Instincts'

Mr Terence Cawthorne, London England, *Guest of Honor*

"Tympanoplasty" (A Panel Discussion)

Moderator Albert C Furstenberg, M D , Ann Arbor, Mich Participants Harold F Schuknecht, M D , Detroit, Mich , Joseph Sullivan, M D , Toronto, Canada , and Nils Richtner M D , (By Invitation), Stockholm, Sweden

Open Discussion

"Dysmetria in Post-caloric Nystagmus Its Clinical Significance"

Malcolm H Stroud, M D , (By Invitation), St Louis, Mo , and J Santiago Riesco M D , (By Invitation), Santiago, Chile

Open Discussion

"Aural Cholesteatoma Experimental Observations"

John R Lindsay, M D , and Cesar Fernandez, M D , (By Invitation), Chicago, Ill

Open Discussion

"The Value of Laryngograms in the Diagnosis of Laryngeal Lesions"

Joseph H Ogura, M D Bruce Ellis, M D , Sumner Holtz, M D , (By Invitation) , Malcolm McGavran, M D , and William Powers, M D , all of St Louis, Mo

Open Discussion

"Tonsillectomy for Acute Peritonsillar Abscess"

Benjamin M Volk, M D and E C Brandow, Jr , M D , (By Invitation), Albany, N Y

Open Discussion

"Method for the Identification and Localization of Cerebrospinal Fluid Rhinorrhea and Otorrhea"

G O'Neil Proud, M D , and F R Kirchner, M D , (By Invitation), Kansas City, Mo

Open Discussion

"Studies on the Effect of Heparin on Inflammation and Allergy"—Motion picture

David A Dolowitz, M D , Salt Lake City, Utah

Open Discussion

"Stapes Surgery—Its Present Status"

Howard P House, M D , Los Angeles, Calif

Open Discussion

"Isolated Keratosis of the Larynx, Its Relation to Carcinoma"

Malcolm A McGavran, M D , (By Invitation), and Walter C Bauer, M D , (By Invitation), St Louis, Mo

Open Discussion

"Connective Tissue A Synthesis of Modern Thought and Its Impact on the Understanding of Nasal Disease"—Candidate's Thesis

Alex Weisskopf, M D , San Francisco, Calif

"The Fractured Mandible—A Method of Treatment for the Otolaryngologist"—Candidate's Thesis

Walter A Petryshyn, M D , Flemington, N J

"The Procurement and Processing of Endoscopic Biopsies"

Paul H Holinger, M D , Kenneth C Johnston, M D , and Edwin F Hirsch, M D , (By Invitation), all of Chicago, Ill

Open Discussion

PROGRAM FOR AMERICAN MEDICAL ASSOCIATION SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY, 109th ANNUAL MEETING

June 15-17, 1960, Miami Beach, Fla

Wednesday, June 15, 1960

Joint Session of the Section on Laryngology, Otology and Rhinology, Section on the Diseases of the Chest and Section on Radiology

1 Panel Discussion on Tumors of the Trachea and Bronchi

Moderator Dr Paul H Holinger, Chicago 11, Ill

Endoscopist Dr Herbert W Schmidt Rochester, Minn

Radiologists Dr Barton R Young Philadelphia, Pa ,
Dr. Wendell G Scott St Louis, Mo

Surgeons Dr Robert J Jensik Chicago 4, Ill ,
Dr Robert W Jamplis, Palo Alto, Calif

2 Panel on Disseminated Diseases of the Chest

Moderator Dr Harold O Peterson, Minneapolis, Minn

Internist Dr James A Wier, Denver, Colo

Chest Physician Dr. David Radner, Chicago, Ill

Endoscopist Dr Walter H Maloney, Cleveland 6, O

Radiologists Dr Eugene F Van Epps, Iowa City Ia ,
Dr Clyde A Stevenson Spokane, Wash

Surgeons Dr Donald L Paulson, Dallas, Tex
Dr William Tuttle, Detroit, Mich

Thursday, June 16, 1960

Audiology Its Progress and Problems

Kenneth O Johnson, Ph D , Executive Secretary, American Speech and Hearing Association Washington 6, D C , (By Invitation)

External Otitis Specific Therapy Based on Accurate Diagnosis
Edley H Jones, Vicksburg, Miss

Address of Guest of Honor
Louis H Clerf, St Petersburg 2, Fla

Nasal Tip Surgery
Sam H Sanders, Memphis 4, Tenn

Address of Section Chairman
Paul H Holinger, Chicago 11, Ill

Problems and Complications in Head and Neck Surgery
George A Sisson, Syracuse 10, N Y , and Norman E Johnson

The Long-Term Care of the Laryngectomized Patient
George F Reed, Massachusetts Eye and Ear Infirmary, Boston, Mass

Friday, June 17, 1960

Cystadenoma of the Parotid and of the Larynx
Arthur J Kuhn, Hammond, Ind

Effect of Premarin on the Ground Substance
Maurice Schiff, Capt (MC) USN, U S Naval Hospital, Oak Knoll, Calif

Cervico-Facial Congenital Anomalies
Richard T Farrior, Farrior Clinic, Tampa 6, Fla

Incidence of Hearing Impairment in a Medical Center Population
Edmund P Fowler, Jr , M D , and Thomas H Fay, Ph D , New York 32, N Y

Otometric Operations
George D Albers, Grand Rapids 3, Mich

Five Year Results of Stapes Mobilization and Current Results with Vein Plug Stapedeoplasty

C M Kos, University Hospitals, Iowa City, Ia

Fenestration of the Oval Window

John J Shea, Jr, Memphis 4, Tenn

Use of Ear Canal Skin in Myringoplasty

William F House, Los Angeles 57, Calif

Problems with Tympanoplasty

Edward C Brandow, Jr Albany 10, N Y

THE MOUNT SINAI HOSPITAL

An intensive Postgraduate Course in Rhinoplasty, Reconstructive Surgery of the Nasal Septum and Otoplasty will be given July 16, 1960, through July 29, 1960, by Dr Irving B Goldman and staff at the Mount Sinai Hospital in affiliation with Columbia University.

Candidates for the course should apply to Registrar for Postgraduate Medical Instruction, The Mount Sinai Hospital, 5th Avenue and 100th Street New York 29 N Y

CASSELBERRY AWARD OF THE AMERICAN LARYNGOLOGICAL ASSOCIATION

A sufficient fund having accrued from the Casselberry Fund encouraging advancement in the art and science of Laryngology and Rhinology, this sum is now available in part or as a whole, for a prize award Theses must be in the hands of the Secretary of the American Laryngological Association prior to January 1 of any given year

The Award is a prize of money with accompanying certificate signed by the officers of the American Laryngological Association The sum of money will be agreed upon by the Council of the Association after the manuscript has been evaluated by the Award Committee It may be awarded in whole or in part among several contestants

Eligible contestants may be *1* hospital interns, residents, or graduate students in Rhinology and Laryngology, *2* an individual with an M D degree who is actively practicing or teaching Rhinology and Laryngology in the Americas, *3* any scientific worker in the field of Rhinology and Laryngology

Manuscripts shall be presented to the Secretary of the Association, Dr Lyman G Richards, 12 Clovelly Road, Wellesley Hills 81, Mass, under nom de plume which shall in no way indicate the author's identity There shall also be a sealed envelope bearing the nom de plume and containing a card showing the name and address of the contestant which the Secretary shall keep in his possession

Manuscripts must be limited to 5000 words and must be typewritten in double spacing on one side of the sheet The thesis shall not have been published elsewhere before submission

The successful thesis shall become the property of the American Laryngological Association, but this provision shall in no way interfere with publication of the thesis in the Journal of the author's choice Unsuccessful contributions will be returned promptly to their authors

The Award which will be made at the Annual Meeting of the American Laryngological Association shall be based on

1 Originality of material

2 Scientific and clinical value

3 Suitability for this Award

4 Method of presentation as to style, illustrations and reference

The maximum amount of the Award shall not exceed $200 00

TEMPLE UNIVERSITY POSTGRADUATE COURSES

Postgraduate course in laryngology and laryngeal surgery, April 4th to 15th, and September 26th to October 7th, 1960

Postgraduate course in bronchoesophagology, February 1st to 12th, and November 7th to 18th, 1960

These courses are to be given in the Department of Laryngology and Bronchoesophagology, Temple University Medical Center, under the direction of Drs Chevalier L Jackson and Charles M. Norris

The tuition fee for each course is $250 Application and information may be obtained by writing to Jackson-Research, Lab 604, Temple University Medical School, 3400 N Broad street, Philadelphia 40, Pa

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Meeting Miami Beach Fla June 13 17, 1960

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Meeting Deauville Hotel Miami Beach Fla March 13 14, 1960

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Annual Clinical Session Chicago Ill (Definite time and place to be announced later)
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Meeting Palmer House, Chicago, Ill, October, 1960

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Secretary-Treasurer Dr Spriestersbach, Ph D, Department of Otolaryngology, University Hospital, Iowa City, Ia
Meeting Palace Hotel, Denver, Colo, May 12 14, 1960

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Meeting Chicago, Ill, October 1960

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Secretary Dr Donald M MacRae, 324 Spring Garden Road, Halifax, Nova Scotia
Meeting Jasper Park Lodge, Jasper National Park, Alberta, June 10 12, 1960

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Secretary Dr Will P Pirkey, Denver, Colo

COLUMBUS, OHIO, OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

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Secretary Dr M L Battles
Meetings First Monday of October through May, University Club Columbus, O

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Secretary Treasury Prof Dr Antonio Carrascosa
Pro Secretary of the Interior Prof Dr Carlos P Mercandino
Pro Secretary of the Exterior Prof Dr James A del Sel
Pro Secretary of the Treasury Dr Jorge Zubizarreta

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First Vice President Dr William H Anderson, Jr, Ocala, Fla
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FOURTH LATIN AMERICAN CONGRESS OF OTORINOLARINGOLOGIA

President Dr Dario
Secretary
Meeting

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President Dr Van D Rathgeber
Vice President Dr William Skokan
Secretary Treasurer Dr Paul Rockwell

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Vice President Dr Joseph Freeman
Secretary Treasurer Dr H Carlton Howard
Meeting Quarterly in March, May, October and December on the second Thursday of the month, 6 30 P M , at the McAllister Hotel, Miami, Fla

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President Dr Jo Ono Tokyo, Japan
Secretary Dr Chevalier L Jackson, 3401 N Broad St , Philadelphia 40, Pa , U S A
Meeting

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President Elect Dr Dick H Underwood
Secretary Dr James T Robison, 4620 J C Nichols Parkway, Kansas City Mo
Meeting Third Thursday of November, January, February and April

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Chairman of Ophthalmology Section Dr Carroll A McCoy
Secretary of Ophthalmology Section Dr Philip D Shanedling
Chairman of Otolaryngology Section Dr Robert W Godwin
Secretary of Otolaryngology Section Dr Francis O'N Morris
Place Los Angeles County Medical Association Bldg 1925 Wilshire Blvd , Los Angeles, Calif
Time 6 30 P M last Monday of each month from September to June, inclusive—Otolaryngology Section 6 30, first Thursday of each month from September to June, inclusive—Ophthalmology Section

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A STUDY OF SPASTIC DYSPHONIA

Neurologic and Electroencephalographic Abnormalities*

EVELYN ROBE Ph D ,
JOEL BRUMLIK M D
and
PAUL MOORE Ph D ,

Chicago, Ill

I INTRODUCTION

Spastic dysphonia often referred to as hysterical dysphonia, has been considered a nosologic entity It is usually not defined in terms of fundamental disturbances in neurophysiology The study reported here, however, indicates that spastic dysphonia is a manifestation of disordered function of the central nervous system, and not a disease *in sui generis*

Berendes[2,3] has described this condition as a disorder of phonation "which is characterized by a strained, creaking and choked vocal attack, tense and squeezed voice sound and is accompanied by extreme tension of the entire phonatory system " The present authors have also observed that there is usually a momentary phonatory interruption that occurs most frequently on initial vowel sounds Whispered speech and extremely breathy phonation are ordinarily spoken without any hesitancy or other abnormal speech characteristics

The present study was prompted in part, by the singular

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*From the Departments of Otolaryngology and Neurology and Psychiatry of Northwestern University Medical School aided by a grant from the United Cerebral Palsy Research and Education Foundation (of Greater Chicago)

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lack of efficacy of voice therapy and psychotherapy in spastic dysphonia, and in part by the lack of constant anamnestic data We have elected to study a group of patients with this clinical disorder for possible neurologic and electroencephalographic abnormalities To our knowledge, there has been no previous attempt to define this disorder by parameters other than psychiatric

Arnold[3] has suggested that spastic dysphonia might be due to removal of higher suppressor influences, allowing primitive subcortical centers to act without inhibition ("by the unconscious withdrawal of a shattered personality from the threats of daily life") , but the fundamental neurophysiologic mechanism must be defined and the terminology made specific The use of such a term as "suppressor influences" may refer to psychoanalytic suppression or to the function of one or more cortical suppressor strips If a disease has a neurologic basis, the description of the underlying processes must be in specific terms germane to the field

Segre[13, 41] has stated that spastic dysphonia is an illness of the third and fourth decades of life The ratio of males to females with this disease varies among authors The precipating factors mentioned in the literature include excessive use of the voice and traumas of daily living The authors beg to be excused from a discussion of psychodynamic abnormalities in spastic dysphonia for, while they may be present, this study is concerned specifically with the physiology of the central nervous system

II MATERIALS AND METHODS

Ten patients with a clinical diagnosis of spastic dysphonia were selected for study Each was given the five different types of tests and examinations indicated in the following paragraphs

A *Examination of the Speech Organs and Adjacent Structures*

Each patient received a complete ear, nose and throat examination by at least two qualified otolaryngologists This evaluation included indirect laryngoscopy with special reference to a study of the movement of the true and false vocal cords In addition, the nasal, pharyngeal and auditory passages were scrutinized Three types of voice production were recorded for study a standard passage was read, the total pitch range was sung, and a representative sample of conversational speech was spoken Finally, a pure-tone audiometric examination was made for each subject, and additional special audiometric tests for central disturbances were administered to five of the patients [21]

B History

An extensive history was obtained from each patient, including neurologic history with special reference to precipitating factors, familial neurologic disease and previous neurologic disease in the patient A review of systems included specific questions as to a history of vertigo, diplopia, headaches, scotomata, weakness, paresthesias, unsteady gait, syncope and convulsions

A history of the origin and development of the voice disturbance was obtained from each patient

C Neurologic Examination

Each patient was given a complete neurologic examination, including an evaluation of the function of the cranial nerves, motor power, reflexes, coordination and sensation A search was made not only for gross signs, but also each patient was combed for delicate signs of central nervous system disturbance

D Electroencephalographic Study

Each patient was studied with standard electrode placement in frontal, anterior temporal, temporal, parietal and occipital areas Monopolar and bipolar recording was used Sleep tracings were obtained in all cases by the use of secobarbital sedation Hyperventilation and photic drive stimulation techniques were employed The alpha blocking response was recorded in all cases lack of efficacy of voice therapy and psychotherapy in spastic dysphonia, and in part by the lack of constant anamnestic data We have elected to study a group of patients with this clinical disorder for possible neurologic and electroencephalographic abnormalities To our knowledge, there has been no previous attempt to define this disorder by parameters other than psychiatric

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E Laboratory Studies

Those patients who merited further work-up received appropriate tests, including lumbar puncture, spinal fluid examination, skull and chest films, visual field examination, blood chemistries and urinalysis Special tests, such as protein bound iodine and urine copper determinations, were performed as indicated

TABLE I

Examination of the Speech Apparatus and Adjacent Structures in Spastic Dysphonia [*]

Patient	Age	Sex	Laryngoscopy	ENT Exam	Audiometry	Recordings[†]
S S	25	F	—	—	—	4
C Y	36	F	—	—	—	1
E F	38	F	—	—	—	1
E T	44	M	—	—	—	4
I R	45	F	—	—	—	4
S K	46	M	—	+	—	3
F O	50	F	—	—	—	3
J O	56	F	—	—	—	2
E R	59	F	—	—	—	2
M C	63	F	—	—	—	2 3

[*] See text for explanation + = abnormal findings — = negative

[†] Scale of severity (average of the evaluation of four examiners) 1 mild 2 moderate 3 severe 4 very severe

III RESULTS OF THE STUDY

A Examination of the Speech Apparatus and Adjacent Structures (See Table I)

The age range included in this study is 25 to 63 years The male-female sex ratio is one to four Laryngoscopy in all patients revealed normal structure No sign of ventricular phonation was observed in any patient Examination of the nasal, pharyngeal and auditory passages was completely normal in all cases except S K, who had an anterior septal perforation

Audiometry (pure-tone audiometry and special tests) was within normal limits in all cases Recordings of voice were analyzed objectively and graded by four specialists in voice disorders according to the scale given in Table I

TABLE II HISTORICAL DATA IN SPASTIC DYSPHONIA

Patient	Age Onset	Age Pt	Duration (yrs)	Hnd[1]	Ppt	Fam[3]	Neur[4]	Ang[5]	Nec[6]	Fat[7]	Prev Rx[8]
S S	20	25	5	L	+	—	+	+	+	+	V, P
C Y	32	36	1	M	—	+	+		+	+	V
E F	21	38	14	R	—	+	+		+	+	—
E T	12	14	2	R	+	+	+	+	+	+	V, H
I R	30	45	15	R	+	+	+	+	+	+	V, P, A
S K	10	16	6	R	—	—	+	+	+	+	V
F O	47	50	3	R	+	—	—	—	+	+	V
J O	51	56	5	M	—	—	—		+	+	V
E R	56	59	3	R	+	—	+	+	+	+	V
M C	63	63	½	R	—	—	+	—	+	+	V
Per Cent					50	40	80	50	100	100	

[1]Handedness: L—Left R—Right M—Mixed

Precipitating Factors: A history of cold, laryngitis or flu prior to the onset of voice disturbance

[3]A family history of neurologic disease

[4]A history of neurologic symptomatology in the patient

[5]Anger: improvement in the voice problem when angry

[6]Necessary: worsening of voice problem when speech is necessary

[7]Fatigue: increase in voice disturbance with fatigue

[8]Previous therapy: V—voice therapy, P—psychotherapy, H—hypnosis, A—intravenous amobarbital

B. *History (See Table II)*

The duration of the illness ranges from six months to 15 years and apparently is not related to the patient's age at the time of onset. Seventy per cent of patients were right handed, 20 per cent ambidextrous and 10 per cent were left handed. Fifty per cent of patients had a history of "flu," "cold," "laryngitis" or upper respiratory infection immediately prior to the onset of voice disturbance. The remainder of patients either could not recall any precipitating factors or mentioned hard work, emotional trauma, etc., as associated with the onset.

Forty per cent of patients had a positive history of neurologic disease in the family. These statements included: *1.* "My mother was born blind because the optic nerves didn't develop" (C. Y.), *2.* a history of intention tremor of the head and hands in mother, sister and brother (E. F.), *3.* a seizure disorder in the father of the patient (E. T.), and *4.* "My mother had a stroke" (I. R.).

Eighty per cent of the patients had a positive history of neurologic disturbance prior to the onset of voice difficulty. These symptoms included: *1.* transient scotomata in both eyes and transient weakness of both legs (S. S.), *2.* tremor of the head and hands of the intention type (E. F.), *3.* paralysis of the lower extremities as a child (S. K.), *4.* a transient episode of clumsiness and weakness of the left leg accompanied by dizziness (E. R.), *5.* several episodes of syncope without convulsions (C. Y.), *6.* attacks of syncope especially associated with coughing and periods of confusion with transient paresthesias in one thumb (E. T.), *7.* paresthesias noted on occasion in the left hand (I. R.), and *8.* intermittent dysphagia (M. C.).

Eighty per cent gave a history of improvement in their voices with emotion (anger). One hundred per cent stated that situations in which talking was necessary aggravated their voice disturbance. Fatigue was universally effective in worsening the symptoms; otherwise, historical findings were not contributory.

TABLE III

Clinical Findings in Spastic Dysphonia

S S
1. Bitemporal pallor of the optic discs
2. Absent abdominal reflexes
3. The left angle of the mouth pulls away better than the right and the patient talks better out of the left side of her face however, this appears to be an asymmetry rather than a true neurological lesion
4. Questionable left pronation sign
5. Hearing lateralizes slightly to the left
6. A tendency to become unsteady as she walks in tandem
7. A suggestion of an adductor response bilaterally when the knee jerks are elicited

C Y
1. Minimal fine rotary tremor of the head of the intention type
2. Absent abdominal reflexes

E F
1. Intention tremor, involving the head and both upper extremities perhaps more so on the left
2. Definite ataxia as the patient walks in tandem tending to fall to the left
3. Deep tendon reflexes of the lower extremities are slightly more brisk on the left with an adductor response on both sides as the knee jerks are elicited, perhaps more prominent on the left
4. The abdominal reflexes are absent
5. There is minimal falling away on both sides as the opposite arm past points
6. The patient tends to swing her right arm better than her left as she walks

E T
1. The tongue protrudes to the left
2. There is minimal falling away of the right upper extremity
3. The right lower limb lies slightly externally rotated as the patient lies upon the examining table
4. The patient swings his left arm slightly better than his right as he walks
5. The left abdominals are greater than the right and the left cremasteric is greater than the right
6. The right knee jerk is slightly greater than the left

I R
1. The left pupil is slightly larger than the right
2. The left face is weak in the manner of a supranuclear facial paresis
3. The left biceps radial and ulnar jerks are greater than the right
4. The right knee and ankle jerks are greater than the left
5. The right lower limb lies slightly externally rotated on the examining table
6. There are questionable Tromner Gonda and Oppenheim responses on the left
7. There is unsustained nystagmus on right lateral gaze with the slow component to the left

S K
1. The left angle of the mouth pulls away better than the right
2. The left platysma contracts better than the right
3. The patient smiles and talks better out of the left side of the face
4. Minimal right pronation sign and falling away
5. The left lower limb lies externally rotated
6. The left biceps triceps radial knee and ankle jerks are greater than the right

TABLE III (Continued)

7 The left abdominals and cremasterics are greater than the right
8 Minimal intention tremor bilaterally
9 Positive Quinquaud sign
10 The right arm swings less well than the left as the patient walks

F O
1 The left pupil is slightly greater than the right
2 The tongue protrudes slightly to the right
3 The right side of the face pulls away a little better than the left The right platysma contracts slightly better than the left The patient talks a little better out of the right side of her face
4 The left biceps, radial and ulnar and knee jerks are slightly greater than the right
5 The patient swings her right arm a little better than the left as she walks
6 The abdominal reflexes are absent

J O
1 The left pupil is slightly larger than the right
2 The right ankle jerk is slightly greater than the left
3 There is an adductor response on eliciting the knee jerks on both sides
4 The right abdominals are slightly more brisk than the left
5 The patient swings her left arm slightly better than the right as she walks
6 There is a more definite flexor response on stroking the right planta than the left
7 There is a fine tremor of the closed eyelids

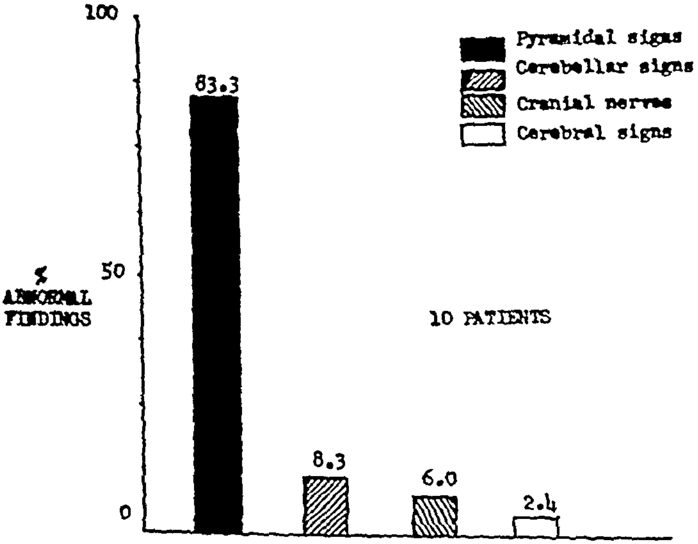
E R
1 Definite snout reflex
2 The left radial reflex is greater than the right
3 The right knee jerk is greater than the left
4 There is a definite left Gonda
5 A minimal falling away of both extremities as the opposite arm past points
6 There is a slight left pronation sign
7 The patient tends to swing her right arm less well than the left as she walks
8 The left lower limb lies slightly externally rotated as the patient lies upon the examining table
9 The patient tends to become unsteady as she walks in tandem and with standing on either foot alone especially on the left

M C
1 A positive snout reflex
2 The right angle of the mouth pulls away slightly better than the left and the patient talks better out of the right side of her face
3 There are bilateral Babinski hand signs, more marked on the right
4 The abdominal reflexes are not elicited
5 The right ankle jerk is greater than the left
6 There is a right Gordon
7 There are bilateral Hoffmann signs and Tromners

Ninety per cent of patients had some previous therapy for their voices Ninety per cent had voice therapy from one or more therapists, 20 per cent had psychotherapy, and one patient each had hypnosis and intravenous amobarbital All these therapies were strikingly ineffective

C Neurologic Examination (See Table III and Figs 1 and 4)

All patients had neurologic signs indicating disease of the central nervous system A total of 84 neurologic signs was elicited, approximately eight per patient By far the most frequently observed abnormalities were pyramidal tract signs (83 per cent), with cerebellar signs also relatively frequent (8 per cent)



NATURE OF ABNORMAL NEUROLOGICAL SIGNS IN SPASTIC DYSPHONIA

(Total abnormal signs = 84 = 8.4 signs per patient)

Fig 1

Forty-three per cent of signs lateralized to the right and 32 per cent to the left cerebral hemisphere (25 per cent were bilateral) If bilateral signs are excluded, right cerebral signs increase to 57 per cent and the left-sided signs to 43 per cent

Coupled with the medical and neurological history, an impression of specific neurologic disease was made in four patients *1* heredofamilial tremor (E R), *2* small cerebral

REPRESENTATIVE ELECTROENCEPHALOGRAMS

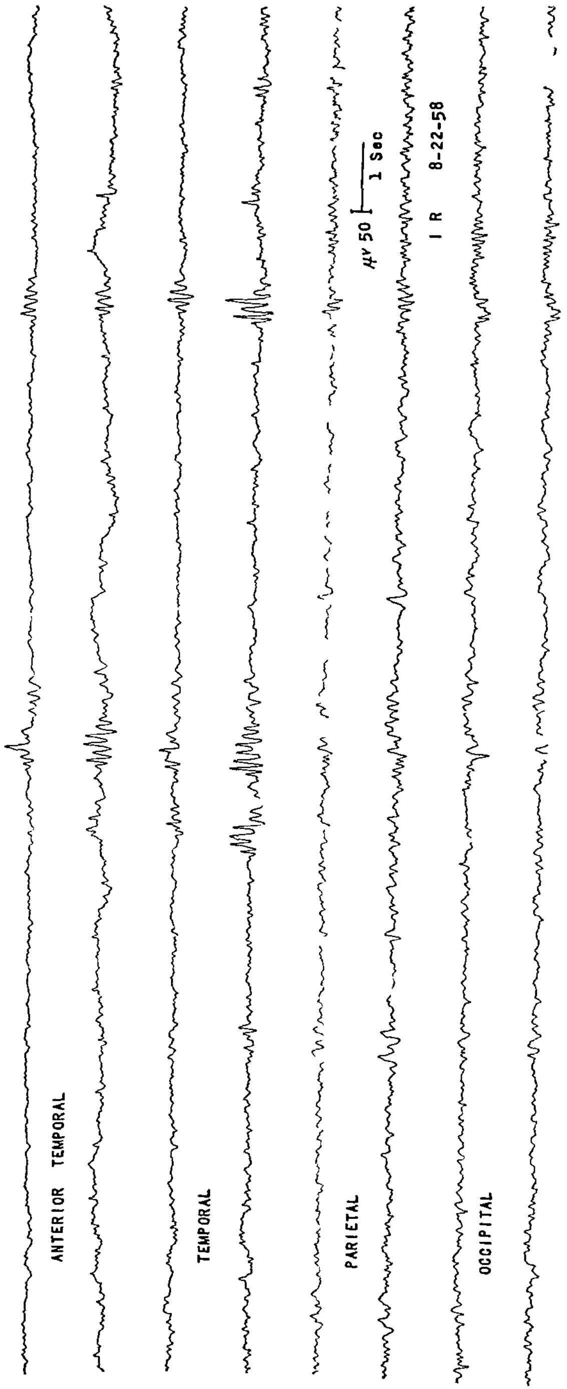


Fig 2-a

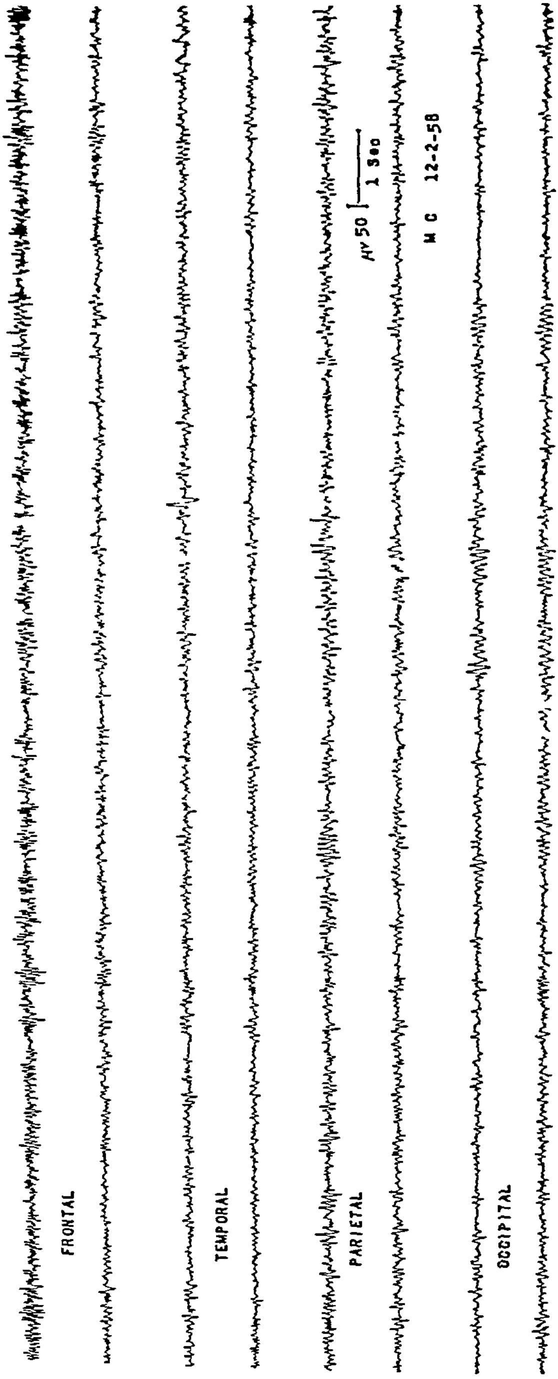


Fig 2-b

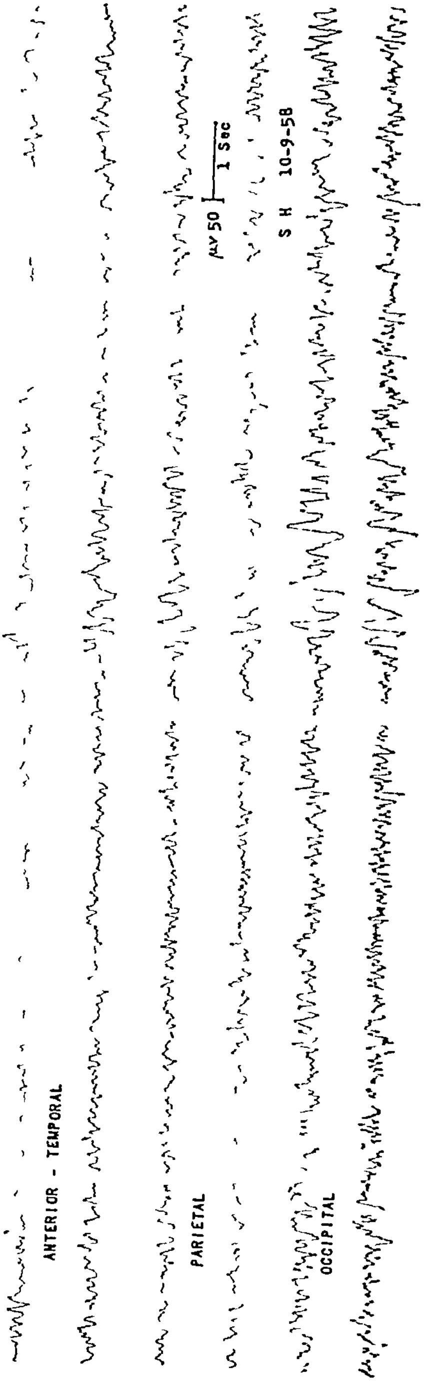
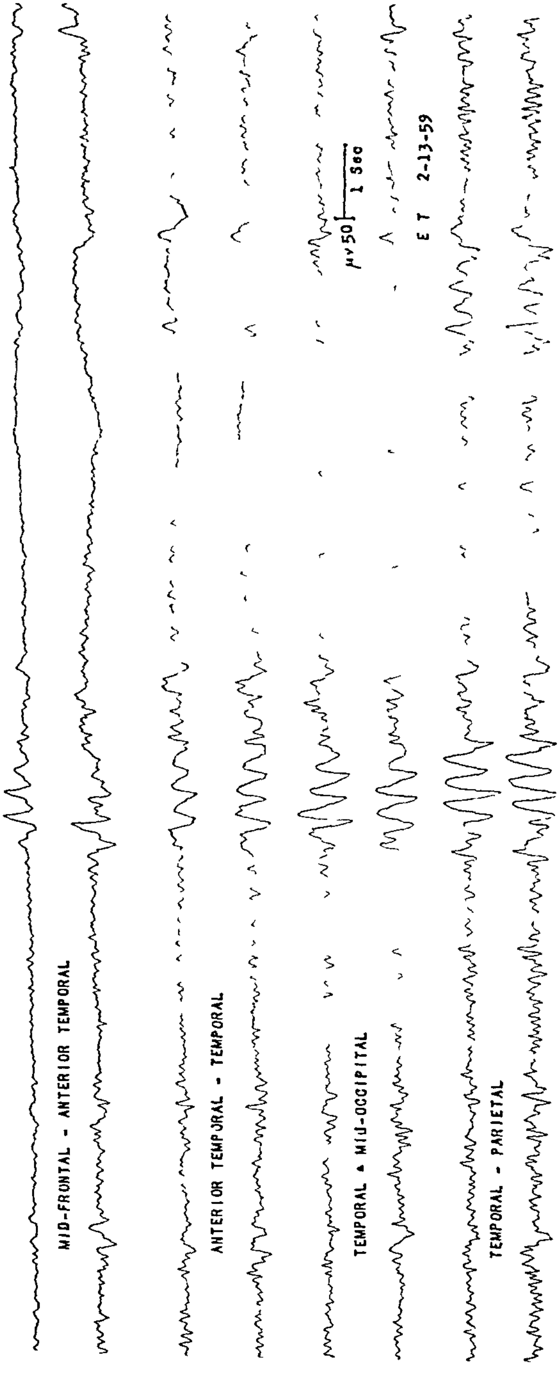


Fig 2-c



E T 2-13-59

Fig 2-d

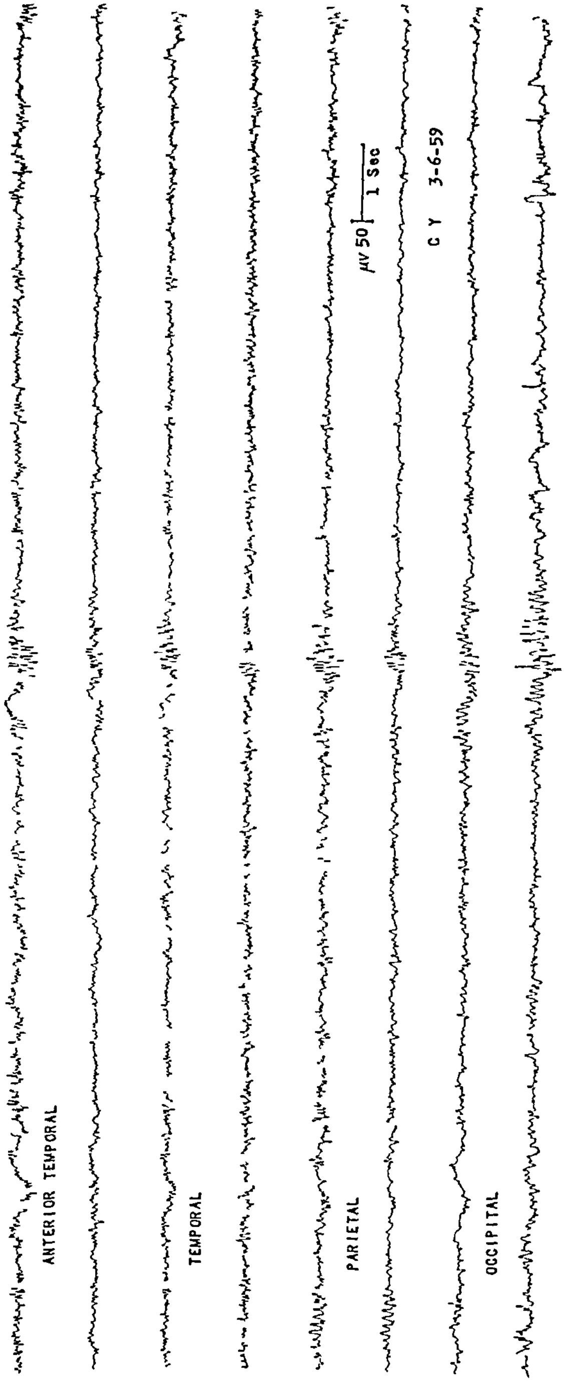
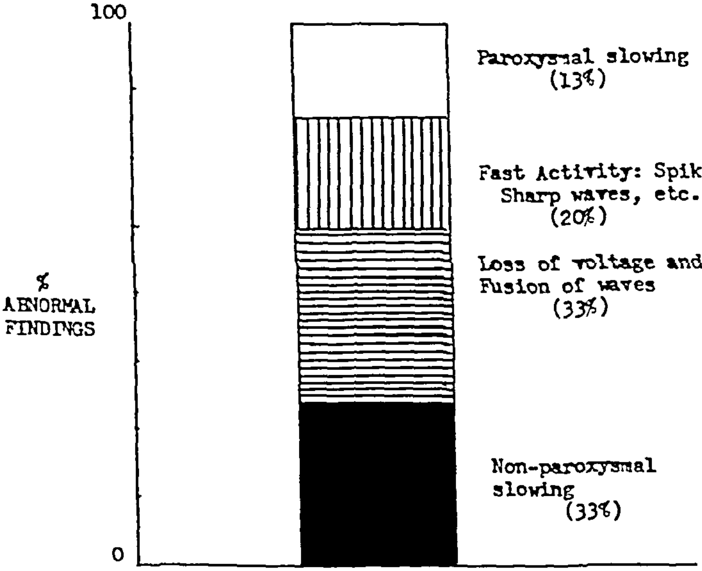

ANTERIOR TEMPORAL
TEMPORAL
PARIETAL
OCCIPITAL
μV 50
1 Sec
C Y 3-6-59

Fig 2-c

thromboses in the frontal area (arteriosclerosis) (M C), 3 transient cerebral ischemic attacks (E R), and 4 multiple sclerosis (S S) The remaining patients had signs and symptoms which did not point to distinct clinical entities, but rather to a diffuse or disseminated disease of the pyramidal and extrapyramidal systems The etiology of the process was not obvious in these cases The possibility of a post-encephalitic process will be discussed below



THE NATURE OF THE ELECTROENCEPHALOGRAPHIC ABNORMALITIES

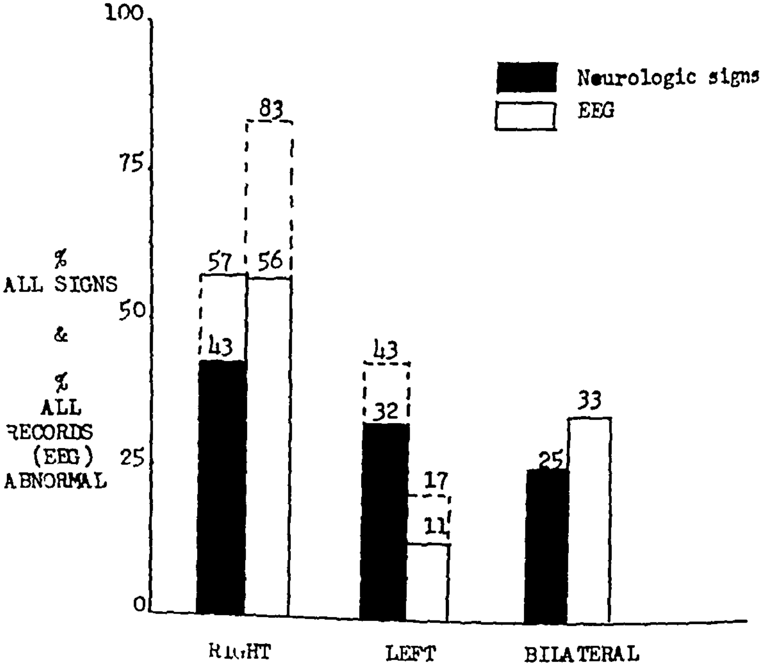
IN SPASTIC DYSPHONIA

(10 PATIENTS. 9 ABNORMAL EEGs)

Fig 3

D *Electroencephalographic Disturbances (See Figs 2, 3, 4 and 5)*

Nine of the ten electroencephalograms taken were abnormal (E F was normal)



THE LATERALITY OF DISTURBANCE IN SPASTIC DYSPHONIA

THE NEUROLOGICAL EXAMINATION

COMPARED WITH THE ELECTROENCEPHALOGRAM.

(Dotted lines indicate the % lateralizing signs and records)

Fig 4

Fifty per cent of patients had records compatible with burst-like discharges Of these five patients, three had findings primarily parietal in location and two mainly frontal In three cases, the findings were bilateral, in one case primarily right sided, and in one case mainly left sided The nature of these paroxysmal discharges were high voltage fast activity in three cases and hypersynchronous slowing in two cases

---

*Though two patients mentioned syncope no patient had a history of clinical seizures

In considering all abnormal findings in the nine cases of abnormal electroencephalograms, no predominant type of abnormality was observed Burst-like discharges (high voltage fast activity and hypersynchronous paroxysmal slowing) were seen in 33 per cent of tracings A reduction in voltage and basic frequency was observed in another 33 per cent of findings, predominantly in the right temporal and right parietal areas Non-paroxysmal slowing constituted the remaining third of the disturbances

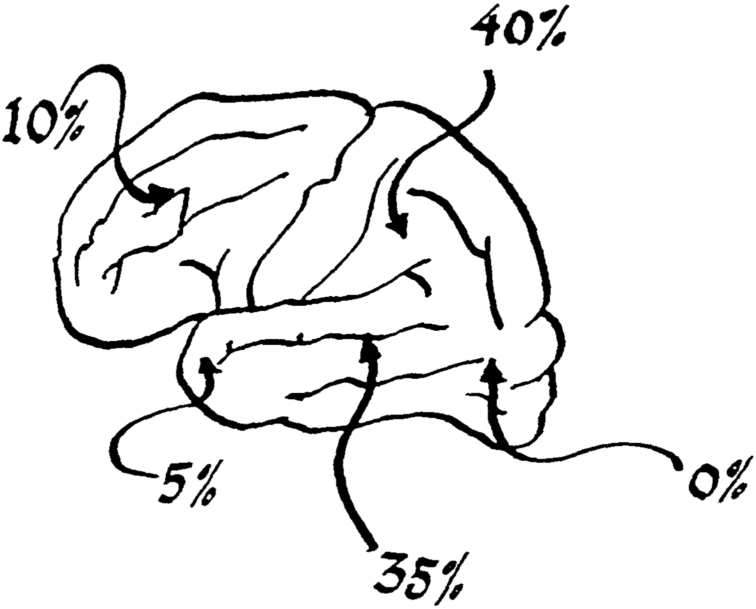


Fig 5 Localization of all EEG findings in spastic dysphonia (10 patients)

The laterality of these abnormalities was 56 per cent right sided, 11 per cent left sided and 33 per cent bilateral Excluding the latter, the right sided findings rise to 83 per cent and the left sided disturbances to 17 per cent

The location of these findings (see Fig 5) is 15 per cent for frontal, anterior temporal and occipital areas combined, as opposed to 75 per cent for temporal-parietal location

In summary, the electroencephalographic findings suggest a cortical disturbance in the temporo-parietal area in the right cerebral hemisphere and paroxysmal in nature

E Laboratory Results (see Table IV)

In four cases out of five with burst-like EEG discharges, the patients were hospitalized for further work-up as indicated in Table IV No spinal fluid finding was abnormal

TABLE IV

Laboratory Results in Spastic Dysphonia

Patient	Visual Field Examination (Central)	Skull Films	Cerebrospinal Fluid Examination*				
			Wassermann	Protein (mg%) Tube 1	Protein (mg%) Tube 3	Cl	Gold curve
S S	Peripheral constriction (to red)	Small punched out lesions (vascular lakes?)	Neg	41 5	33 0	718	0111000000
C Y	Negative	Negative	Neg	29 5	28 0	730	0011000000
I R	Negative	Osteoma, rt occipital bone	Neg	34 5	34 5	717	0111110000
E T	Negative	Negative	Neg	29 5	29 0	742	0111000000

*All cell count within normal limits

Skull films revealed only incidental pathology (S S and I R) Central visual field examinations revealed pathology (peripheral constriction) in one case (S S)

A high erythrocyte sedimentation rate was found in three patients (S S 35, I R 23, J O 27) and glycosuria was noted in one patient (I R), otherwise, blood chemistries were within normal limits, including a protein bound iodine (S K) and a urine copper determination (E F)

IV DISCUSSION

A survey of the experimental neurophysiology of phonation and the contributions of clinical neurology are pertinent to the present discussion Both fields of study offer evidence to explain the phenomena of spastic dysphonia

A The Neurophysiology of Vocalization

1 The frontal-parietal areas Furstenberg[11] has provided direct evidence that laryngeal closure may follow cortical stimulation The larynx of primates was visualized by direct laryngoscopy and various foci on the cortex were stimulated Laryngeal closure, with either apnea or diaphragmatic contraction, resulted from stimulation of five areas *1* the anterior temporal lobe, *2* the frontal operculum, *3* the anterior island of Reil, *4* the temporal-parietal area, and *5* the ventral part of the motor cortex The lowest threshold for response was associated with the first-named area Sugar, et al,[46] has described a second motor area in the monkey, buried deep in the Sylvian fissure When the para-insular portion is stimulated, adduction-abduction movements of the vocal cords are observed under direct vision From the adjacent insula, stimulation produced respiratory slowing and arrest

Penfield and Jasper[38] describe vocalization that follows stimulation of the motor cortex (lower part of area 4) as a long-drawn-out sound made by symmetrical use of mouth, pharynx, larynx and muscles of mastication Occasionally respiratory arrest has been produced by stimulation of the lower part of area 4 Penfield and Rasmussen[40] describe similar localization Either speech arrest or vocalization may be produced by stimulation of *1* the superior frontal gyrus, or *2* the lower part of area 4 So-called aphasic arrest can be elicited by stimulating *1* area 44, *2* the supramarginal gyrus, and *3* the posterior temporal lobe, moreover, removal of the pre-central gyrus will cause a temporary dysphagia and dysphonia The authors believe that since these speech areas can be isolated from the rest of the cortex without producing disturbance in speech, the pathways for vocalization are subcortical

2 *The temporal lobe* Kaada[22] described various rhinencephalic areas in lower animals which produce alterations in respiration following stimulation The extrapyramidal nature of the pathways involved was demonstrated Other authors have confirmed the observation that stimulation of the temporal tip, limbic area and amygdaloid complex can produce respiratory arrest in lower animals[13], moreover, from these areas (in the monkey),[47] vocal responses have been produced similar to those the animal usually makes Characteristically, the sound continues after the stimulation stops

Baldwin, et al.,[5] noted that a patient reported at surgery for temporal lobectomy, "I felt like my throat was closing up somewhat, and I could not talk." This comment was made as the para-amygdalar area was stimulated.

Ablation of the tip of the temporal lobe also has its effect on speech production. Bucy[10] states that, following temporal lobectomy in monkeys, there is a loss of vocal activity—the animals remain mute. Removal of the temporal lobes in man produces profound emotional changes. Boshes[9] has stressed the intimate relationship of the temporal lobe to the limbic system and emotion. Other effects of temporal lobectomy have been outlined by Bucy and Kluver.[11]

Thus the temporal lobe is intimately connected with emotional-behavioral responses on the one hand, and autonomic nervous system responses on the other, and is related to vocalization and respiration. The subcortical pathways needed for cortical expression have been outlined anatomically and physiologically[1, 4, 18, 23, 28, 30, 42, 48] as they proceed through the extra pyramidal system. An interesting condition known as "pathological" or "involuntary" laughing and crying has led Monrad-Krohn[32] and Wilson[17] to postulate separate motor systems for "voluntary motion" and "emotional movement." Monrad-Krohn states that the latter is normally under suppressor control from the former and may appear in disease states. Wilson, in 1924,[17] postulated a coordinated facio-vocal-respiratory center in the upper pons, which is normally under double control—emotional and voluntary—with symptoms resulting if one or the other is destroyed. He cites the pathological laughing of the patient with pseudobulbar palsy as an example of destruction of the voluntary system, and the masked facies of the Parkinsonian as a loss of emotional system function.

3. The brainstem. That a center for vocalization exists in the brainstem has been demonstrated by Magoun, et al.[31] Stimulation of the periaqueductal gray matter and tegmentum in cats and monkeys produces coordinated facio-vocal activity resembling that seen during unpleasant emotion in the normal animal. Such responses are seen from stimulation of the hypothalamus and in the decerebrate cat, hence this must be an "efferent" center While stimulation proceeds, various sympathomimetic effects, including mydriasis, are noted. Luchsinger[29] reported mydriasis as a specific accompaniment of attempted speech in the patient with spastic dysphonia In addition, ipsilateral mydriasis follows amygdaloid stimulation[26] Kelly, et al,[24] abolished facio-vocal activity by lesions placed in the periaqueductal gray matter and tegmentum of cats The authors state that this is a specific center and is concerned not solely with the integration of facio-vocal impulses but also with the precipitation of their integrated activity "at times when it forms an appropriate part of the animal as a whole"

B Contributions of Clinical Neurology

Classical neurology has concerned itself largely with the effects of ablative cortical lesions on the speech mechanism The cardinal sign of cortical speech dysfunction is an alteration of language Hughlings Jackson[20] distinguishes two varieties of speech automatic and intellectual An aphasic patient may lose voluntary (intellectual or propositional) language, but retain emotional speech, *i e*, he may laugh, cry, vary the tone of his voice and sing This is extremely reminiscent of the so-called "psychogenic" variations in the voice disturbance in spastic dysphonia Hughlings Jackson felt that the right (non-dominant) cerebral hemisphere was for automatic, and the left hemisphere was for intellectual, speech Kussmaul[27] mentions pseudobulbar palsy, where voluntary speech may be paralyzed but speech accompanying emotion is preserved, moreover, the symptoms of organic neurologic disease are frequently altered by emotion[37]

As far as the role of the non-dominant hemisphere in speech is concerned, very little basic information is extant Nielsen[34] went so far as to say "Even today there is no unanimity of opinion concerning localization of cerebral function in the sphere of language, and this lack of agreement is dependent on our ignorance of the function of the minor cerebral hemisphere" Nielsen has little new to add on the subject[33 34 35] and cites the theory of Jackson as the probable function of the non-dominant hemisphere in speech Both Head[19] and Freud[14]

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Classical neurology has concerned itself largely with the effects of ablative cortical lesions on the speech mechanism The cardinal sign of cortical speech dysfunction is an alteration of language Hughlings Jackson[20] distinguishes two varieties of speech automatic and intellectual An aphasic patient may lose voluntary (intellectual or propositional) language, but retain emotional speech, *i e* he may laugh, cry, vary the tone of his voice and sing This is extremely reminiscent of the so-called "psychogenic" variations in the voice disturbance in spastic dysphonia Hughlings Jackson felt that the right (non-dominant) cerebral hemisphere was for automatic, and the left hemisphere was for intellectual, speech Kussmaul[27] mentions pseudobulbar palsy, where voluntary speech may be paralyzed but speech accompanying emotion is preserved, moreover the symptoms of organic neurologic disease are frequently altered by emotion[37]

As far as the role of the non-dominant hemisphere in speech is concerned, very little basic information is extant Nielsen[34] went so far as to say "Even today there is no unanimity of opinion concerning localization of cerebral function in the sphere of language, and this lack of agreement is dependent on our ignorance of the function of the minor cerebral hemisphere" Nielsen has little new to add on the subject[33 34 37] and cites the theory of Jackson as the probable function of the non-dominant hemisphere in speech Both Head[19] and Freud[14]

Baldwin, et al,[7] noted that a patient reported at surgery for temporal lobectomy, "I felt like my throat was closing up somewhat, and I could not talk" This comment was made as the para-amygdalar area was stimulated

Ablation of the tip of the temporal lobe also has its effect on speech production Bucy[10] states that, following temporal lobectomy in monkeys, there is a loss of vocal activity—the animals remain mute Removal of the temporal lobes in man produces profound emotional changes Boshes[9] has stressed the intimate relationship of the temporal lobe to the limbic system and emotion Other effects of temporal lobectomy have been outlined by Bucy and Kluver[11]

Thus the temporal lobe is intimately connected with emotional-behavioral responses on the one hand, and autonomic nervous system responses on the other, and is related to vocalization and respiration The subcortical pathways needed for cortical expression have been outlined anatomically and physiologically[1, 4, 18, 23, 28, 30, 12, 18] as they proceed through the extra pyramidal system An interesting condition known as "pathological" or "involuntary" laughing and crying has led Monrad-Krohn[32] and Wilson[17] to postulate separate motor systems for "voluntary motion" and "emotional movement" Monrad-Krohn states that the latter is normally under suppressor control from the former and may appear in disease states Wilson, in 1924,[17] postulated a coordinated facio-vocal-respiratory center in the upper pons, which is normally under double control—emotional and voluntary—with symptoms resulting if one or the other is destroyed He cites the pathological laughing of the patient with pseudobulbar palsy as an example of destruction of the voluntary system, and the masked facies of the Parkinsonian as a loss of emotional system function

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yet that this small group of patients manifests so many and such significant signs, leads to the admonition to perform a minute neurologic examination on each patient with spastic dysphonia. The patient with this disease almost assuredly will have neurologic abnormalities.

The authors believe that the most significant findings in this study are electroencephalographic. Several conclusions can be drawn: *1* there is an electrical cortical disturbance in spastic dysphonia, though the primary disturbance may be partially subcortical and cause cortical disturbances over the projection systems; *2* the commonest location of the disturbance is temporal-parietal; *3* its laterality is primarily in the right (non-dominant) cerebral hemisphere, and *4* its nature is mainly burst-like or paroxysmal. Of interest are electroencephalographic findings in stutterers which are almost identical to those found in this study.[36, 4] Coupled with the neurologic findings in these patients, a disturbance in neural function and possibly structure is highly suspect, if not definite, in spastic dysphonia.

Several patients have noted difficulty in speaking when they *have* to, but ease up when they are not so pressed or when swearing. This is reminiscent not only of certain aphasic patients (see above) but also of the "separate" emotional and voluntary motor systems proposed by Monrad-Krohn and Wilson.[32, 47] Arnold[3] mentioned that whispering, choral speaking, singing, talking in sleep are not affected in spastic dysphonia. Coughing, laughing and moaning are preserved. "Even singing may be perfectly normal as long as the patient does not attach any meaning to his emotional expression . . . ."

We are impressed by the high incidence of extrapyramidal signs in our patients. This would point to disturbances in subcortical pathways, which every author seems to think are the paths for speech production; moreover, the intimate connection of the temporal lobe with the pathways for emotional expression suggest a possible mechanism by which psychic factors alter the symptoms of spastic dysphonia.

Finally a possible post-encephalitic etiology in some cases of spastic dysphonia is suggested by the incidence of upper agree that it is dangerous to define well-circumscribed speech areas in the cortex The process of speech production is a dynamic one, involving countless reverberating circuits These clinical observations on the role of the non-dominant hemisphere in the production of language disturbances correlate well with the lateralization of abnormalities in this study

The literature on the temporal-parietal junction is also meager, being confined to ablative procedures on the dominant side There is no doubt, however, that the angular and supramarginal gyri are essential to the circuitry of speech Lesions of these areas produce the syndromes of word-blindness and word-deafness, and every clinician is familiar with the sensory aphasia resulting from such lesions Irritative cortical disturbances in these areas are infrequently reported in the literature, and with even greater rarity on the minor side[39-5 11 12] No evidence of abnormal phonation due to irritation of the cortical speech areas (with special reference to the temporal-parietal junction) can be found, however, cases of stuttering associated with temporal lobe epilepsy have been reported- in which there were focal findings on electroencephalograms of the right temporal area

In summary, there is no evidence in the literature for a disorder of phonation accompanying a cortical disturbance in the area indicated by our study, although lesions in this area are known to produce aphasia, and the temporal-parietal junction is a recognized "speech area" It is possible that the disturbance in spastic dysphonia is partially subcortical and that the cortex is involved by electrical excitation along corticopetal pathways, however, the laterality of the disturbances and the fast activity suggest primary cortical abnormality It is possible that both cortical and subcortical disturbances exist, the abnormal EEG pattern being the result of more than one lesion

C Discussion of Results of the Present Study

The examiner received the impression of a specific neurologic entity in four of the ten cases We do not attempt to ascribe undue significance to small neurologic signs nor do we wish to weigh the laterality percentages too heavily, yet that this small group of patients manifests so many and such significant signs, leads to the admonition to perform a minute neurologic examination on each patient with spastic dysphonia. The patient with this disease almost assuredly will have neurologic abnormalities.

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C. Discussion of Results of the Present Study

The examiner received the impression of a *specific neurologic entity* in four of the ten cases. We do not attempt to ascribe undue significance to small neurologic signs nor do we wish to weigh the laterality percentages too heavily, pathways for speech production, in some cases perhaps on a post-encephalitic basis

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respiratory infections prior to the onset of voice disturbance Such viral infections have a predilection for subcortical structures, produce EEG findings similar to those in this study, and may antedate the onset of neurologic symptoms by many years, however, the variability of symptoms, signs and course from patient to patient precludes a general statement

The authors do not mean to imply that there are no psychiatric abnormalities in spastic dysphonia, or that psychotherapy does not have a place in treatment (as it has in the treatment of all chronic diseases) , however, we wish to emphasize that each patient with spastic dysphonia should have a neurologic work-up which will more than likely be illuminating, and that spastic dysphonia has a neurologic basis, probably of specific location, but most likely of varied etiology It cannot be stated that the neurologic and electroencephalographic abnormalities are related to the voice disturbance as cause and effect, yet such a strong association suggests that this phonatory disturbance is not a clinical entity, but a *symptom* in a broader syndrome of neurologic disease Just as a fever prompts the physician to search for the underlying disease process, so should the symptoms of spastic dysphonia indicate an examination of the patient for disease of the central nervous system

V SUMMARY AND CONCLUSIONS

1 Ten patients with spastic dysphonia were studied with neurologic examinations and electroencephalograms

2 Every patient had signs of central nervous system disease, with suggestive specific clinical entities in four cases

3 Ninety per cent of electroencephalograms were abnormal, with the disturbance generally being paroxysmal in the right temporal-parietal area

4 The authors consider spastic dysphonia to be a symptom of disease of the central nervous system, both from the standpoint of this study and from the clinical and experimental facts in the literature

5 It is proposed that there is a specific disturbance in the

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CARCINOMA OF THE TONSIL

Surgical Treatment *†‡

GEORGE W ALLEN, M D ,
and
WILLIAM GARTH HEMENWAY, M D ,

Chicago, Ill

INTRODUCTION

Carcinoma of the tonsil has been given relatively little attention in the English-language literature in spite of its being one of the more common malignancies of the head and neck region According to Martin and Sugarbaker[1] it ranks second to carcinoma of the hypopharynx in frequency among carcinomas of the pharynx Although it has a reputation of poor prognosis, several authors-[3,4] have recently reported impressive results of treatment, both by radiation and by surgery The results of treatment for the largest series since 1941 are summarized in Table I The primary type of treatment in all of these cases was some form of radiation External radiation was mainly relied upon, but peroral and interstitial techniques were also used Nineteen cases in the series of Scanlon, et al ,[3] had local excision of the primary in addition to radiation

No comparable five year survival rates for surgical treatment are available This is understandable in view of the earlier discouraging reports on the use of surgery in the treatment of this disease, but several recent reports[2,4,5] of short term results have renewed interest in this method of treatment Klopp and Schurter[2] report nine out of 11 cases (81 per cent) free of recurrent or metastatic disease one year or more after surgical treatment They employ an incision

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*Read at the meeting of the Middle Section American Laryngological Rhinological and Otological Society Inc Chicago Ill Jan 18 1960

†From the Section of Otolaryngology of the University of Chicago

‡This investigation was supported by a special traineeship (BT-356) from the National Institute for Neurological Diseases and Blindness Public Health Service

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which splits the lower lip in the midline. Mandibulotomy is performed, also in the midline, if necessary for exposure. For cases without palpable nodes, an upper neck dissection with excision of the primary is performed *en bloc*. For cases with palpable nodes a complete neck dissection is performed. The reconstruction employs a pedicle flap from the tongue to reconstruct the palate and pharynx. Only one of these cases had palpable cervical metastases originally. They also reported ten cases followed for less than one year with recurrences in only one. Five of these cases had cervical metastases before treatment.

Barbosa[4] reported ten cases treated by similar *en bloc* excision including partial mandibulectomy, but only four had been followed longer than one year. Of these four cases, two were free of recurrent carcinoma.

TABLE I

Results from Other Centers

Author	No. Cases	Overall Results per cent 5 yr Survivals	No Cervical Nodes per cent 5 yr Survivals	Cervical Nodes per cent 5 yr Survivals
Martin and Sugarbaker[1]	148	18%		
Walker and Schulz[2]	90	15	25%	9%
Teloh[6]	162	7.2	20	4
Sheline et al.	82	20	33	9
Scanlon et al.[3]	46	43	57	32

It has been our impression that carcinoma of the tonsil almost invariably recurs within one year if it does so at all. One of our main purposes is to examine this hypothesis. Our case material consists of 64 cases of carcinoma originating in the tonsil. All were treated at least one year ago. Almost half, 27 cases, have been treated within the past five years, and follow up for the early years was not good enough for meaningful five year survival statistics.

CLINICAL DATA

Of the 64 cases, 56 were male and eight female. Ninety

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Stage II—Carcinoma extending into the immediately adjacent tissues—soft palate, tonsil pillars, and tongue No nodes palpable

Stage III—Carcinoma with local extension beyond Stage II or nodes palpable but not fixed

Stage IV—Involvement of skin, fixed nodes, or distant metastases

Unfortunately, many cases were not described adequately enough to distinguish between stages III and IV, so an "unclassifiable" category has been added to the Tables Our classification corresponds to that of the Mayo Clinic group[3]

TABLE III

Classification of Cases According to Stage

	No
Stage I	1
Stage II	16
Stage III	25
Stage IV	11
Unclassifiable	11
Total	64

73% with Cervical Metastasis

for stage I and II Their stage III includes both of our stages III and IV Their 46 cases were broken down as follows stage I, 11 cases, stage II, 10 cases, stage III, 25 cases Our stage distribution is given in Table III

METHODS OF TREATMENT

Radiation A variety of treatment techniques were employed, but external irradiation has been the primary technique Most patients before 1953 were treated at 250 KV Since 1953 most cases have been treated with supervoltage radiation The time-dose relationship was approximately 5500-6500 r in about six weeks In ten cases some form of interstitial irradiation was used as an adjunct to other therapy Eleven cases had some previous treatment elsewhere

Surgery Eight patients had a radical neck dissection to

per cent of those for whom habits are recorded smoked, and 78 per cent drank alcoholic beverages

Sore throat was the earliest symptom in 34 cases The average duration was 4 2 months with a range of one-half to 12 months Presence of a visible or palpable lump in the neck was the earliest symptom in 15 cases Average duration of this symptom was 8 5 months with a range of one to 36 months Three cases were discovered on examination for unrelated problems Four cases could not be distinguished grossly in spite of strong suspicion based on the presence of cervical metastasis In these the primary was demonstrated by multiple small biopsies in one case and by excision of the suspected tonsil in three cases

TABLE II

Age Distribution

Age in Years	Cases
40-50	6
50-60	22
60-70	26
70-80	9
80-90	1
Total	64

The distribution according to age is shown in Table II

The histological type was judged to be squamous cell carcinoma in 60 cases and transitional cell carcinoma in four cases There were no adenocarcinomas Sarcomas and lyphomas were excluded from this series

CLASSIFICATION

A system of stage grouping of these lesions is highly desirable in comparing results of treatment The staging system proposed here is patterned after that described for the larynx by the International Committee for Stage-grouping in Cancer and for the Presentation of the Results of Treatment of Cancer[9] in 1953

Stage I—Carcinoma localized to the tonsil

Stage II —Carcinoma extending into the immediately adjacent tissues—soft palate, tonsil pillars, and tongue No nodes palpable

Stage III —Carcinoma with local extension beyond Stage II or nodes palpable but not fixed

Stage IV —Involvement of skin, fixed nodes, or distant metastases

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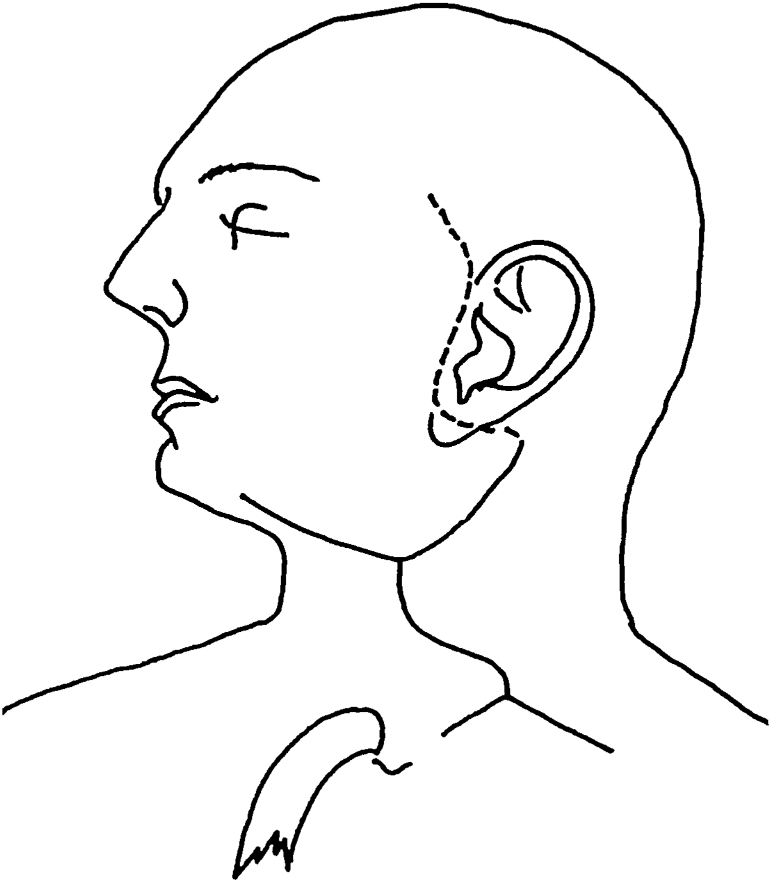

Fig 1

supplement external irradiation Five patients had local excision of the primary lesion In four cases this was supplemented by external irradiation

Twelve cases were treated by *en bloc* excision of the primary, plus neck dissection A basic technique has been evolved which can be varied to meet the requirements of the situation This is outlined in the following paragraphs

A preliminary tracheotomy is usually performed A modified double-Y incision is then made This features a relatively large upper flap which extends below the hyoid bone and will completely cover the pharyngeal suture line The middle branch is undulated, so that the incisions all meet at equal angles and the carotid vessels are crossed at right angles (see Fig 1) We believe this incision to have several important advantages 1 Sharp corners are avoided, 2 the size of the flaps is balanced, 3 healing does not produce "checkrein" scars, and 4 it may be continued upward in front of the ear in order to provide maximum exposure of the infratemporal fossa The platysma is usually included in the skin flaps The neck dissection is performed from below and behind in standard fashion, to remove the sternocleidomastoid muscle, the internal jugular vein and all of the contents of the posterior cervical triangle and the infrahyoid portion of the anterior triangle The anterior belly of the digastric is then divided at its insertion, Wharton's duct is divided, the external maxillary vessels are ligated and divided, and the fascia colli is incised along the lower border of the mandible The trunk of the facial nerve is dissected out The mastoid insertion of the sternomastoid muscle, upper end of the internal jugular vein, posterior belly of the digastric and stylohyoid muscles, and the tail of the parotid gland are all dissected out and divided The hypoglossal nerve is then dissected out well forward into the tongue and retracted downward with tape The external carotid artery is ligated and divided and the internal carotid and vagus nerve retracted posteriorly with tape The dissection is now varied in one of three ways depending on the location of the primary and its size

First, if the primary is small and does not invade the soft palate, the pharynx is entered at the base of the tongue and the incision continued upward behind the posterior pillar of the tonsil If the growth is invading the base of the tongue, the tongue is incised at a distance of 2 5 cm around the mass In any case the incision is made in front of the anterior pillar, and in joining the posterior incision some tongue is removed The internal pterygoid muscle is stripped from the mandible This and all the previous dissection is done from the outside The final division of the soft palate and internal pterygoid muscle is then performed through the open mouth

Second, for primary lesions invading the soft palate but not fixed and well removed from the mandible, the mandible is divided in a fish-tail cut with the oscillating saw just in front of the masseter muscle The ramus of the mandible is then retracted laterally by stripping away the insertion of the internal pterygoid muscle Excellent exposure can be obtained in this way with several advantages over the lip splitting incisions *1* Less mandible is exposed to possible infection, *2* collateral circulation from the other side to the

TABLE IV

Overall Results of Radiation Therapy

	Total No	Failures No	Failures Per cent
All cases treated by radiation	54	48	89
Radiation 5000 r or more	35	29	83

TABLE V

Results of Treatment with Radiation Alone

Stage	Total No	Failures Within 0-1 yr	Failures Within 1-2 yrs	Failures Lost to Follow-up	Living Without Recurrence 2-3 yr	Living Without Recurrence 3-4 yr	Living Without Recurrence 4-5 yrs	Living Without Recurrence 5 yrs
I	1	0	0		1			
II	11	3	1	3	1			3
III	9	6	0	2		1		
IV	8	6	0	2				
Unclassified	8	8	0					
Total	37	23	1	7	2	1		3

84% Failures

upper skin flap is not disturbed, *3* if removal of the ramus of the mandible appears necessary as the dissection proceeds, then as much of the body of the mandible as possible can be conserved with good blood supply, much more than if the mandible had already been divided near the midline

Third, for very large lesions the incision can be extended in front of the ear, the parotid gland mobilized, and working first above and then below the gland, the ramus of the mandible, the zygomatic arch and the entire contents of the infratemporal fossa can be removed in the manner described by Conley[10] With either of the two more extensive operations the pterygoid plates are usually removed, and if necessary the maxillary tuberosity and part of the hard palate may be chiseled off In the first and third types of operation a linear closure of the mucosa is possible In the second type a pedicle flap of tongue is cut and rotated, to be sutured into the remaining soft palate This is made large enough to provide good coverage for the mandible, which is then reunited by criss-cross wiring The mucosa is closed with a continuous inverting suture of 000 chromic catgut reinforced by a layer of interrupted sutures of 000 chromic catgut in the muscle and fascial layers The skin is closed after insertion of drains A tracheotomy tube is inserted and a pressure dressing applied

TABLE VI

Results of Irradiation to Primary Plus Neck Dissection

Stage	Total No	Failures Within 1 yr	Lost to Follow-up
II	1	1	
III	3	3	
IV	2	2	
Unclassified	2	7	1
Total	8	7	1

100% Failures

TABLE VII

Results of Irradiation Plus Local Excision

Stage	Total No	Failure	
		0 - 1 yr	1 - 2 yr
II	1		1
III	1	1	
Unclassified	2	2	
Total	4	3	1

100% Failures

RESULTS

The results of treatment for all cases in which radiation was the primary method of therapy are given in Tables IV, V, VI and VII. This includes all cases treated at the University of Chicago Clinics, regardless of whether or not therapy was initiated elsewhere. Failures are defined as *1* Cases showing no response to treatment, or *2* cases showing resumption of growth after an initial response, or *3* cases dying with carcinoma present, or *4* cases lost to follow up. Most cases showed at least some favorable response to X-ray, but the exact number is impossible to determine. No cases lost to follow up were free of carcinoma when last seen.

TABLE VIII

Results of En Bloc Resection

Stage	Total No	Failure Within 1 yr	Living Without Recurrence 1 - 2 yr	Living Without Recurrence 2 - 3 yr
II	1	1		
III	10	5	4*	1
Total	11	6	4	1

55% Failures
Five Cases were Recurrences Following Radiation,
Two Living Without Recurrence

---

*One case had prophylactic radiation following surgery

It is obvious from Table IV that even for the group treated with more than 5000 r, the most that the five year survival rate could be is 17 per cent.

It can be seen from Tables V, VI and VII that all recurrences, except two, that have appeared to date, have been within the first year. All cases of recurrence following surgery have been within one year. This suggests that the number of cases surviving for one year without recurrence may furnish a good standard for comparison of results of treatment by radiation and by surgery. It should be noted that only one case in which there were abnormal cervical lymph nodes responded to radiation. Radical neck dissection and local excision used as adjuncts to radiation were completely ineffective. Interstitial radiation controlled a recurrence following external radiation in one case, and this case is listed on Table VI only as a survival without recurrence.

In the nine other cases interstitial radiation was not successful in controlling recurrences

Surgery We have not yet treated any early lesions primarily by surgery The one stage II case had very extensive local spread Table VIII shows the results of surgery. Two patients died during the early postoperative period, one of hepatic failure at three months and the other of an acute, diffuse hemorrhagic gastritis at one week

One case had local excision only and died within one year Three cases were not treated, being seen for opinion only

Complications of Surgery Two patients developed large fistulae One occurred in a patient who had surgery for a recurrence following radiation therapy This fistula was closed successfully in two stages by a lined pedicle flap Swallowing and speech remain unsatisfactory in this case Swallowing and speech are practically normal in the other cases

DISCUSSION OF RESULTS

The tendency for early recurrence following either radiation or surgery seems clear Of all cases seen to recur, only two have done so one year or more after treatment This makes the early results reported by Klopp and Schurter[2] look promising Although we have not had experience with these early lesions, our experience in cases with cervical node involvement leads us to advise surgery as the primary method of treatment in these cases When cervical metastasis is present initially, the outlook with treatment by radiation is poor Out of 40 of our cases with cervical metastasis treated by radiation, only one remains free of carcinoma This was not a proved metastasis, but was described as a hard node one centimeter in diameter by a reliable observer

Since the primary lesion often responds well to radiation, our original approach was to radiate the primary and nodes and to follow this by neck dissection when necessary All eight of our cases treated in this way were failures It is now our policy, when the patient has already received radiation, to remove the region of the original primary together with the neck dissection *en bloc*, although there may be no gross tumor in some parts of the primary region

Our early success and that of others[2,4] with this more radical surgery has been encouraging Five cases out of 11, 45 per cent, have survived longer than one year without recurrence All were advanced cases and required extensive resections, but there is little residual disability except in one case The outlook for functional rehabilitation is better than that of cases treated by surgery for carcinoma of the larynx or tongue

In spite of the accessibility of the tonsils to examination, there seems to be little hope for diagnosing more of these lesions before metastasis has occurred, because of the tendency to early metastasis and the relatively prolonged asymptomatic period Many of these lesions are of small size and are very inconspicuous at a time when the cervical lymph nodes are quite obviously the site of metastatic disease As in the nasopharynx early metastasis is probably a result of the intimate contact between the epithelium of origin and the lymphoid tissue Careful examination will reveal the primary in many of these cases and should always include retraction of the pillars and palpation In the presence of nodes without an obvious primary the tonsil should be removed and serially sectioned for microscopic examination

SUMMARY

Techniques are described for the surgical treatment of carcinoma of the tonsil Our case material shows these neoplasms to have a very strong tendency to recur within one year, if they do so at all For this reason we feel justified in comparing the results of radiation and surgery for cases followed for one year or longer For 40 cases with enlarged cervical nodes treated by radiation there was only a 2 5 per cent rate for survival without recurrence For 11 similar cases treated by definitive surgery, 45 per cent are alive and free of carcinoma Because of this experience, we now advise surgery as the primary method of treatment in all cases of carcinoma of the tonsil with involvement of the cervical nodes

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SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY OF SOUTHERN MEDICAL ASSOCIATION

The Section of Ophthalmology and Otolaryngology of the Southern Medical Association announces that they are now accepting papers by physicians of either specialty living in the area of the Southern Medical Association for consideration for presentation at the next annual meeting to be held in St Louis, Mo, from October 31 to November 3, 1960

The paper or an abstract of the paper may be sent directly to the Secretary, Dr Albert C Esposito, Suite 1212, First Huntington National Bank Building, Huntington, W Va, as soon as possible

THE USE OF THE RHINOTOMY OPERATION IN NASAL SURGERY [†]

JOHN E BORDLEY, M D ,
and
JERRY CHERRY, M D ,

Baltimore, Md

Since September, 1948, some 40 rhinotomy procedures have been carried out at Johns Hopkins Hospital on patients of the Otolaryngological Service This particular technique, which was first described in 1949,[1,2] is somewhat similar to that used by Bruns in the latter part of the last Century[3,4] It was designed to afford better access for the surgical removal of extensive lesions occurring within the nasal and paranasal spaces, while preserving to a great extent normal nasal physiology

Inadequate exposure has long been recognized as a limiting factor in nasal surgery where extensive lesions were encountered Each year in every large clinic three or four cases with extensive intranasal and paranasal lesions are seen, which are difficult to treat adequately by the usual intranasal techniques

In the procedure, adopted by our clinic, the nasal pyramid is swung to one side using the soft tissues of the naso-facial angle as a hinge (see Fig 1) This is accomplished by means of a bilateral osteotomy and an incision crossing the bridge of the nose in the region of the naso-frontal suture extending down one naso-facial angle and around the ala nasi A second incision is made across the base of the columella The septum is then divided with scissors on a same plane as the lateral incision of the nose (see Fig 2) After the nose is swung aside a submucous resection is carried out on the intranasal septal

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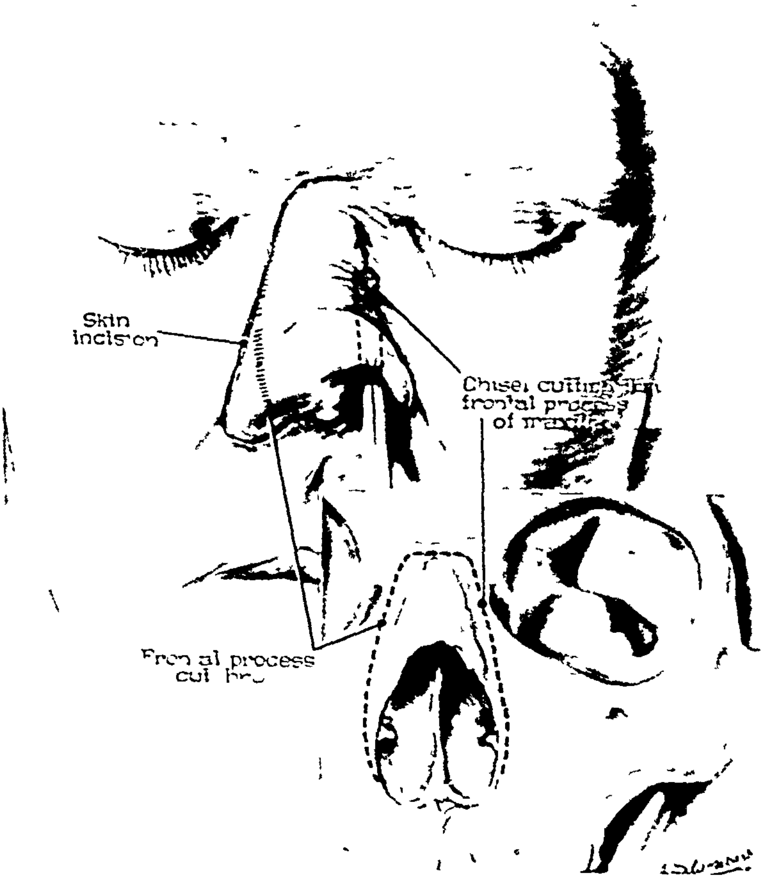

Fig 1

cartilage and perpendicular plate of the ethmoid bone, care being taken to leave a small anterior column of septal cartilage for later anterior support of the nose With the nasal pyramid removed, one has a much more direct approach to the nasal sinuses By retracting the soft tissues at the nasal-facial angle, laterally, the anterior wall of the maxillary sinus can be exposed By freeing up the periosteum of the orbit the lamina papyracea can be visualized as well as the more medial ethmoid cells in the nose itself (see Fig 3) By extending the transverse portion of the incision across the bridge of the nose up over the brows, both frontal sinuses can be easily ap-

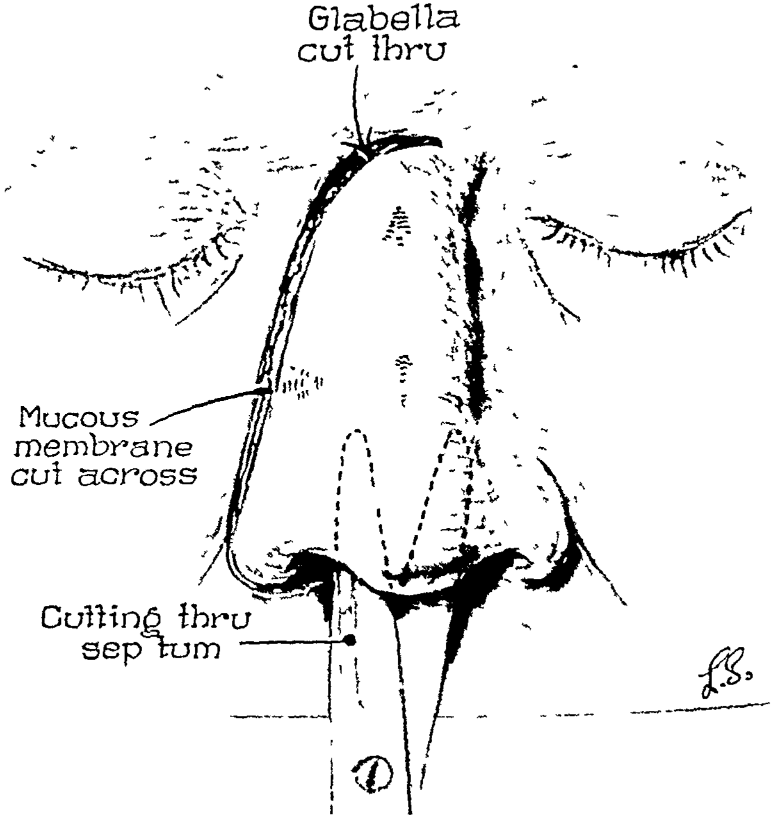

Fig 2

proached The distance is shortened to the anterior wall of the sphenoid sinus and with an easily movable septum an excellent exposure of the sphenoid is obtained

The closure after this approach is quite simple The most important step is to resuture and realign the intranasal and pyramidal portion of the septum This is done by placing interrupted sutures in both portions of the septum before the pyramid is swung back in place The nasal incisions can be closed in two layers, as can the secondary incision over the frontal sinuses In all of our cases, where removal of the septum or a portion of the septum was not required, we have had complete restoration of the septum with no postoperative perforations This is very important in those cases where a arge portion of the lateral wall of the nose has had to be emoved, as it considerably cuts down on subsequent crusting nd discharge

Care should be taken to keep any pressure of instruments r drapes away from the hinged pyramid during the operative rocedure and the pyramid should be kept wrapped in gauze

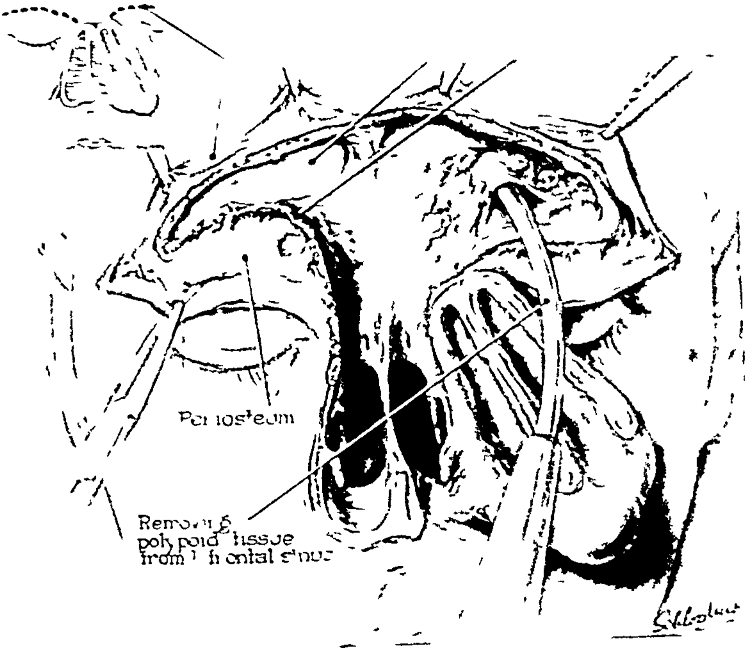

Fig 3

soaked in warm Ringer's solution or normal saline until it is replaced in its normal position

Forty patients have undergone this procedure since 1947 For brevity of presentation they have been grouped into three classifications *1* those patients suffering from long standing chronic sinusitis (see Fig 4) who have undergone numerous intranasal surgical procedures with resultant loss of intranasal land marks, severe scarring and degenerative changes in the membranes of the nose Some of these patients have developed local osteomyelitis as a result of long standing infection, *2* the second classification covers those patients suffering from benign cysts or benign growths (see Fig 5) within the nasal spaces or paranasal spaces, *3* the third classification consists of those individuals suffering from malignant disease of the nose and paranasal sinuses (see Fig 6)

INFECTION

		Number
A	Chronic sinusitis with obstructive polyps and scarring following repeated intranasal surgery	7
B	Osteomyelitis associated with chronic sinusitis	4
	Total	11

Fig 4

BENIGN CYSTS AND TUMORS

		Number
A	Chromophobe Adenoma of pituitary gland	3
B	Retention Cysts	2
C	Encephalocele	3
D	Osteofibroma	1
E	Angiofibroma	1
F	Hemangioma	1
G	Embryonal Angioma	1
	Total	12

Fig 5

It would be well to point out several particularly interesting cases in which we have found this type of surgery to be extremely helpful

In two patients suffering from chromophobe adenomas of the pituitary gland, this approach was used to reach the tumor from below One of these patients had previously had two intracranial operations and had lost the sight of one eye This patient subsequently had to have a second operation on the adenoma and this was done through a secondary rhinotomy approach He has remained well for the past six years After the two operations his septum is still intact

Recently a chondrosarcoma involving the ethmoids, the sphenoid and the lateral nasal wall was operated upon. It was possible to do a block resection which included the orbital floor from just lateral to the infra-orbital foramen, the lateral nasal wall, the ethmoid complex including the lamina papyracea, a portion of the cribriform plate and the anterior wall of the sphenoid. The tumor was so extensive that one would hardly believe that a cure was accomplished, however, the patient has been symptom free for the past six months.

Another case was that of a hemangioma in a boy 14 years of age involving the ethmoid complex, the middle turbinate and the medial and superior wall of the orbit. This tumor was so extensive and so close to the eye that irradiation was contraindicated. It was removed through a rhinotomy some five years ago, and to date there has been no recurrence. The patient's visual apparatus is normal and there is very little external deformity.

MALIGNANT TUMORS OF THE NOSE AND PARANASAL SINUSES

	Number
A Adeno Carcinoma	4
B Squamous Cell Carcinoma	2
C Basal Cell Carcinoma	1
D Papilloma (Low grade malignancies?)	4
E Plasmocytoma	1
F Lethal Midline Granuloma	4
G Chondrosarcoma	1
Total	17

Fig. 6

Of particular interest were three cases requiring rhinotomy suffering from encephaloceles. An encephalocele consists of brain tissue which has grown through some defect in the calvarium and extends into the neighboring structures. As the brain grows, the brain tissue in the encephalocele grows, displacing and destroying adjacent tissues just as a benign tumor would do elsewhere.[5,6,7,8,9,10,11,12,13,14,15]

There are some 50 nasal encephaloceles reported in the literature since 1900. Statistics show that the instance of encephaloceles are about one in every 4,000 births. They seem to be non-hereditary, non-familial and not associated with other congenital defects About 15 per cent of encephaloceles described are anterior and 85 per cent are occipital in presentation The surgical approaches that have been listed are *a* intranasal, *b* external ethmoid, and *c* craniotomy Prior to the advent of the antibiotics, the death rate following attempts to remove these tumors was extremely high

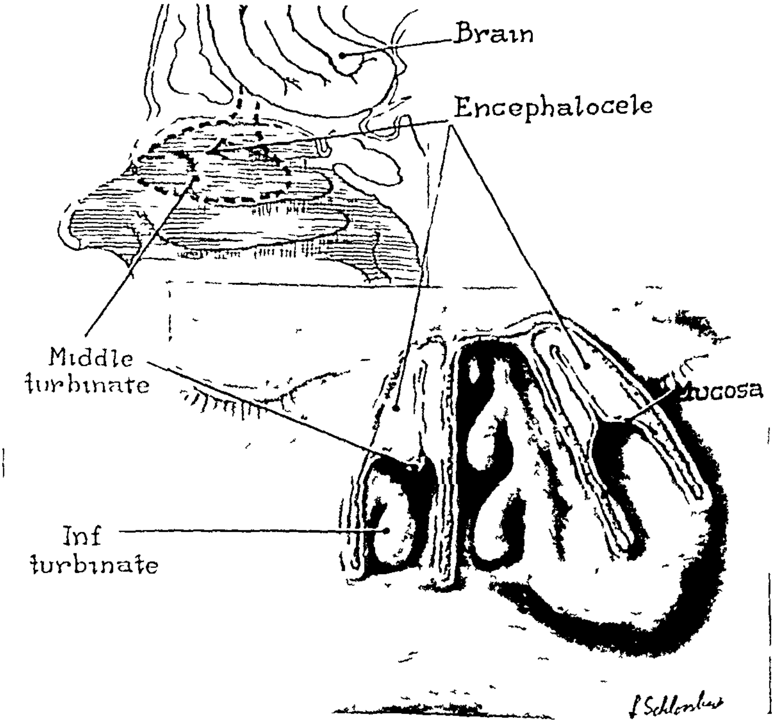

Fig 7

To cure such growths, the classic procedure has been to sever the stalk of the tumor at the surface of the brain by an intracranial approach, then to close the defect in the skull Following such intracranial surgery, the mass will shrink down and in some instances apparently disappear Under certain circustances, however, the extra cranial brain tissue will parasite a blood supply and begin to grow again Such growths can become locally quite destructive This is particularly true in individuals who have not reached maturation, and where growth is still taking place in the brain itself The excellent blood supply to the paranasal area offers an optimum opportunity for such growth In cases where continued growth is probable or in situations where the mass of the encephalocele intereferes with normal physiological function, removal of the extra cranial growth is indicated

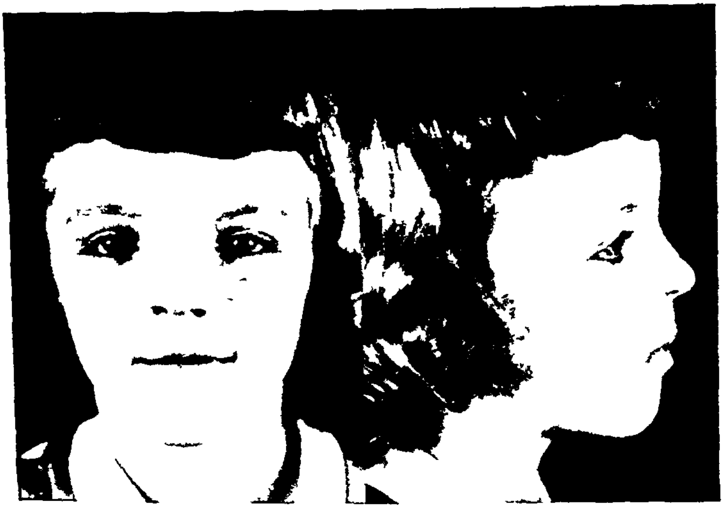

Fig 8

In the face, the most common pathways of extension of encephaloceles are *1* the naso-frontal, in which the growth extends between the nasal and frontal bones destroying a portion of the nasal bones and producing a swelling at the nasal root, *2* the naso-orbital, where the brain tissue pushes through the lacrimal-frontal suture line and produces a swelling near the inner canthus of the eye, *3* the naso-ethmoidal, where the defect lies between the nasal, frontal and ethmoidal bones This permits growth downward into the nose, which involves the turbinates and the septum, as well as the nasal vault, *4* extension downward through a defect in the cribriform plate (see Fig 7) As all of these growths enter the

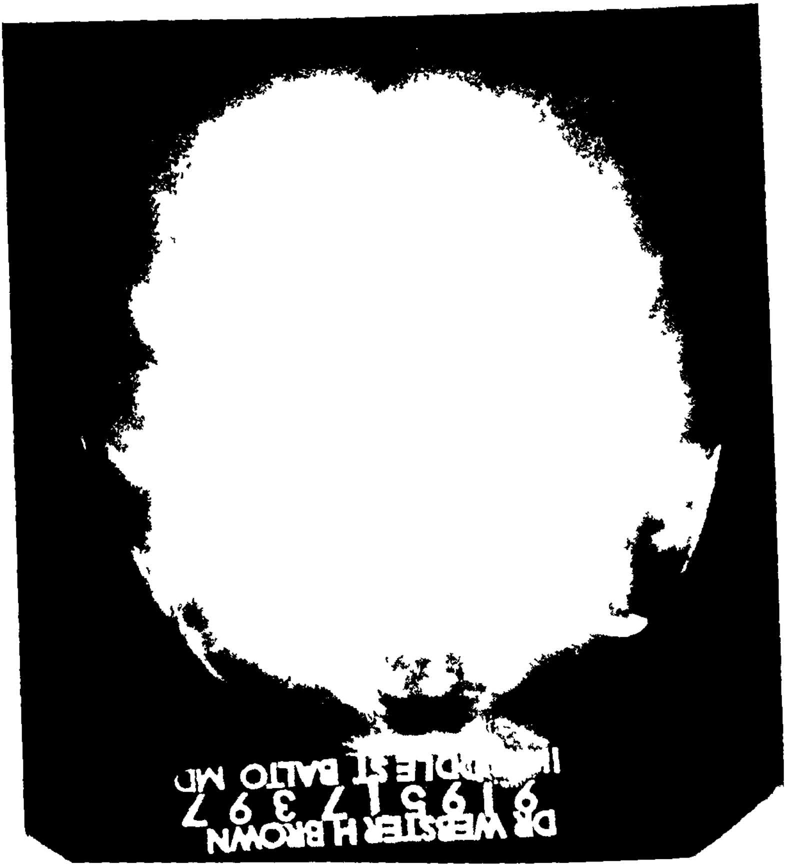

Fig 9

sinuses and nose from outside of the nasal spaces, they present in the nasal cavity as submucosal swelling

In order to remove those encephaloceles that interfere with normal nasal function, it is necessary to get a good exposure of the growth Such exposure may be obtained by using the rhinotomy approach The three cases presented here illustrate very well some of the characteristics of these growths

Case 1 W G, age three months white female (see Fig 8—Photographs eight years postoperative)

Shortly after birth a swelling was seen about one half a centimeter in diameter just to the right of the midline of the naso frontal region The mass felt slightly cystic Examination at the age of six months showed an external mass with a diameter of about one centimeter involving the right nasal bone, just below the level of the inner canthus of the eye The right nostril was seen to be filled with a mass covered by normal looking mucous membranes Breathing space was obliterated The mass was not fluctuant X rays showed a defect in the right naso frontal area (see Fig 9) Biopsy showed brain tissue covered by respiratory mucous membrane (see Fig 10)

At the age of six months Dr Earl Walker of the Neurosurgical Service using a frontal approach cut the stalk of the growth and closed the defect in the skull with a bone implant and a fascial graft At nine months of age, the nasal swelling was slightly reduced but still obstructive and a rhinotomy was performed A relatively vascular mass was exposed filling the anterior portion of the right nostril extending down over the upper portion of the right side of the septum It is found to have extended externally through a defect in the upper portion of the right nasal bone This growth was entirely submucosal Nine years later this child showed no evidence of recurrence her breathing space is excellent, and her facial growth is normal

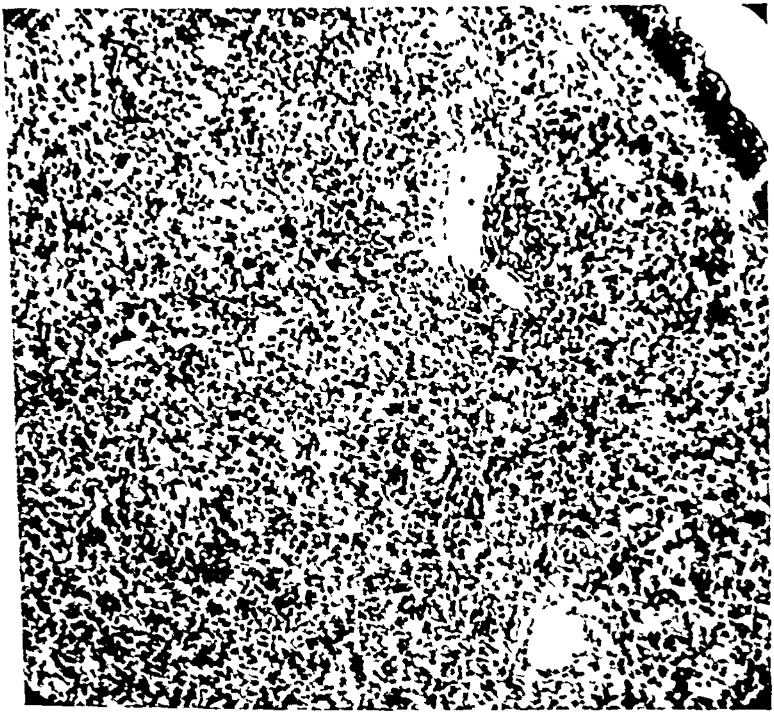

Fig 10

Case 2 J S age three-and a half months white male (see Fig 11—Photographs five years postoperative)

This patient was admitted because of an obstructive mass in the right nostril which was discovered shortly after birth He had had two previous nasal operations before entering the Clinic These operations were for the removal of nasal polyps Sections made of material removed at the second operation showed brain tissue covered by respiratory epithelium Examination disclosed an external fullness of the right side of the nose The right nostril was filled with an edematous mucous membrane covered mass The septum was pushed over to the left side At four months of age a frontal lobe exploration was carried out by the Neurosurgical Service The stalk of the tumor was found to extend through a defect about 4 mm in diameter in the right cribriform plate This was severed, the defect was closed with a bone implant and covered with dura from the crista galli The mass decreased rapidly in size, the breathing space improved, and the nose became symmetrical However at the age of four years the mass began to increase again and once more filled the right nostril Through a rhinotomy approach the mass was found to extend over the vault of the nose and to involve the upper half of the nasal septum The mass extended inferiorly on the lateral wall to the level below the middle turbinate Superiorly, it extended to the cribriform plate The right leaf of the septum was removed with the specimen Postoperatively the patient has continued to do well Three years after operation the breathing space is good There is no nasal crusting, and the septum is intact

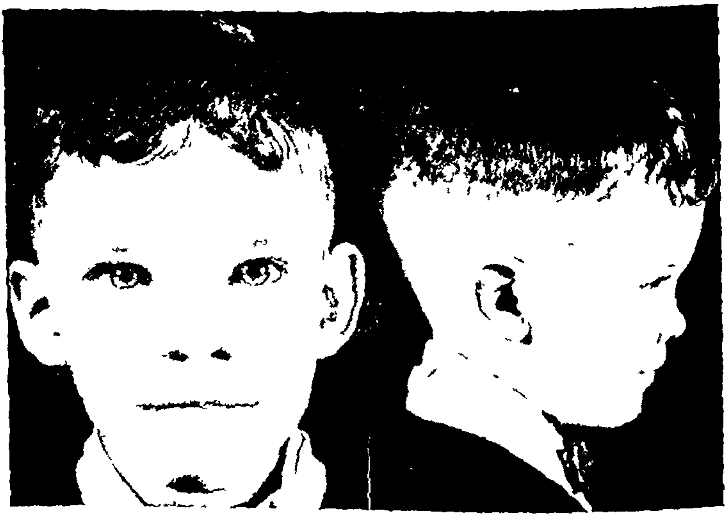

Fig 11

Case 3 E S, age 12 years, colored female

This patient was admitted complaining of bleeding from the left nostril Examination showed a mass involving the vault of the left nostril the left upper lateral cartilage and the septum The biopsy report was "necrotic tissue and inflammation, possible lethal midline granuloma' A rhinotomy revealed a growth involving the whole upper left nostril with destruction of the upper septal cartilage and a portion of the ethmoid plate Frozen sections showed brain tissue covered with respiratory epithelium A stalk was exposed which extended through a defect in the left cribriform plate This was removed from below, and the defect was closed from below with a flap of mucous membrane and a gelfoam pack The neurosurgeons decided not to do an intracranial exploration as the brain had reached its full growth The patient has been symptom free for the past three years Breathing space is satisfactory and the nasal septum is intact

As to cosmetic results, they are acceptable Of course, those individuals losing the anterior wall of their frontal sinuses will suffer at least temporary deformity of their brows It is possible, if the patient desires it, to later rebuild the brow with bone chips If care is taken to follow the facial lines carefully with the original incision, satisfactory cosmetic results should be achieved

CONCLUSIONS

Some 40 rhinotomies have been performed at Johns Hopkins since 1947 Each patient has been carefully selected for this procedure Lesions that do not involve the lateral portions of the maxilla must be chosen, as this surgical approach is not satisfactory in such instances All the lesions presently reported would have been extremely difficult to expose and remove by other less deforming approaches The operation has the advantage of maintaining the septum which helps in restoring a relatively normal nasal physiology, postoperatively The procedure is time consuming, but has shown little tendency toward shocking the patient The rhinotomy has a place in modern nasal surgery but should be limited to specially selected cases

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AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY HOME STUDY COURSES

The 1960-1961 Home Study Courses in the basic sciences related to opthalmology and otolaryngology, which are offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on September 1 and continue for a period of 10 months Detailed information and application forms can be secured from Dr William L Benedict, the executive secretary-treasurer of the Academy, 15 Second street S W , Rochester, Minn Registrations should be completed before August 15

CLOSURE OF PERFORATIONS OF THE TYMPANIC MEMBRANE BY VEIN GRAFTS

A Preliminary Report of Twenty Cases *†

HAROLD G TABB, M D ,

New Orleans, La

Early in 1959, after watching Dr John J Shea, Jr , of Memphis, perform his ingenious fenestra ovalis operation, I became interested in his use of vein grafts, which, he informed me, he had also used to repair lacerations of the eardrum, with good results It later occurred to me that these grafts would be useful for the closure of large perforations of the tympanic membrane, particularly those of long standing (see Fig 1)

By this time I, like many others, had become dissatisfied with the use of skin for this purpose, as skin grafts were hard to manage, and they were not usually an acceptable substitute for normal tympanic membrane They often remained thick and moist, and normal processes of desquamation continued They showed little resistance to infection, which was often followed by perforation or sloughing of the graft There was also the constant danger of cholesteatoma formation on the under surface of the graft, from the downgrowth of squamous epithelium through one of its sectioned appendages Dr Harold F Schuknecht demonstrated such a case to me in August, 1959

With this climate for reform, I decided to test vein grafts in the repair of perforations of the tympanic membrane The results were so gratifying that a preliminary report of our cases, which now number 20, seemed justified

It was not until after the report was completed that I

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*Read at the meeting of the Southern Section American Laryngological Rhinological and Otological Society Inc New Orleans La Jan 16 1960

†From the Department of Otolaryngology Tulane University of Louisiana School of Medicine

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learned that Dr Shea had been working on closure of perforations of the tympanic membrane, in addition to repair of tympanic lacerations, and that he had reported a method of closure which had been successful in 13 of 14 cases[1]

Although we had worked entirely independently, his technique is similar to one of the techniques which I have been using

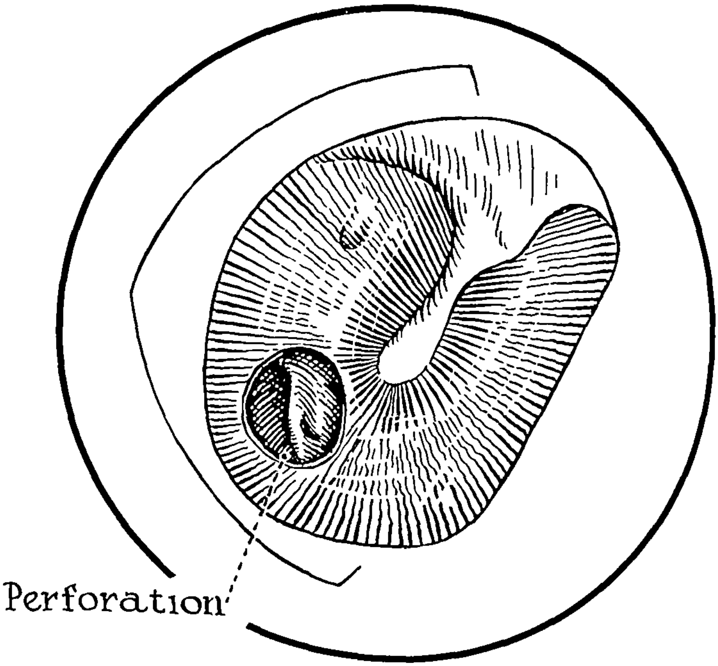


Fig 1 Typical long-standing perforation of posteroinferior quadrant of tympanic membrane

To date, 20 operations by this technique have been performed on our service, with a follow-up varying from two to ten months I am presenting five of these cases in detail (see Fig 2) because they represent the simple, persistent type of tympanic perforation, without associated middle ear or mastoid disease, which responded to this technique after they had resisted other techniques of closure In five of the other 15 cases the perforations were closed at the time of mastoid surgery

Preoperative examination in each of these five cases ruled out any underlying infection and demonstrated the patency of the Eustachian tube on the affected side After an initial audiogram, a paper patch was applied to the perforation and allowed to remain *in situ* for a week or more, to ensure that the ear was dry In each case the ear was dry, and in each case there was prompt improvement in hearing This preliminary use of the paper patch as a sort of therapeutic test was suggested by Dr Robert Henner, of Chicago

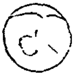
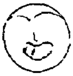
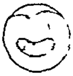
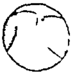
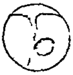
Patient	Age	Perforation Data Ear Type		Method used Previously	No Days Healing (Inflation Pneumomassage)	Vein Donor Site	Post Op Observation Period without Sequela
			Duration				
TH	8		AD Central 9 mos	Acid cautery patch	14	Foot	10 mos
DM	7		AS Central 7 mos		13	Arm	9 mos
DWM	37		AS Central 3 yrs	Acid cautery patch Irritant patch Myringoplasty	18	Hand	6 mos
LL	8		AD Marginal 7 yrs	Acid cautery patch	12	Arm	4 mos
CB	30		AS Central 5 yrs		14	Hand	2 mos

Fig 2 Summarized case histories of five closures of perforations of the tympanic membrane by vein grafts

CASE REPORTS

Case 1 T H an 8 year-old boy, had a 4-mm central perforation of the right tympanic membrane which had persisted after a suppurative otitis media nine months earlier Repeated attempts at closure by the trichloracetic acid cautery paper patch technique had failed

There was no underlying middle ear or mastoid disease and the right Eustachian tube was inflatable A 15 db hearing loss in the speech frequencies was eliminated when a paper patch was applied

A vein graft was placed under the perforation, with the endothelial surface facing the tympanic cavity, through a stapes incision Four days later a Staphylococcus aureus infection developed It was controlled with specific antibiotic therapy without evident damage to the graft On the seventh postoperative day numerous blood vessels could be seen crossing the tympanic membrane just under the epithelial surface and

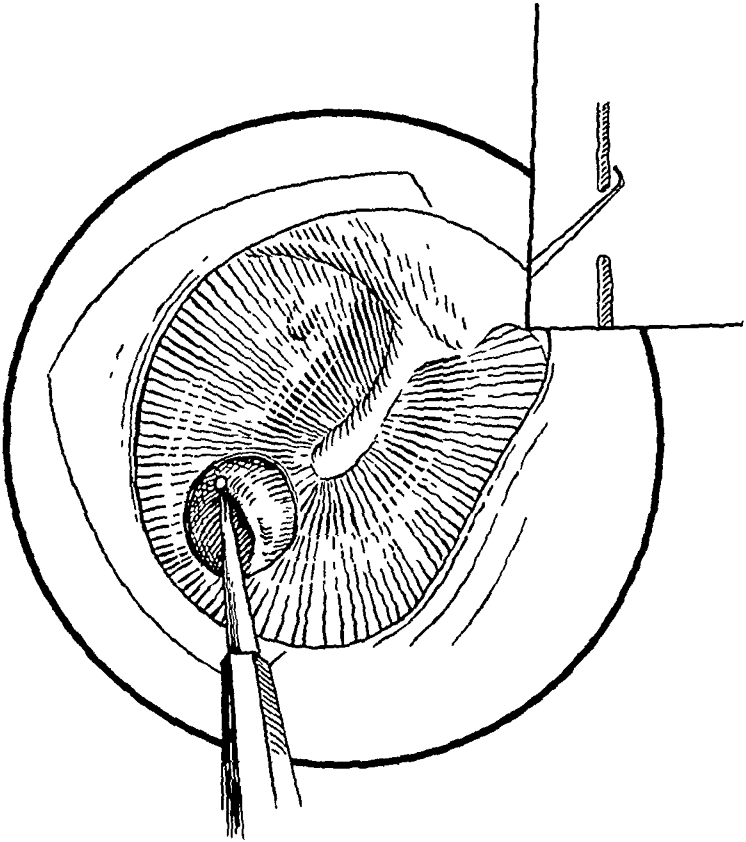


Fig 3 De-epithelization of under surface of *eardrum by right-angle* knife Insert shows detail of technique

apparently supplying the graft On the fourteenth postoperative day, the perforation appeared to be firmly closed The drum was then massaged and the tympanic cavity inflated

At the present time, 10 months after operation, the perforation has remained firmly closed, and the eardrum appears practically normal except for a whitish plaque at the site of the former opening For three months during the Summer this ear was exposed to the hazards of swimming and suffered no ill effects although it was not protected in any way

Case 2 D M , a seven year old boy, had a central perforation, 3 4 mm in diameter, of the left tympanic membrane, it had developed seven months earlier, after an upper respiratory infection and purulent otitis media There was no underlying infection and the ear remained dry for a month, during which time a 10 db hearing loss was eliminated

Thirteen days after closure of the perforation by a vein graft placed through a stapes incision, healing was sufficiently firm to permit massage of the drum and inflation of the middle ear

Swimming was allowed a month after operation A month later, the patient appeared with an acute external otitis, from a *Proteus* infection which was controlled with specific antibiotic therapy The repaired tympanic membrane was as resistant to spread of the infection as if it had been a normal structure At the end of nine months the results are entirely satisfactory

Case 3 D M, a 37-year old man was first seen in July 1958 with drainage from the left ear of three weeks' duration A perforation of the tympanic membrane had been successfully repaired a year earlier by myringoplasty and the patient had had no difficulty until the present episode of otitis media When the infection was controlled a large kidney shaped central perforation was left The Eustachian tube was patent The patient had a 10 to 15 db hearing loss

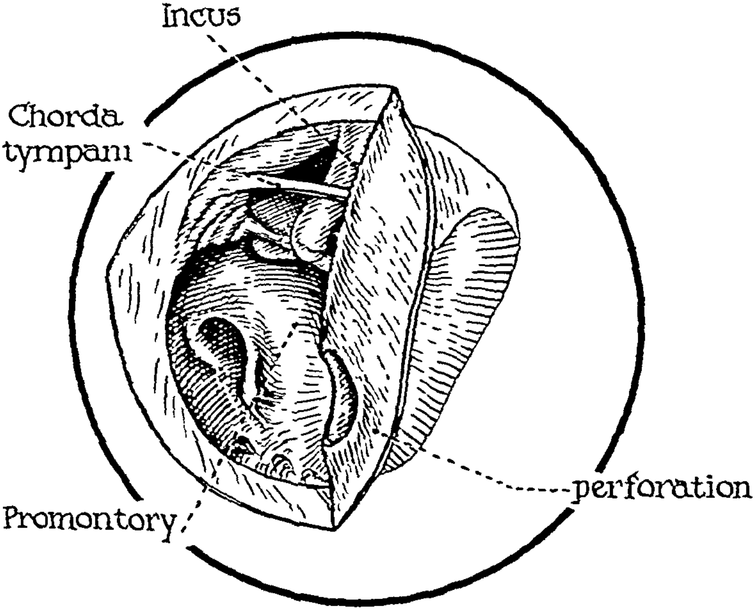


Fig 4 Reflection of tympanic membrane after stapes incision to expose perforation

After the ear had been dry for a month, closure was accomplished by office treatment with the acid cautery and irritant drops At the end of five months the drum appeared normal and hearing was improved

Three months later the patient returned with acute suppurative otitis media After it was controlled, there was again a large central perforation which this time did not respond to office methods of closure In June, 1959 closure was effected by the use of a vein graft applied through a stapes incision Healing was complete in 18 days and the patient has had no hearing or other difficulties in the six months since operation

Case 4 L L an 8 year-old girl had had a marginal perforation of the right tympanic membrane after a purulent otitis media when she was a year old Drainage developed whenever she went in swimming An adenotonsillectomy two years before had had no effect on the perforation

and an attempt to close it with the acid cautery and a paper patch 18 months later also had failed. There was no underlying middle ear or mastoid disease and the Eustachian tube was patent, but there was a mild conductive hearing loss.

Under general anesthesia, a stapes incision was made, to permit examination of the middle ear structures, and a vein graft was placed under the perforation and held in place by Gelfoam®. Healing was firm 12 days after operation and at the end of four months the results, including improvement in hearing, are still good.

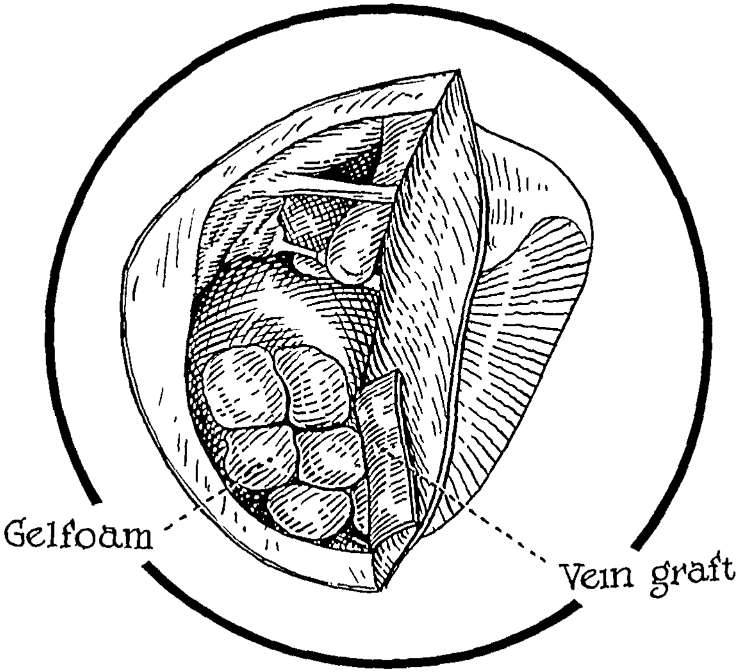


FIG. 5. Vein graft applied to under surface of tympanic membrane and held in place by Gelfoam®.

Case 5. C. B., a 30-year-old man, had had a large central perforation of the left tympanic membrane for the past five years. Suppurative otitis media developed whenever he had an upper respiratory infection. There was a conductive loss of 25 db in this ear. The Eustachian tube was patent and the ear was dry. The use of a paper patch eliminated the hearing loss.

A vein graft was placed through the central perforation and positioned satisfactorily without the use of a stapes incision. The perforation healed in 14 days, and at the end of two months the results, including the restoration of hearing to normal, are entirely satisfactory.

TECHNIQUE

In the first cases in this series it was our practice to em-

ploy the so-called stapes incision (the incision used in mobilization of the stapes), adapted from Lempert,[2] and the vein graft was placed on the inferior surface of the drum through it. Later, it was realized that if the perforation were sufficiently large for the necessary manipulations, the incision was not necessary. Thereafter, it was omitted unless it was thought that exploration of the middle ear or some procedure on the middle ear would be necessary.

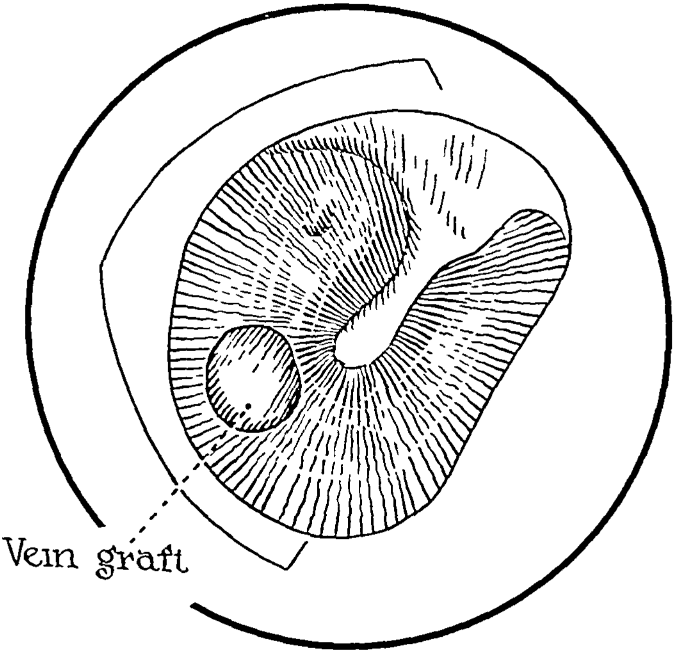


Fig. 6. Vein graft in situ after replacement of reflected tympanic membrane.

Whether the incision is or is not used, the initial steps of the procedure are the same.

1. The auricle and external auditory meatus are very carefully cleansed under microscopic observation.

2. Basal sedation is used before operation. The procedure is carried out under local infiltration anesthesia in adults and under general anesthesia in children.

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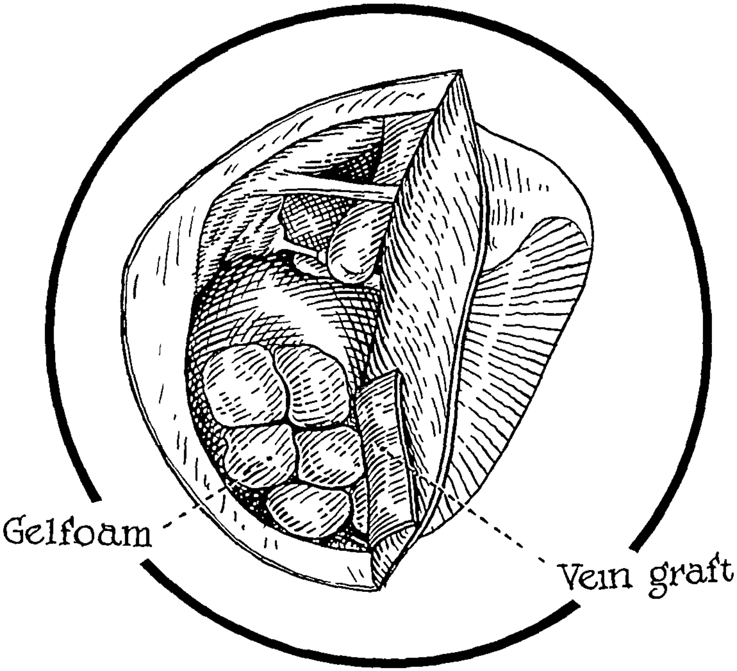

Fig 5 Vein graft applied to under surface of tympanic membrane and held in place by Gelfoam®

Case 5 C B, a 30 year old man, had had a large central perforation of the left tympanic membrane for the past five years. Suppurative otitis media developed whenever he had an upper respiratory infection. There was a conductive loss of 25 db in this ear. The Eustachian tube was patent and the ear was dry. The use of a paper patch eliminated the hearing loss.

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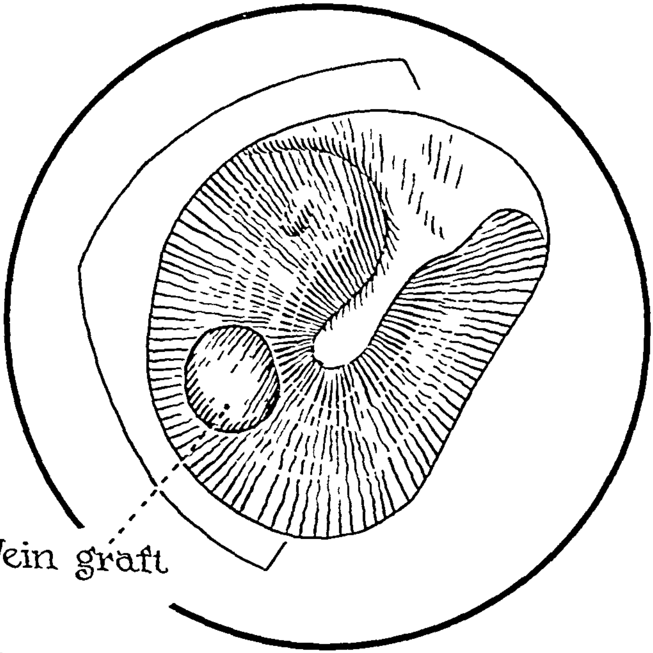

Fig 6 Vein graft in situ after replacement of reflected tympanic membrane

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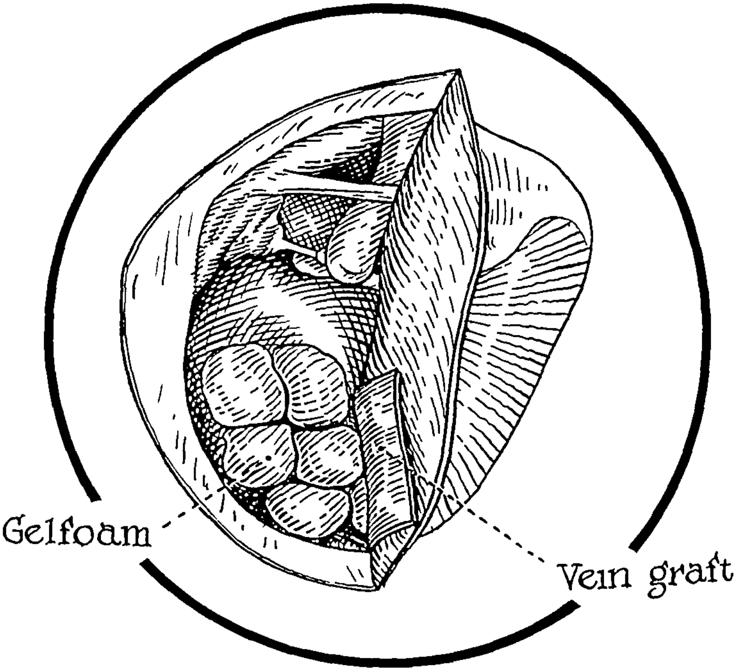

Fig 5 Vein graft applied to under surface of tympanic membrane and held in place by Gelfoam®

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A vein graft was placed through the central perforation and positioned satisfactorily without the use of a stapes incision The perforation healed in 14 days, and at the end of two months the results, including the restoration of hearing to normal, are entirely satisfactory

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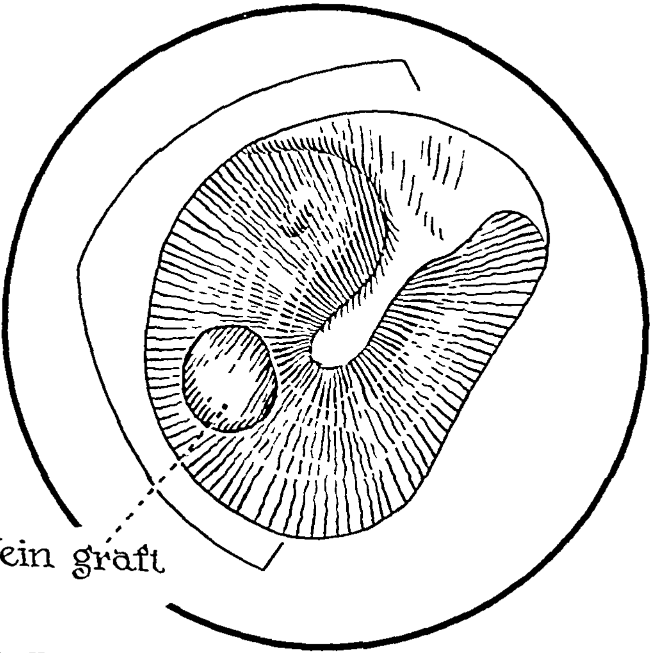

Fig 6 Vein graft in situ after replacement of reflected tympanic membrane

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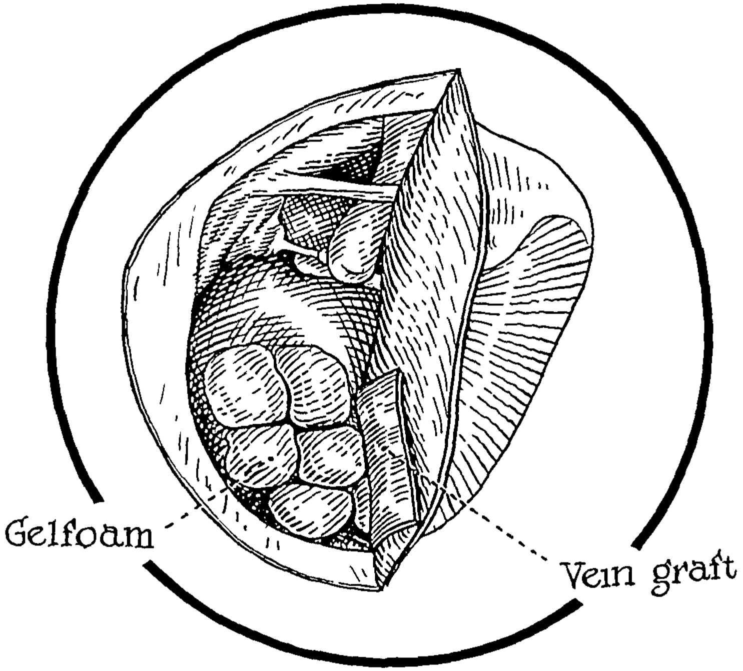

Fig 5 Vein graft applied to under surface of tympanic membrane and held in place by Gelfoam®

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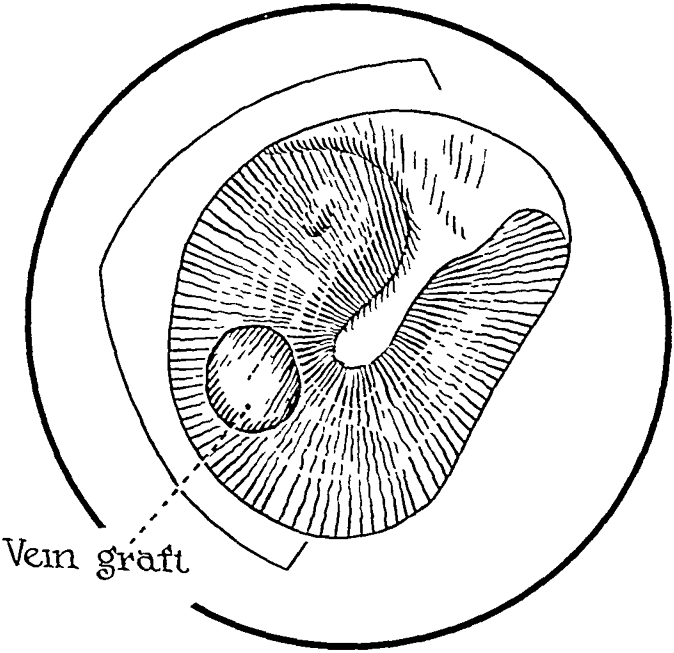

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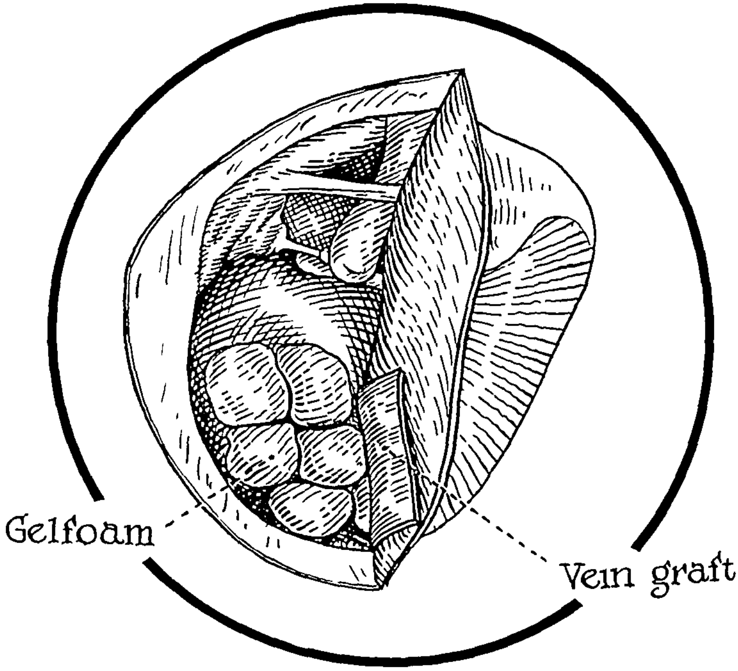

Fig. 5. Vein graft applied to under surface of tympanic membrane and held in place by Gelfoam®.

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TECHNIQUE

In the first cases in this series it was our practice to em-

foot or arm in a child, by the Shea technique The resected vein is sectioned vertically on a lateral aspect and is opened, thus creating a flat, rectangular graft It is kept moist with a drop of saline solution while excess adventitia is removed under magnification

The graft is then placed over the perforation, so that the edges of the aperture are overlapped by it for 2 - 3 mm The endothelial surface of the graft lies toward the tympanic cavity and its adventitial or raw side lies in apposition to the vascular bed created on the under surface of the drum Gelfoam® is applied to hold the graft in position across the perforation (see Fig. 5) The tympanic membrane is replaced in its original position (see Fig 6) and the external

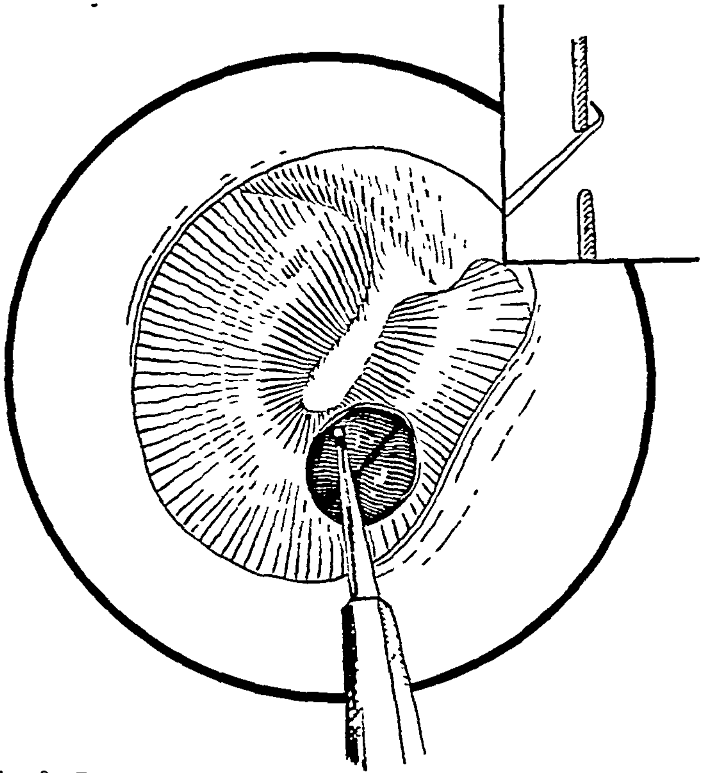

Fig 8 De-epithelization of under surface of eardrum by right-angle knife Insert shows detail of technique

3 The perforation of the tympanic membrane is visualized and inspected with an operative microscope with 10-power magnification

4 A right-angle flap knife with a 2-mm blade (see Fig 3) is inserted through the perforation and is used to de-epithelize the under surface of the eardrum for a distance of 2 - 3 mm

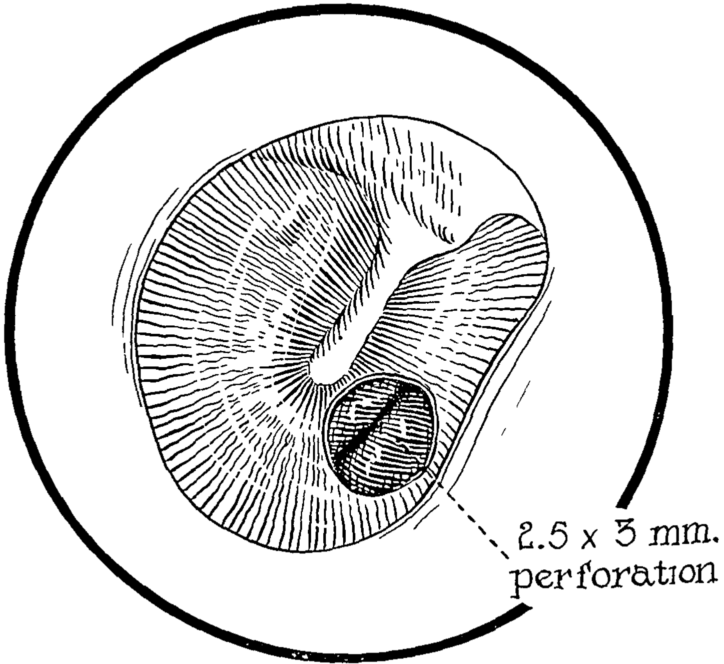

Fig 7 Typical long-standing perforation of anteroinferior quadrant of tympanic membrane

from the edges of the perforation, in order to create a raw vascular bed for nourishment of the vein graft

If the technique first employed is used, a stapes incision is made parallel to the margins of the posterior half of the tympanic membrane The tympano-meatal flap thus created (see Fig 4) is reflected anteriorly, to expose the tympanic cavity and the under surface of the eardrum

A tubular section of vein, usually about 1 cm in length, is taken from the dorsum of the hand in adults, and from the

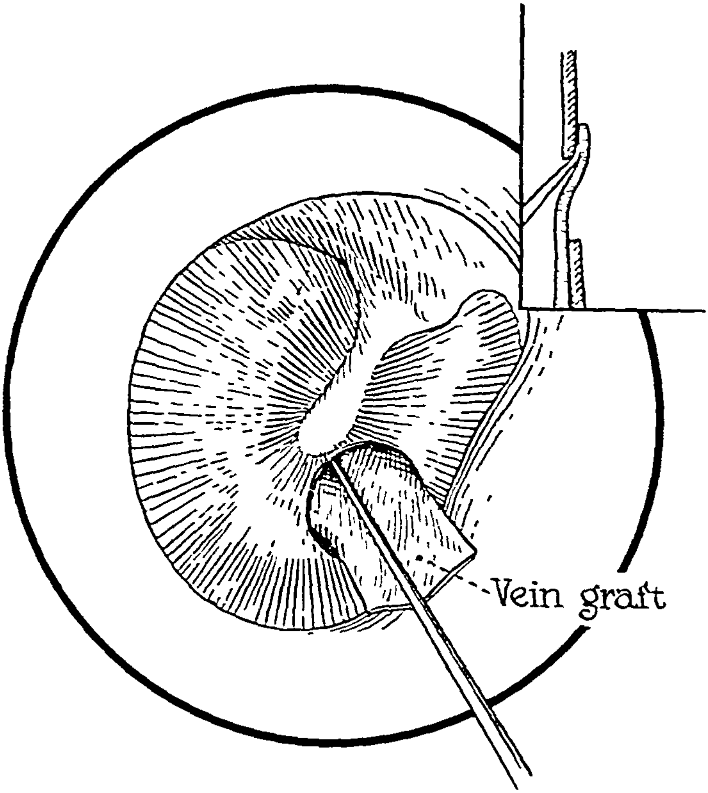

Fig 10 Application of vein graft through perforation Inset shows graft being tucked under edges of perforation

cosal layer of the tympanic membrane In all of our cases, the graft was placed either underneath the tympanic membrane or on top of it, after de-epithelization of the bed on which the graft was to rest This technique was followed whether or not a stapes incision was used

COMMENT

There is no question that perforations of the tympanic membrane should be closed, to preserve aural health and function There is also little argument that surgery is the technique of choice for all children and for most adults It accomplishes healing in a shorter time than office methods and the percentage of permanent good results is higher surface of the drum is covered with Gelfoam® to a depth of about 0 5 cm

The initial steps of the second technique, as already noted, are the same as far as the de-epithelization procedure (see Figs 7, 8) The stapes incision is omitted, and Gelfoam® soaked in physiologic salt solution is introduced through the perforation (see Fig 9), to ensure good contact of the graft and the under surface of the tympanic membrane The graft is then introduced through the perforation (see Fig 10) and fitted to the under aspect of the drum, so that, as in the first technique, there is an overlap of 2 mm around the edges of the perforation (see Fig 11) It is held in place by Gelfoam,® which is also used to cover the external surface of the tympanic membrane

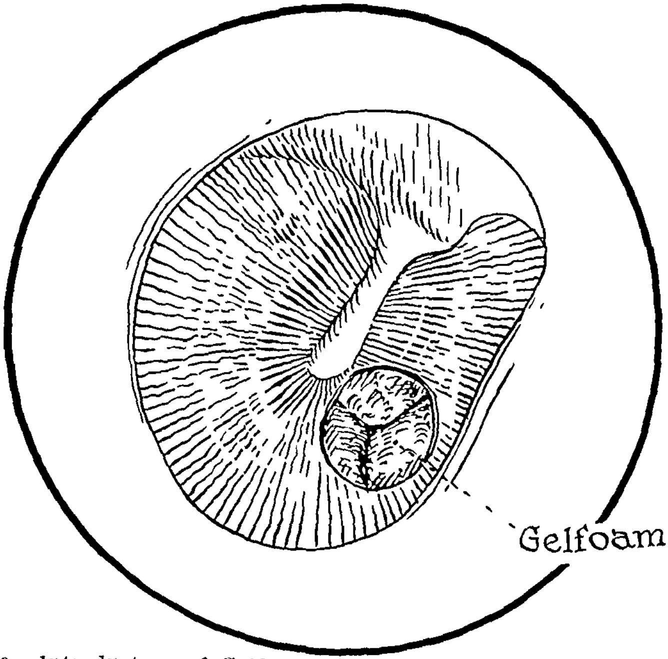

Fig 9 Introduction of Gelfoam® through perforation to ensure good contact of graft and under surface of tympanic membrane

In the technique devised by Dr Shea, the vein graft is placed between the outer epidermal layer and the inner mufor a distance of a few millimeters away from its margin not only provides a good bed for the graft but also removes any epithelium which may have grown around the edges of the perforation onto the under surface and which would interfere with closure The presence of epithelium in this location was advanced by Dunlap and Schuknecht[3] in 1947 as a possible cause for non-closure of these perforations

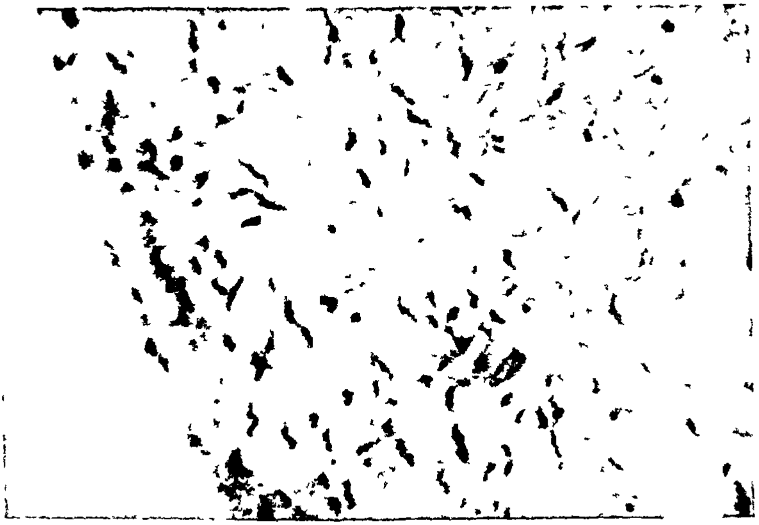

Fig 12-a Microscopic demonstration of vein from hand showing endothelium muscular coat and adventitia

We have demonstrated that autogenous vein grafts will grow over or under perforations of the tympanic membrane (see Fig 12-b) and will seal perforations in old skin grafts (see Fig 13)

Vein grafts under perforations of the tympanic membrane appear grossly to serve as tissue splints Apparently they have the ability to extract an extra blood supply and produce an inflammatory response around the perforation, so that natural closure, by proliferation of the squamous epithelium, is stimulated

When healing of the perforation is observed under the ear

These facts were generally true even when skin grafts, with their undesirable features, were used to close the perforated tympanic membrane Dr Shea's experience, like my own, indicates that results are much better when vein grafts are used For this there are a number of reasons Veins (see Fig 12-a) are thinner and more pliable than skin and, therefore, easier to work with They can be procured in suitable sizes from the hand, foot, arm, or even the neck, with hairline incisions They are easy to prepare for grafting They contain no keratinizing epithelium They are easily applied with an ear microscope through an incision in the canal or through the perforation itself, without an endaural or postauricular incision They adapt well to the contours of the ear They usually require very little contact for nourishment and will bridge areas of 7 - 8 mm without sloughing

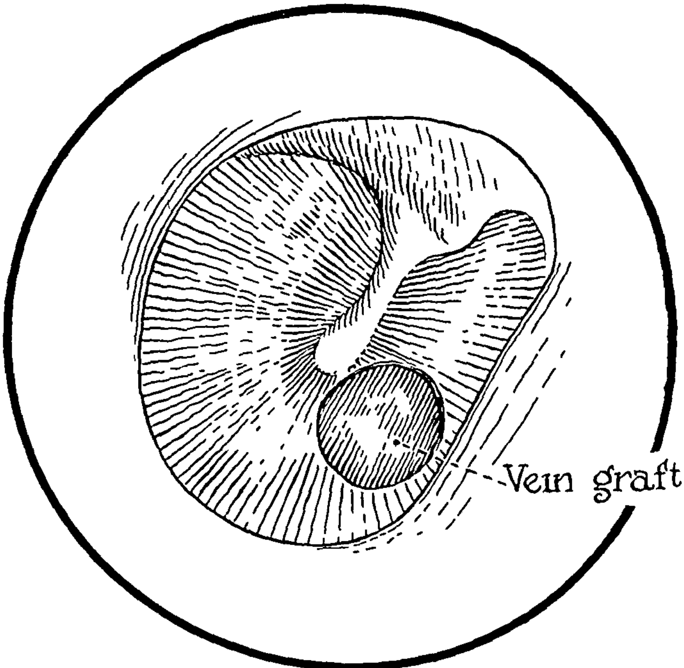

Fig 11 Vein graft in situ under perforation The operation is easily performed without the stapes incision

De-epithelization of the under surface of the perforation

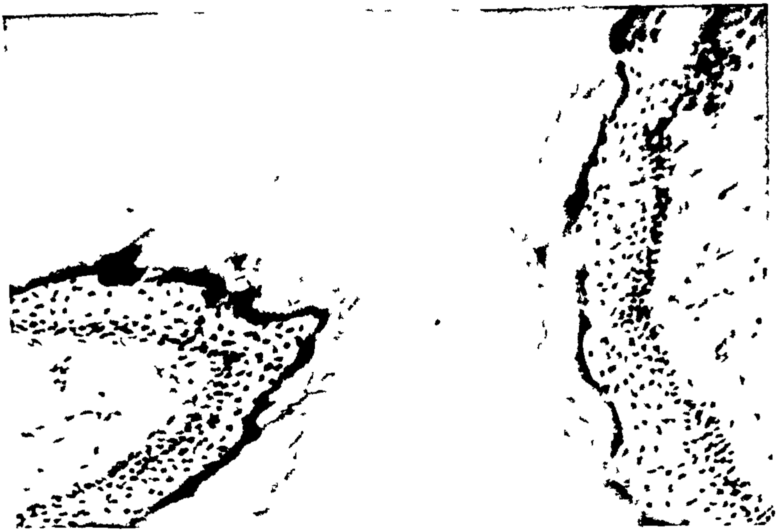

Fig 13 Microscopic demonstration of squamous epithelium growing around edges of old perforation in full-thickness skin graft Secondary vein graft repair was successful

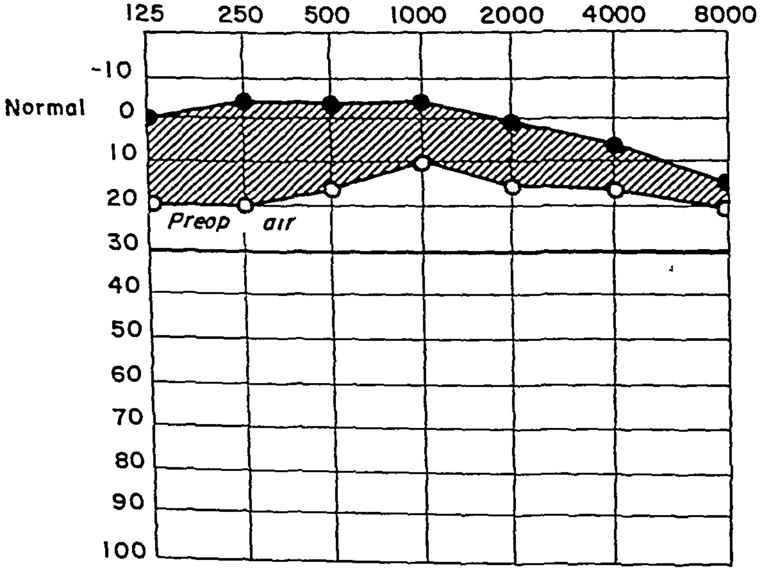

Fig 14 Average gain in hearing in five cases of successful vein-graft closure of perforations of tympanic membrane

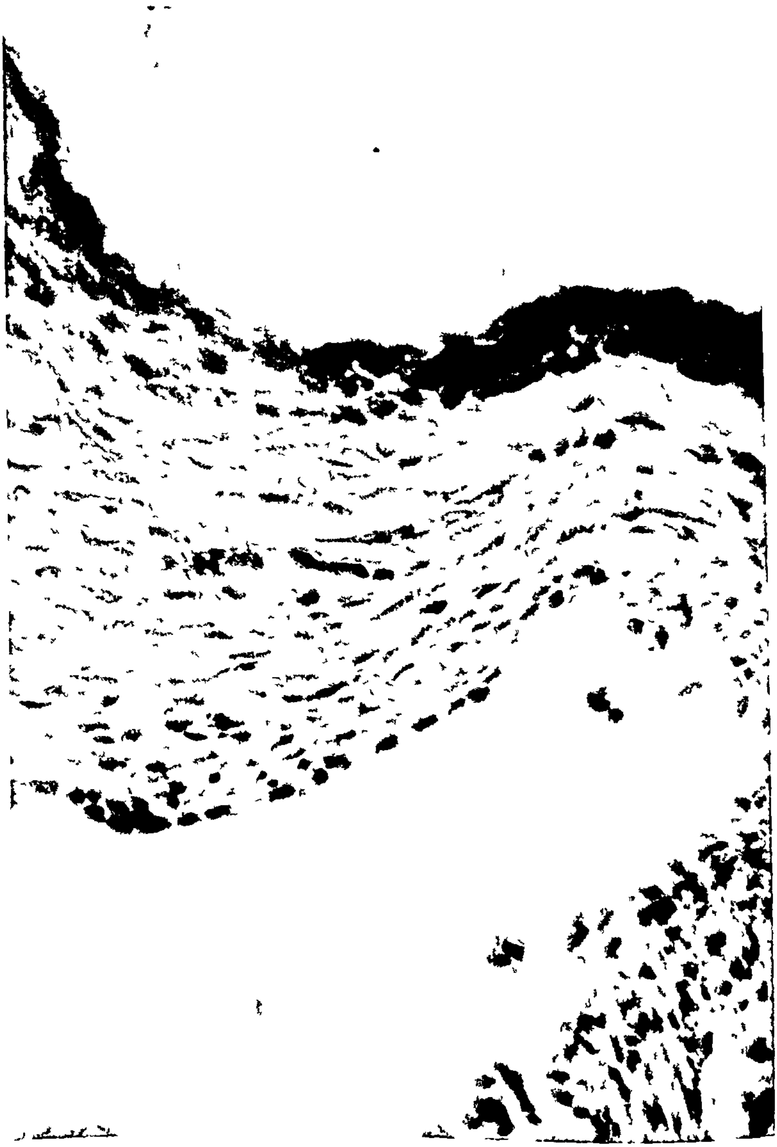

Fig 12-b Experimental demonstration showing healing of perforation of drum four weeks after application of vein graft Note squamous epithelium on graft

ALLERGIC MANIFESTATIONS IN OTOLOGIC DISEASE *†‡

BEN H SENTURIA, M D ,

St Louis, Mo

One finds many reports in the literature purporting to deal with allergic manifestations in the external and middle ear, but little basic investigative work is presented to substantiate these claims In this presentation, some widely accepted allergic concepts dealing with the external and middle ear are considered and discussed in the light of our own experience and investigative work[1] These problems have been reviewed recently by Hoople,[2] Koch,[3] Derlacki,[4] and Kuhn[5]

It is necessary, first, to define my concept of the allergic state before we enter into any discussion of the role it may play in these otologic conditions It is agreed by most workers that the original Von Pirquet definition of allergy as an "altered reactivity of the tissues" is much too broad in the light of our present knowledge, furthermore, it is difficult to accept the suggestion[6] that any phenomena which result from autonomic dysfunction without obvious antigenic stimuli or classical immunological changes should be included under the term "allergy"

I do not insist that the term "allergy" be restricted to an acquired specific alteration in the capacity to react predicated on an antigen antibody mechanism,[7] but I prefer to narrow the definition of an allergic state to those listed by Rackemann and others[8,9]

1 A history of either past or current allergic symptoms or signs

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microscope, within three or four days large blood vessels can be seen crossing the tympanic membrane under the epidermis and penetrating the graft The entire membrane and graft are diffusely hyperemic Within seven days, a seal is present between the graft and the membrane, and the perforation is perceptibly smaller Within two to three weeks, healing is complete The surface epithelium bridges the perforation and the vein appears as a pink, subepithelial area of thickening

In a number of the cases in this series, as indicated in those presented in detail, the vein-grafted tympanic membrane successfully withstood the hazards of swimming as well as suppurative middle ear and external ear infections, without damage to the graft In all of the cases treated, hearing losses were improved or completely eliminated (see Fig 14) Dr Shea in his report does not comment on his results in regard to hearing

CONCLUSION

At the present time, surgical repair of perforations of the tympanic membrane, by the Shea technique of autogenous vein grafts, seems to offer a quick and apparently certain method of closure with, at the same time, restoration of maximal hearing

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are frequently associated lesions of the eyelids, face, sides of the neck, popliteal and anti-cubital fossae The skin affected is erythematous, scaly and excoriated Such a reaction may occur in any part of the ear, in which case the area involved often becomes thickened and inflamed with oozing patches In about 50 per cent of the cases there is an associated history of other allergic diseases such as asthma, hay fever, migraine and urticaria A familial history of these diseases is common Such patients possess a high percentage of circulating antibodies in their blood serum This disease does not respond well to the allergic approach, however, and there is thought to be some additional factor responsible for the reaction

C *Drug Eruptions*

Morphologically the drug eruption may assume any form from simple erythema to vesiculation, papule and bullae formation With very few exceptions, any drug given orally or parenterally is capable of producing specific sensitization Some of the common offenders are penicillin, "sulfa" drugs, barbiturates, salicylates, bromides, iodides, arsenic, quinine, etc As a rule, the rest of the body is also affected, but the eruption may be "fixed" to the ear alone

D *Infectious Eczematoid Dermatitis*

In this disease there are visible on the skin of the auricle and periauricular tissues, discrete erythematous, weeping, vesiculated and crusted lesions An adjacent focus of infection which is usually a pyoderma or a draining purulent otitis media and/or mastoiditis can be demonstrated It is presumed that the discharge sensitizes the peripheral skin over which it passes

E *Physical Allergy*

The ear may be affected as part of the general reaction which results from exposure to cold, heat, pressure, ultra-violet rays, or rays from the visible spectrum The usual manifestation is urticaria, erythema or eczematization The mechanism may be based on a true antigen-antibody reaction, the antigen being a normal occurring protein which is altered when it is exposed to the physical agent

2 A history of allergy in antecedents or descendants

3 Remission of symptoms or signs on elimination and recurrence on re-exposure to specific allergen

4 Blood or secretion eosinophilia

5 Positive scratch, intradermal or patch tests

6 Demonstration of circulating antibodies (passive-transfer tests)

THE EXTERNAL EAR

Many otologists are convinced that allergy plays a major role in external otitis With the above concept of the allergic state in mind, let us examine some of the diseases which we have listed as "allergic" in our classification of diseases of the external ear [1,10]

A Contact Type Dermatitis (Dermatitis Venenata)

The allergic inflammation herein produced is a result of the contact of the offending allergenic substances (animal, vegetable or mineral) with the hypersensitive epithelial cells of the epidermis A so-called period of incubation or sensitivity varying from approximately five to 21 days is required for the production of large quantities of antibodies and the development of the hypersensitive state The reaction consists of erythema, edema, vesiculation, bullous formation and crusting, and is accompanied by a sensation of itching, burning, stinging and pain in varying degrees Involution occurs when the contactant is removed or neutralized The usual substances responsible for this type of dermatitis in the ears are ear medications, cosmetics applied to the scalp and face, eyeglass temples, telephone receivers, hair lacquers, perfumes, colognes and earrings Actually, any medication used in and around the ear is potentially capable of sensitizing the skin Patch tests with the suspected agents are very successful in isolating the responsible allergens

B Atopic Dermatitis

This is a chronic dermatitis with intense pruritis There in common pyogenic inflammation, and were not found in skin biopsies of patients with chronic otitis media [12]

One might argue in retrospect that there are certain clinical and pathological aspects of external otitis which are compatible with a state of allergy Among these are the recurrent nature of external otitis, dry skin and persistent pruritis prior to the acute exacerbation, vascular changes and a high percentage of eosinophiles On the other hand, the mere presence of eosinophiles does not provide a definite diagnosis of allergy since these cells may be associated with many other conditions such as tissue anoxia, parasitic infections Loeffler's disease, etc , and it is not unusual to find an increase in eosinophiles in some inflammatory processes

Of much significance was the recent study by Jones and Norman,[13] who reported on biopsies obtained from 20 patients with external otitis In only two specimens was there any striking increase in tissue eosinophiles and the authors expressed the opinion that "nothing in the histological features of the inflammatory process suggest the hypersensitive mechanism '

It is interesting that 14 of their patients showed 3 per cent or more eosinophiles in the peripheral blood, and two showed a distinct increase in the number of eosinophiles present in the tissue exudate If these patients gave a positive family history of allergy and of persistent pruritis or chronic eczema it might be desirable to consider some of these ears as allergic-suspect, and a search for further evidence of the allergic state would be indicated

I am actually pleased that in this large series reported by Jones and Norman, only a small number of the biopsies showed a significant increase in eosinophiles On the basis of my clinical experience with acute diffuse external otitis I was not happy with the implications of our own histopathologic findings, however, although I do not believe that a state of hypersensitivity plays a frequent role in acute external otitis, I would urge that we keep in mind that a chronic allergic dermatosis may provide the fertile substrate in which there *may* be superimposed infection with Gram-negative

The largest area of difficult differential diagnosis lies in the overlapping of neurogenic and allergic dermatoses In the neurogenic group we include simple pruritis, neurodermatitis, neurotic excoriations and artifact dermatitis The persistent itching which is invariably present may easily be interpreted as a manifestation of an allergic reaction If none of the postulates of an allergic state exist one should attempt to ferret out basic underlying neurogenic factors

In simple pruritis and localized neurodermatitis a proper understanding on the part of the patient will frequently bring about rapid control of the condition On the other hand, in many cases superficial psychotherapy by the otologist, or more profound therapy by the psychiatrist will be necessary to control properly generalized neurodermatitis, neurotic excoriations, etc The sympathetic and conscientious allergist will provide the neurogenic patient with a much needed crutch and in many instances will give relief as a consequence of the interest and care provided, but we must not misinterpret these results as evidence for an allergic etiology when in fact we are dealing with a skin manifestation of a severe underlying psychological conflict

In an effort to understand something of the pathogenesis of external otitis, Dr Lawrence Sophian and the author obtained biopsies of the skin from five patients with the history and findings of acute diffuse external otitis[11] No allergic history was elicited from any of the subjects, but four of them reported recurrent ear infections Because of the very severe acute dermatitis, incision and drainage were required, thus it was possible to take biopsies Serial sections were prepared

In addition to the very acute inflammatory changes present, a differential count on the dermal infiltrate revealed an average for all specimens of 24 per cent *eosinophiles* (range 12 to 31 per cent) The capillary and arteriolar vessels showed dilatation, endothelial swelling, hyperplasia and sometimes thrombosis, necrosis, and vascular reactions which can be associated with allergic inflammation These findings were at a substantial variance with what would be anticipated

It is necessary that we clearly differentiate the chronic draining ear from that category of middle ear disease which we will refer to as otitis media with effusion In this latter condition we are dealing with various types of middle ear effusions with an intact tympanic membrane

Lewis[20] and Proetz[21] suggested that localized allergic edema in the tympanum could simulate acute purulent otitis media King[22] studied 72 ears of 56 children and found that seven of the 39 ears with mucous secretion showed five to ten per cent eosinophiles, while none of the children with serous effusion had any eosinophiles Ivstam[23] reported smears from 33 cases with secretory catarrh, and 13 of these showed eosinophiles in very small numbers

Derlacki[4] stated that "the majority of the cases of chronic secretory otitis with either a clear serum or a much thicker mucus are due to a definite allergy to house-dust or to a food" but offers no substantiating evidence Jordan[24] reported on 123 patients with middle ear effusion among whom 91 (74 per cent) were diagnosed as allergic on the basis of the findings of eosinophiles in nasal smears, positive skin tests and response to allergic therapy, yet Jordan found that direct smears of the effusion showed only occasional eosinophiles, scattered leukocytes and no bacteria

Suehs[25] saw only one eosinophile in smears obtained from some 50 patients Robison and Nicholas[26] found no eosinophiles in a hundred subjects with acute and chronic otitis media and secretory catarrh In some 50 cases of serous otitis studied by the Bryans[27] all smears were negative for eosinophiles

As part of a comprehensive study of tubo-tympanitis, Carr and Senturia[28] examined the histories and aspirated effusions obtained from 151 ears (107 patients) Despite a thorough cytologic study using Wright's and Papanicolaou stains, only six of the smears showed a single eosinophile on the entire slide, one specimen showed five cells per high power field

A short time ago we presented our evidence in support of the view that the largest percentage of middle ear effusions bacilli[14] A picture of fulminating acute diffuse external otitis might then overshadow the basic underlying allergic state

THE MIDDLE EAR

Much has been written about the allergic middle ear Unavoidably, some confusion has resulted from combining under one title of chronic otitis media, those ears with middle ear effusion and an intact drum (otitis media with effusion, secretory catarrh, catarrhal otitis) and those with a perforated tympanic membrane and chronic otorrhea

Dohlman[15, 16] and Koch[3] have written at great length regarding the frequency of allergic changes in the chronic draining middle ear Dohlman emphasized the importance of an allergic evaluation in patients with non-responsive chronic otitis media and demonstrated eosinophiles in the secretions Koch described in great detail the cytologic changes present in the secretion of cases with chronic otitis media with perforation He found a significant number of eosinophiles and changes compatible with the allergic state in 19 8 per cent of his 262 cases

Derlacki[17] described the characteristic picture of allergic chronic otitis as showing viscous or gelatinous secretion with a considerable number of eosinophiles, thickened mucoperiosteum, protracted healing occurring in allergic individuals and good response to anti-allergic treatment He stated that in recurrent acute otitis media an infectious inflammatory process may be superimposed on an allergic tympanum and tubal mucosa Derlacki is of the opinion that in chronic allergic suppurative otitis media with a benign central perforation, an acute infection of the tympanum either precedes or is concurrent with an allergic involvement of the tubal and middle ear mucosa

On the other hand, Dean and Pfingsten[18] found an increase in eosinophiles in only one of 26 cases of chronic otitis media studied Ojala and Palva[19] observed 124 cases of acute and chronic otitis media and mentioned only one instance of apparent allergic origin impression that the data provides additional support for the earlier conclusion that most effusions in humans are of inflammatory origin

I am in agreement that many patients with middle ear effusions show an allergic diathesis, including a family or personal history of allergy, pale, hypertrophic nasal mucous membrane, mucopurulent secretion in the nasal fossae with increased numbers of eosinophiles and lymphoid hyperplasia of the naso- and oropharynx These individuals appear to be more susceptible to upper respiratory infections and possibly are more prone to middle ear effusions

From what has been said above, it is clear that there is an imposing lack of evidence to substantiate the claim of an allergic etiology for the great majority of cases of otitis media with effusions, therefore, it is suggested that immediate treatment should be directed to the control of the inflammatory process in the middle ear and sinuses and, following this, attention should be given to the discovery and elimination of the factors which are contributing to the allergic state

SUMMARY

As indicated at the beginning of this presentation, it is my feeling that there is a distinct place for the diagnosis of allergic otitis externa and otitis media There is some clinical evidence that these conditions constitute a consistent though small percentage of our cases in otologic practice They are frequently misdiagnosed and, therefore, respond poorly to the conventional therapeutic approach

I am hopeful that a greater appreciation and understanding of the allergic dermatoses will permit earlier diagnosis and therapy and, therefore, will effectively circumvent the aggravating superimposed acute dermatitis

More investigative work is necessary to delineate adequately the allergic middle ear since we have few reliable yardsticks to differentiate clearly these allergic effusions from inflammaare not allergic but inflammatory in origin[29] A number of the specimens revealed a thin, low viscosity secretion in which there was an absence of inflammatory cells (serous type) Some of the specimens were typical of suppurative otitis media and presented classical inflammatory cytologic findings (purulent type) The largest number had microscopic characteristics of a subacute or chronic inflammation (mucopurulent)

A very small number of our specimens showed a large amount of mucoid material and no bacteriologic or cellular elements The findings suggested that there had been an outpouring of sterile mucoid secretions without any classical inflammatory components It is conceivable that these may constitute the allergic group, notwithstanding our inability to demonstrate the presence of eosinophiles (see Table I)

TABLE I

Diagnosis	Synonyms	Characteristics		
		Cellular Remnants	Neutrophiles	Mucous Strands
Serous	Otitis media with effusion Secretory otitis Secretory catarrh	Rare	Rare	None
Purulent	Suppurative otitis media Acute otitis media	Moderate	Numerous	Rare
Mucopurulent	Chronic otitis media Glue ear Catarrhal otitis media Mucoid ear	Numerous	Few	Numerous
Mucoid	Allergic otitis media (?) Glue ear	Rare	None	Myriads

The following working classification of middle ear effusions was set up on the basis of the available literature and our own clinical and cytologic findings[28]

In an effort to understand the pathogenesis of these conditions, effusions were produced experimentally in dogs and were studied in the same manner as in humans[30] It is our

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OTOLOGICAL FUNCTIONAL MICRO-SURGERY POST GRADUATE COURSE

A practical course of surgical techniques in otological surgery will be conducted by Dr Michel Portmann at the University of Bordeaux, France, Ear, Nose and Throat Department, June 26 to July 5, 1960 This course will be given in English

The number of assistants will be limited to twenty, divided into two groups The fee of $100 00 is to be paid at the Faculty of Medicine of Bordeaux, at the beginning of the course Applications should be sent to Dr Michel Portmann, 45 Cours Foch, Bordeaux, France

tory conditions in the tympanum We are confident that progress is being made in understanding the allergic otitides

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The author is indebted to Dr Carl Gessert Charles Carr, Elizabeth Baumann and Dorothy Baldwin for assistance in the preparation of this manuscript, and to Dr M D Marcus for suggestions and criticisms

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several specialized fields concerned so that the patient may benefit from an organized attack on the problem

Following a survey of most of the tumors that may occur in the area under discussion, with examples of some of them, the case history of a patient will be given in some detail This case demonstrates the very real problem of diagnosis that may exist in certain cases even in the hands of competent observers

Primary tumors of the frontal sinus may be benign or malignant The benign tumors consist of osteomas, mucoceles and cystic tumors resembling mucoceles, including hemorrhagic cysts and cholesteatomas

In 1938 Hempstead[1] wrote an excellent article on osteomas The literature in more recent years has included articles by Brunner and Spiesman,[2] Gibson and Walker,[3] Zovickian and Cooper,[4] Vadala and Somers[5] Several of these referred to an earlier article by Teed[6]

Osteomas may be composed of eburnated or cancellous bone or a mixture of the two They may be attached to the interior of the frontal sinus at any point, but the attachment is usually by cancellous bone Small osteomas not obstructing the drainage of the sinus are usually discovered only on roentgenological examination The density of the shadow is that of bone Further roentgenological examination of these tumors should be made from time to time, and if there is evidence of enlargement, removal should be advised This should be possible through the floor of the frontal sinus If small tumors obstruct drainage, symptoms of the obstruction may be present, or a mucocele may be formed requiring operation

If they are first seen at a stage when erosion through the floor, intersinus septum or anterior or posterior table may have taken place, there may be evidence of involvement of the orbit, the other sinus, brow or meninges Expansion toward the brain may result in meningitis, brain abscess, pneumatocele or cerebrospinal rhinorrhea, and there may be deep involvement of the brain substance Removal of the larger tumors requires wider exposure It may be necessary to

TUMORS OF THE FRONTAL SINUS

DELMAR F WEAVER, M D ,

Detroit, Mich

A survey of the medical literature dealing with tumors originating in the frontal sinus and those of the surrounding areas indicates that a number of competent observers have written articles on the subject down through the years Since many of these tumors rarely occur, often the article follows a general pattern First there is a review of the literature on the particular tumor followed by statistics regarding the relative frequency of occurrence, the total number of cases reported to date, the various theories as to their origin and helps in diagnosis and methods of treatment Usually there are reports of several cases from the authors' personal experience Each article adds to the total store of information and is of immeasurable value towards a complete understanding of the subject, however, it may be worth while to approach the subject from an overall standpoint in an effort to facilitate the evaluation and handling of patients who present themselves with evidence of such tumors

The frontal sinus is a mucous membrane lined space which connects the orbit, nasal cavity, brain and brow It appears to be of no use to modern man Only a few types of tumors originate in it, and each of these is seen infrequently Tumors originating in the surrounding structures are relatively uncommon, and invasion of the frontal sinus by these is seen infrequently also, however, the presence of a tumor in this general area may portend a very grave problem for any particular patient Not only is visual acuity apt to be affected when the orbit is involved but erosion of the bone over the dura mater also may lead to dangerous and fatal intracranial complications Consequently, it is essential that there be reciprocal understanding between the representatives of the

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fronto-ethmoid incision is indicated and if present, it should be removed in its entirety

A variety of suggestions regarding the nasofrontal duct has been made by many authors however, since this is an individualistic problem and more pertinent to chronic infections of the sinus, no attempt will be made to evaluate it at this time There is some reason to believe that frontal duct problems may be less likely to arise after mucocele removal than following operation for chronic infections of the frontal sinus

Simonton and Medwick[8] have discussed tumors of the frontal sinus simulating mucocele, including hemorrhagic cysts and cholesteatomas

Primary malignant tumors of the frontal sinus are quite rare Sciarra and Hallberg[9] reported one case of squamous cell carcinoma, grade III, which arose in the frontal sinus without question It had eroded both tables of the frontal sinus, and the dura was involved The patient was alive and well for at least 12 years after simple excision and implantation of radium They found a total of 90 cases reported as primary malignant tumors of the frontal sinus This included their own case, however, the possibility of some duplication of cases was raised and also a question of whether or not all of them actually originated in the frontal sinus, since in some cases the other sinuses also were involved

The term secondary tumors is used to denote those that have invaded the frontal sinus from a primary origin in surrounding areas and includes those tumors of the skin, subcutaneous tissue and muscle which lie in such close proximity to the sinus that it is thought inadvisable to exclude them This group includes nevi, dermoid, epidermoid and sebaceous cysts, fibromas, lipomas, fibrolipomas, neurofibromas, hemangiomas and certain inflammatory tumors as tuberculomas and Boeck's sarcoid When small enough these tumors can be removed in one stage by simple excision Certain superficial skin tumors such as pigmented nevi may be so large that they must be removed in stages several months apart to prevent distortion of the lids or brow Cavernous remove the anterior table without conserving the supraorbital ridge Zovickian and Cooper[4] have used cancellous bone chips from the iliac crest to repair the defect resulting from the removal of a large osteoma from the frontal sinus

Brunner and Spiesman[2] feel that all osteomas of the frontal sinus can be removed by the external frontal approach Hempstead,[1] however, stated that while most of them can be removed through the fronto-ethmoid incision, if there is evidence of involvement of the dura mater sufficient to indicate the likelihood of a tear or if other intracranial complications are present, the transfrontal approach from the dural side is best

The mucocele is a tumor which has some of the characteristics of an osteoma Kaplan, Schwartz and Metson[7] have discussed various of its aspects It consists of a closed cavity lined with mucous membrane which continues to secrete mucus and to fill the cavity with these secretions It begins as a mechanical block to the drainage of the sinus itself or to the glands of its lining mucosa The mucosa of the sinus continues to produce its normal mucoid secretion, but lacking normal outlet, the secretion accumulates and fills the sinus Continued expansion may cause erosion of the sinus wall and encroachment on any of the adjacent cavities If into the nose, air may pass along the sides and result in a large extra or intradural pneumatocele The cavity most commonly involved secondarily is the orbit and, for this reason, the patient is generally first seen by the ophthalmologist With dural involvement the intracranial complications may be the same as those associated with osteoma

When the contents of a mucocele are invaded by bacterial infection a pyocele is formed

On roentgenological examination the mucocele is represented by a fluid or soft tissue density which may lessen as bony erosion progresses The presence of a cystic tumor in the superior or supermedial quadrant of the orbit should incite one's suspicion of mucocele Primary orbital tumors are more likely to involve the lateral quadrants If there is evidence of a mucocele of the frontal sinus, exploration through a was attached to the floor of the frontal sinus, and it was removed along with the floor of the frontal sinus Plastic closure of the wound was carried out immediately

Four of the patients are alive and without evidence of recurrence 18, 14, 12 and two-and-a-half years since operation

A fifth patient, an 83-year-old man, had been free of recurrence for 12 years when he died soon after an accident This accident occurred in June, 1959 while escaping from a hotel fire in Stalheim, Norway

The sixth patient is under observation now, having been operated on first in September, 1959

Both tables of the frontal sinus exposing the dura widely were removed from one patient The outer table of the frontal sinus with the associated membrane was removed from two patients This exposed the sinus cavity

The outer table of the frontal sinus without perforation of the lining membrane was removed from two patients, and only the floor was removed from the patient who had cold instrument surgery as mentioned above

Five of the postoperative defects were covered with contiguous pedicled flaps Three of the flaps had a single pedicle, and a free graft was used to cover the donor area Two of the flaps were bi-pedicled, and no free graft was used Three of these patients have been mentioned in more detail in other publications[10 11]

Benign tumors such as osteomas and cysts originating in the ethmoid sinus and invading the frontal sinus may produce a frontal duct problem In small frontal sinuses this problem may be resolved in certain cases by removing the entire frontal sinus floor enabling the orbital contents to obliterate it All of the lining membrane must be removed in such cases

While malignant tumors arising in the ethmoid sinus region which invade the frontal sinus are usually squamous cell carcinomas, adenocarcinomas and sarcomas do occur The removal of them whether by cold instrument surgery, electrohemangiomas in this location may be best handled by the injection of sclerosing material or subcutaneous electrocoagulation with an electrode insulated except for its tip

Malignant tumors of the overlying skin which invade the frontal sinus consist chiefly of basal or squamous cell epitheliomas or a mixture of the two I have never seen a melanoepithelioma in this location but presume it could occur When these tumors have not advanced beyond the subcutaneous tissue and muscle and the periosteum has not been invaded, the chance of curing them by excision is above 90 per cent The margin of normal tissue removed should be as wide as feasible, however, if the periosteum has been invaded as indicated by fixation of the tumor or the tumor has been previously irradiated and recurred or if it is infected following inadequate removal, the chance of a cure is much less

In these cases more extensive surgery in indicated and should be planned with the eradication of the tumor the primary objective Repair of the postoperative defect can be carried out later While cold instrument removal of the tumor and the surrounding soft tissue and bone may be suitable in some cases, more often destruction by electrocoagulation is best It eliminates the chance of grafting free tumor cells onto normal tissue and will probably destroy a larger area of tumor with less postoperative defect, however, there is no limit to the extent of removal of tissue that can be carried out with this method When bone has been electrocoagulated to any extent, it will be necessary to remove a sequestrum after about two months

I have operated on six patients who had basal or squamous cell epitheliomas which had originated in the skin around the frontal sinus and had invaded it when first seen by me All had had a variety of combinations of treatment which included cold knife excision, electrocoagulation and irradiation with both radium and X-ray Such treatment had been carried out on several different occasions by several different clinicians

Our treatment was thorough electrocoagulation in five cases and cold instrument excision in one case This patient's tumor

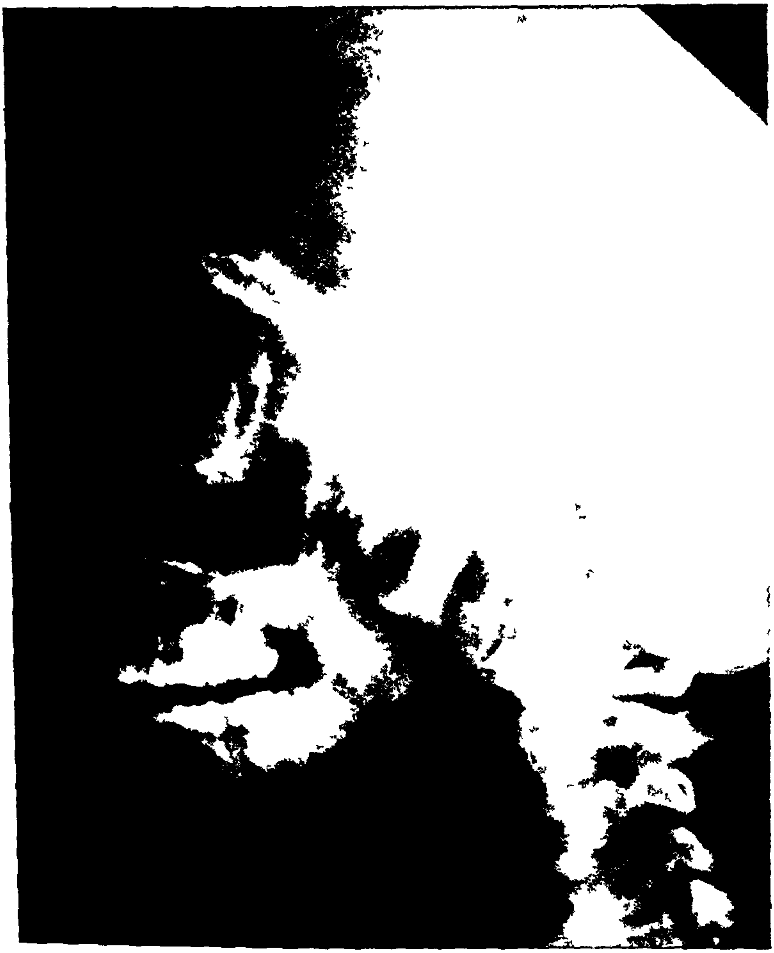

Fig 2 Chondrosarcoma involving skull in region of cribriform plate of ethmoid bone

A 14-year old boy was sent to me for a tonsillectomy and adenoidectomy in October 1959 My attention was called to a tumor in the right nasal cavity It was quite firm After slight manipulation it was necessary to pack the nose tightly to stop profuse hemorrhage Roentgenograms of the sinuses showed extensive involvement of the nasal cavity both ethmoid and frontal sinuses and the right maxillary sinus region (see Figs 1 and 2) Biopsy was reported as chondrosarcoma

On October 15 1959 under pentothal sodium anesthesia with an intratracheal tube in place, the tumor in the right nasal cavity was electrocoagulated as thoroughly as possible and then through a fronto-ethmoid incision extensive electrocoagulation was carried out to provide adequate drainage This extended through the nasal septum into the opposite

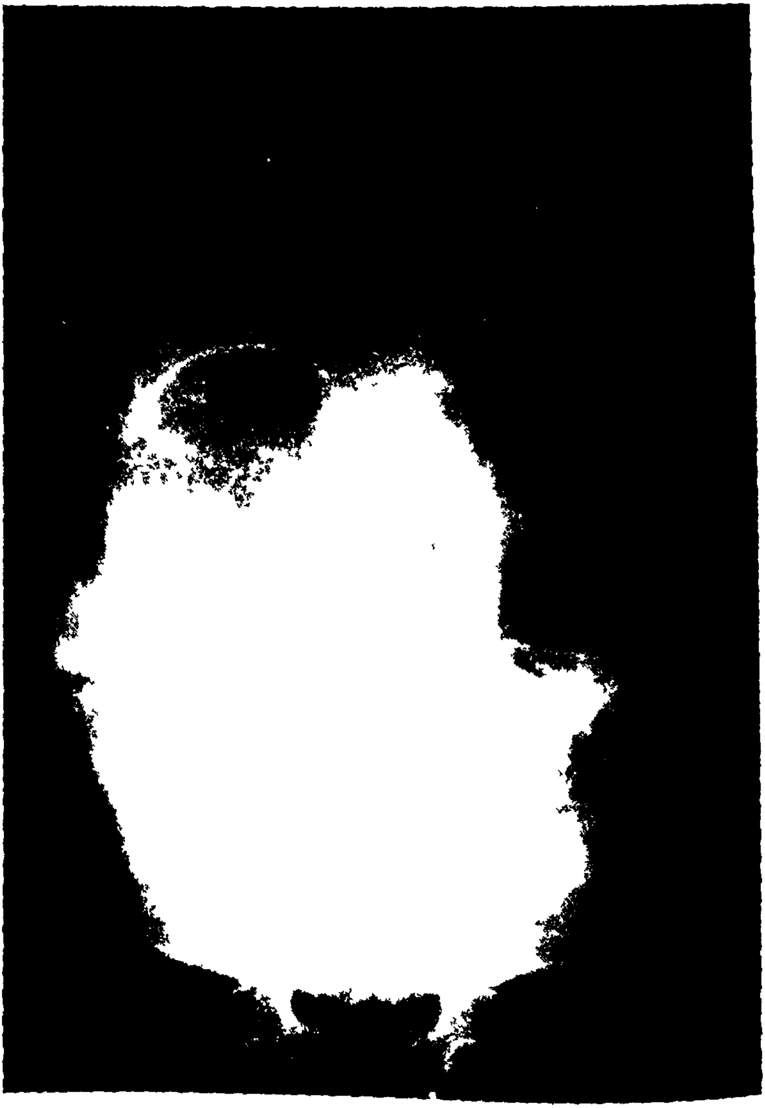

Fig 1 Chondrosarcoma involving both frontal and ethmoid sinuses, nasal cavity and right maxillary sinus region

coagulation or irradiation is simply an extension of the treatment from the primary location

At times it may be necessary to extend surgical treatment into the frontal sinus to provide drainage even though it is not involved by tumor

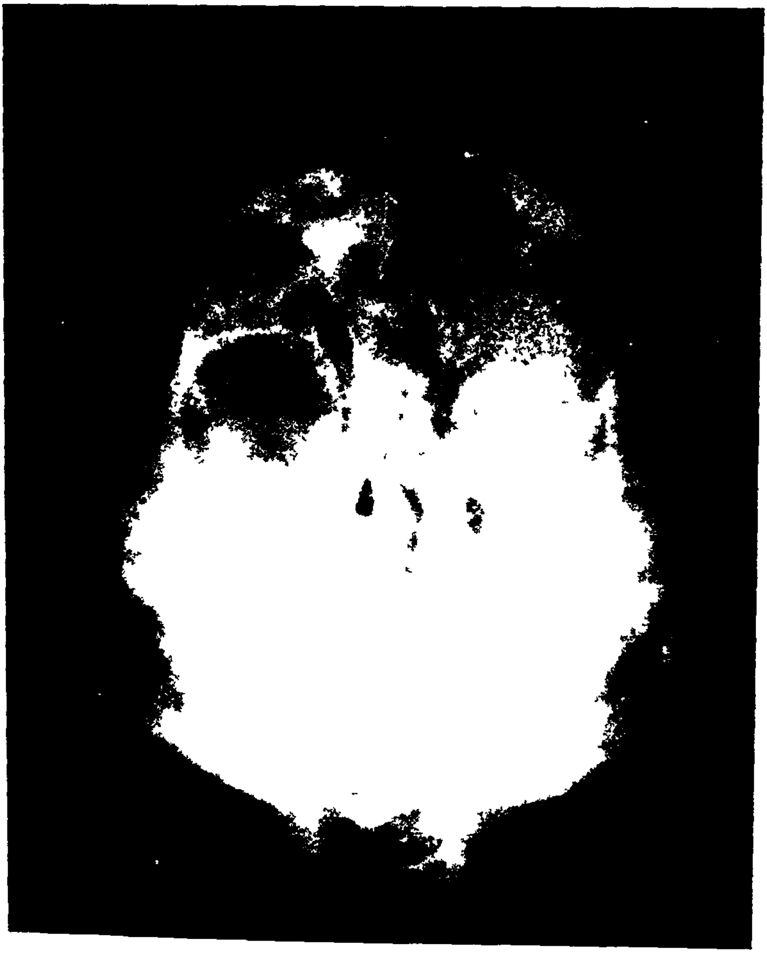

Fig 4 Complete erosion of supraorbital ridge by large mucocele

Little encouragement has been given his parents regarding the final outcome

While tumors originating in the orbit are not common in the practice of the average ophthalmologist there are publications which analyze rather large groups of these tumors

One such report was published by Benedict[12] in 1949 and was based on a study of 740 orbital tumors There were 33 pathological types diagnosed, and 420 of them were operated on

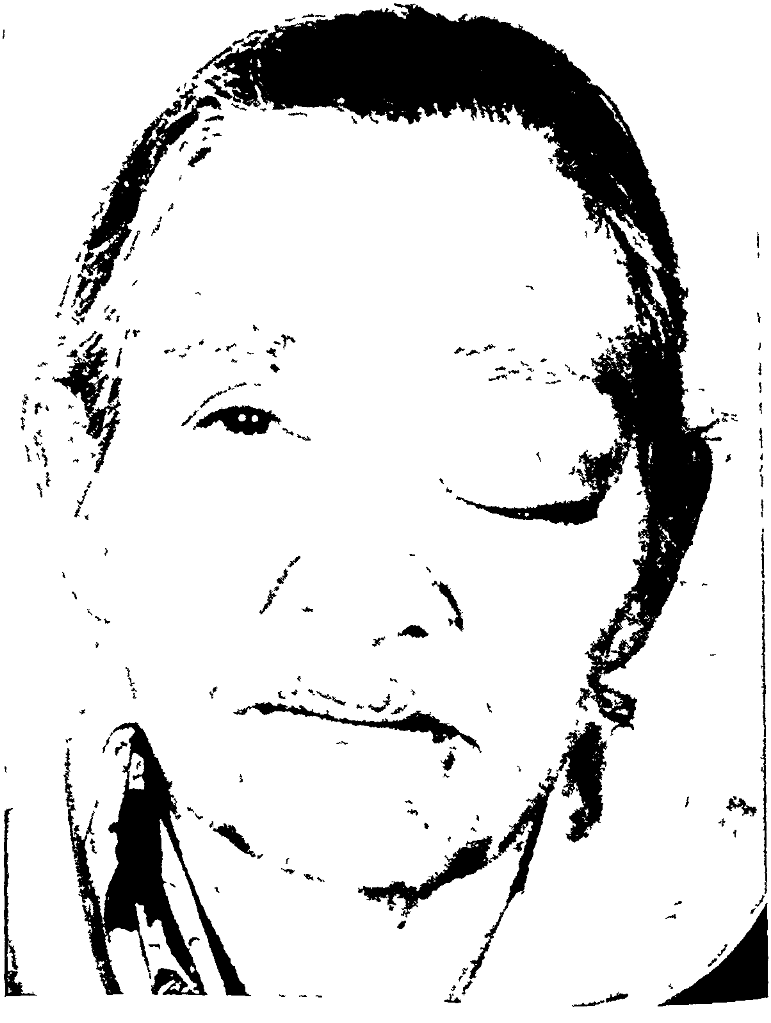

Fig 3 Mucocele of left frontal sinus

ethmoid region The tumor appeared to involve the skull in the region of the cribriform plate The right nasal bone and nasal process of the superior maxilla had been destroyed by the tumor The floor of the right frontal sinus was removed and the sinus found to be filled by the tumor Six capsules containing 10 milligrams of radium each were distributed throughout the cavity and left in place for 46 hours (2760 mgm hours) His condition following operation appears to be improved

Progressive loss of visual acuity may be the outstanding sign of orbital tumors and is a definite indication for operation

Three-hundred-and-forty of Benedict's 420 operated tumors were removed by the frontal or fronto-lateral approach This approach is usually proper for tumors of the anterior third of the orbit as well as those that lie along the superior wall

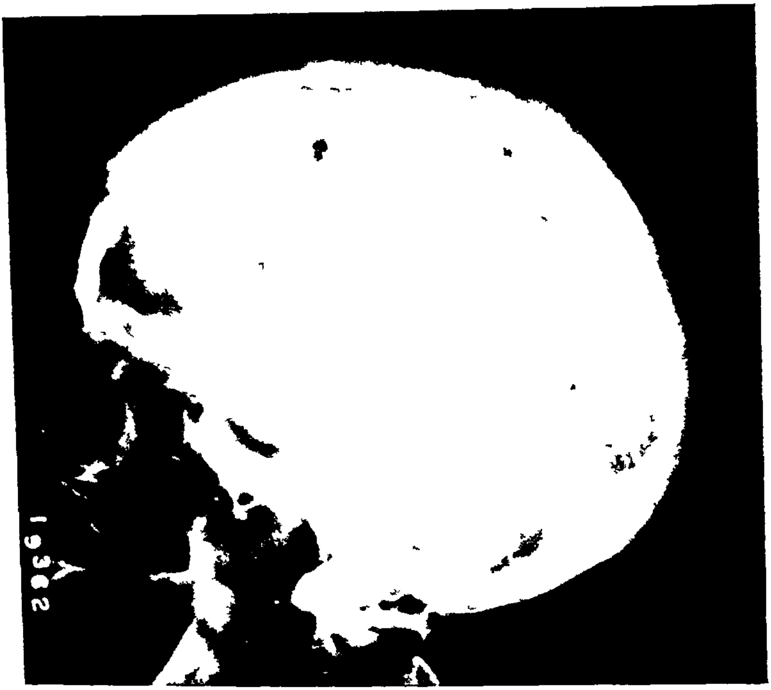

Fig 6 Marked erosion of posterior table of frontal sinus in 1958

Resection of the lateral wall of the orbit (Kronlein's operation) is most suitable for orbits with small outlets and for certain tumors situated in the posterior third of the orbit Other tumors situated in the posterior third and especially those that may be present in both the orbit and cranial cavity, as endotheliomas and hemangiomas, can be reached more directly through a transcranial approach

While, theoretically, many orbital tumors may involve the

These tumors produce exophthalmos, but the amount of proptosis of the globe and direction of its displacement only in general indicate the size and position of the tumor within the orbit. Exophthalmic goiter may produce unilateral exophthalmos and certain inflammatory diseases may do so also.

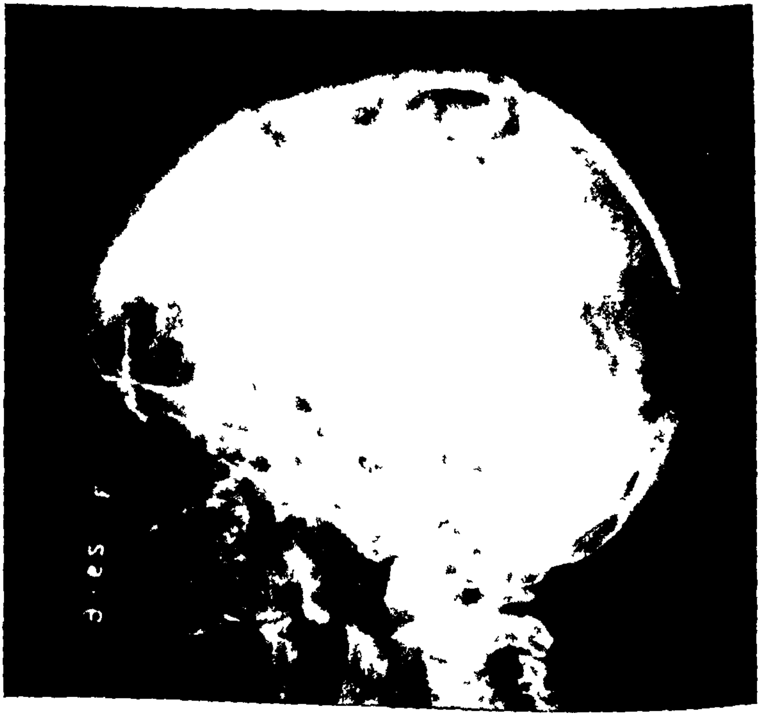

Fig. 5. Posterior table of frontal sinus intact in 1936.

A list of orbital tumors includes hemangiomas, which constitute about a third of them, endotheliomas, osteomas, meningiomas, dermoid cysts, melanomas and mixed tumors and cylindromas (in the lacrimal gland). Metastatic carcinoma of the breast, stomach and intestine may involve the orbit. It is seldom that one can make a diagnosis of any particular type of orbital tumor from clinical evidence alone. They may be confused with mucocele or pyocele of the frontal sinus.

CASE REPORT

A 73 year-old white woman was first seen by me on November 29 1958

She stated that four or five years previously she developed a slight swelling over the left eye associated with periodic closure of the left eyelid due to periodic increase in the swelling The permanent swelling had gradually increased until the present time

She had a history of moderately advanced generalized Paget's disease, moderately severe diabetes with far advanced vascular disease and arteriosclerotic and hypertensive heart disease There was a history of having had a coronary thrombosis and she had been in congestive failure She was taking Quinidine for paroxysmal tachycardia

She had been seen alternately by two excellent ophthalmologists over a period of years These men were not associated Their reports are as follows

"There was no swelling of the orbit in May 1952 In 1955 vision was 20/20 in both eyes and no tumor or swelling was present There was some retinal hemorrhage at that time, however On October 30 1956 there was invasion of the orbit by tumor and blurring of the nasal margin of the nerve head with increased pressure In April 1957 this tumor was palpable at the orbital rim nasally above and below Her vision was still good so in view of the presence of the general diseases surgery was not advised at that time In January 1958 there was a massive tumor in the supraorbital region depressing the eye Retinal hemorrhages were present Surgery was urged at that time"

In March 1958 she was complaining bitterly of pressure and discomfort Neurological consultation was obtained at this time and arteriograms were made The patient was boarded for intracranial surgery but it was decided against due to the apparent likelihood of hopeless intracranial involvement. Her internist considered it likely that the tumor was from sarcomatous degeneration of the frontal bone since sarcomas develop in a significant number of cases of Paget's disease He felt that removal of the tumor with the eye was justified in view of the discomfort she was having He referred her to a third ophthalmologist in May 1958 He aspirated the tumor found that it contained fluid and referred her to me At this time visual acuity was very poor in the affected eye

On examination a large firm tense tumor was evident in the superior half of the left orbit This markedly displaced the globe downward It was difficult to separate the eyelids (see Fig 3)

Roentgenograms showed the tumor extensively involving a large frontal sinus with complete erosion of the supraorbital ridge (see Fig 4) Lateral films made in 1956 showed the posterior table of the frontal sinus intact (see Fig 5) while recent films showed it markedly eroded (see Fig 6) Arteriograms showed moderate posterior displacement of the anterior cerebral artery (see Fig 7)

On December 4 1958 under pentothal sodium anesthesia with an intratracheal tube in place a fronto-ethmoid incision was made which extended to the outer extremity of the eyebrow Almost immediately a large mucocele was exposed which contained at least four ounces of thick liquid of a light green color It did not appear to have been invaded by infection to a significant degree The floor of the frontal sinus was completely absent due to erosion of the cyst, and this was also true of the supraorbital ridge The posterior table of the frontal sinus was likewise eroded exposing the dura over a circular area 4 cm in diameter All of the cyst wall was removed, and the frontonasal duct was recon

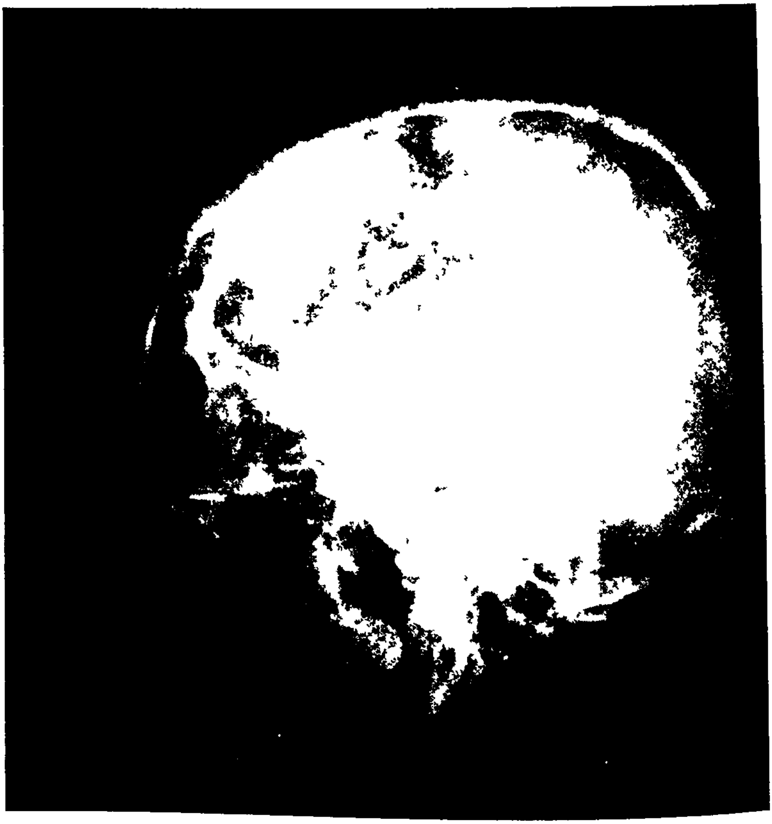

Fig. 7. Arteriogram showing moderate posterior displacement of anterior cerebral artery.

frontal sinus, and we may be called in consultation on these cases, our chief interest in them concerns the problem of diagnosis. In general, those tumors which can be palpated in the frontonasal or medial quadrants of the orbit would tend to be associated with the frontal or ethmoid sinus. These tumors can be approached through the fronto-ethmoid incision. Tumors that lie in a more lateral position, especially if roentgenograms of the sinuses are negative, are apt to be in the realm of ophthalmology.

The meningioma is about the only intracranial tumor that may invade the frontal sinus often enough to merit consideration. This occurrence is quite rare since they usually arise more posteriorly and seldom reach so far forward.

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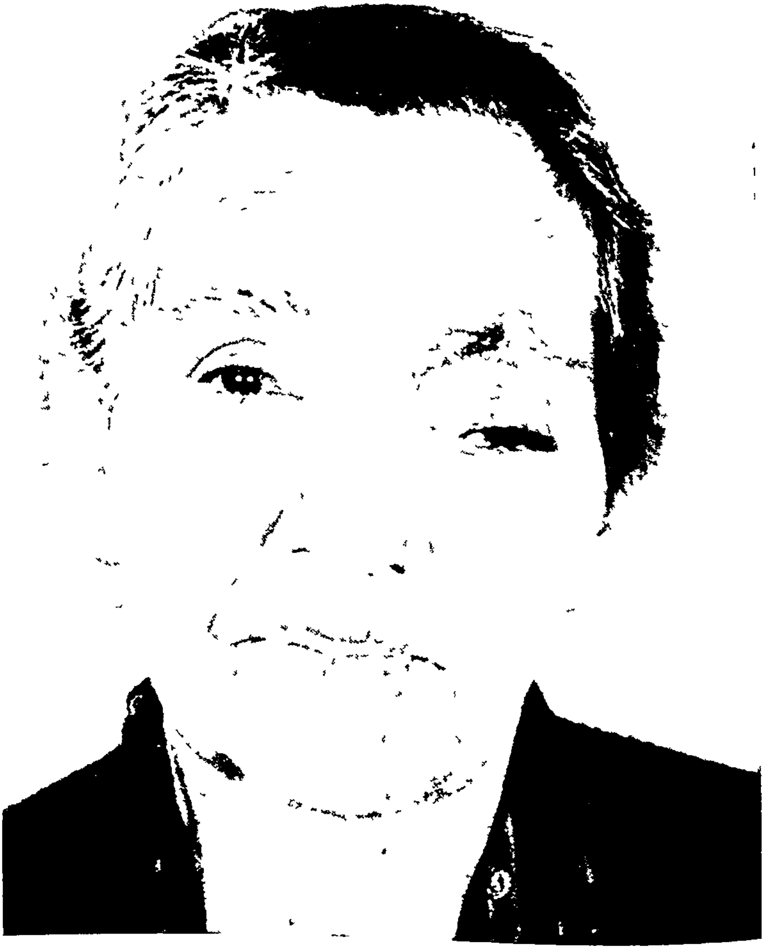

Fig 8 Appearance five weeks after operation

structed A size 26 F rubber catheter was placed through the nose and frontal duct into the frontal sinus cavity however hemorrhage was so troublesome that it was necessary to remove the tube and pack the nasal cavity with Iodoform gauze This was removed in about a week The postoperative course was uneventful except for a moderate mental depression which disappeared in a few days The temperature did not become elevated above normal during the nine days she was in the hospital The eye soon tended to return to a more normal position and there was some improvement in vision (see Fig 8)

The pathological report was acute and chronic inflammation of the frontal sinus

Three months after operation the patient developed slight swelling of the left lower eyelid A large curved suction tip was passed into the sinus through the frontal duct dilating the duct sufficiently so that the swelling soon subsided Several months later she developed swelling but was seen by an otolaryngologist near her home who drained the sinus externally A recent report from him states that she is having no trouble now

An attempt has been made to present a bird's-eye view of tumors of the frontal sinus in an effort to facilitate the handling of patients with evidence of these tumors

The case history of a patient with a large mucocele of the frontal sinus has been presented in some detail This history demonstrates the extreme confusion that may be associated with the diagnosis of tumors that arise in the frontal sinus even when observed by thoroughly competent men in fields closely allied to otolaryngology

One is tempted to suppose that inadequate drainage of the frontal sinus resulting from the deformity of Paget's disease was responsoble for the formation of the mucocele

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THE WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

The program of the West Virginia Academy of Ophthalmology and Otolaryngology has now been completed for their annual meeting at the Greenbrier Hotel, White Sulphur Springs, W Va, April 10-12, 1960 Guests speakers will be Dr Harold G Scheie, Professor of Ophthalmology, University of Pennsylvania School of Medicine, Philadelphia, Pa, "The Surgical Management of Glaucoma," Part I and Part II, Dr Charles E Iliff,Associate Professor of Ophthalmology, John Hopkins University School of Medicine, Baltimore, Md, "Adult Ocular Tumors", Dr Oscar T Becker, Clinical Associate Professor of Otolaryngology, University of Illinois School of Medicine, Chicago, Ill, "Plastic Surgery in Otolaryngology", Dr Helen J Ossofsky, Assistant Professor of Pediatrics, Georgetown University, Washington, D C, "Pediatric Ocular Tumors", Dr John T Dickinson, Department of Otology, University of Pittsburgh, Pittsburgh, Pa, "Modern Treatment of Otosclerosis"

There is a non-member registration fee of $10 00 to cover all the social and scientific sessions For further information contact the secretary, Dr A C Esposito, First Huntington National Bank Building, Huntington, W Va

LARYNGEAL STENOSIS[*]

MERCER G LYNCH, M D,
and
FRANCIS E LEJEUNE, M D

New Orleans, La

We herewith present a method for the correction of laryngeal stenosis produced by web formation between the cords

Laryngeal stenosis as the result of infectious, inflammatory reactions, such as diphtheria, tuberculosis, syphilis, typhoid fever, erysipelas, and smallpox is seen only in exceedingly rare instances now Today trauma to the larynx from transportation accidents, war wounds, surgical injury, and irradiation is much more likely to be the cause of laryngeal stenosis Trauma to the larynx from transportation accidents, war wounds and irradiation usually produces a form of stenosis with involvement of the entire larynx These cases are dealt with in an entirely different manner I will stress mainly those cases of congenital web formation or those following surgical injury As a general rule, congenital web formation involving the larynx occurs from the formation of a web between the vocal cords Usually, this web is very thin or moderately thick, seldom is it ever very dense fibrous tissue and it may be corrected by the method to be mentioned

Surgical injury to the anterior commissure is usually the result of over-enthusiasm on the part of the surgeon In the removal of multiple papillomas of the vocal cord or of tumors involving the anterior commissure, the virginity of the commissure is violated, and there is a raw surface involving both cords in the anterior third As a result of this, scar tissue develops with formation of a web This web is usually moderately thick, but involves only the commissure itself, extending posteriorly on each side, depending upon the amount of injury and raw surface produced by removal of the growth

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[*] Read at the meeting of the Southern Section American Laryngological Rhinological and Otological Society Inc New Orleans La Jan 15 1960

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or unnecessary trauma to the cords, however, the web itself is limited entirely to the cords If this web is extremely thick and fibrous, it must be approached from an external laryngo fissure incision, with removal of the web and insertion of a tantalum splint, to prevent approximation of the raw surfaces of the cords, or, it may be removed and some form of laryngeal stent inserted between the cords in the form of a plastic mold, a hard rubber mold, or a metal mold This, however, necessitates tracheotomy for a period of time In either of these procedures, an external incision is necessary In an attempt to overcome the necessity for external surgical procedure we have tried several methods We present the latest procedure This method, however, can be used only in those patients in whom the web itself is thin or moderately thick, in a dense fibrous scar it is not practical

The patient is suspended, using the suspension laryngoscope In this way, one is able to use both hands to perform the operation After adequate exposure of the larynx a small opening is created as close to the anterior commissure between the vocal cords as is feasible A small polyethylene tube, through which heavy black silk thread or silver wire has been passed, is then inserted through the opening created The polyethylene tube and silver wire now extend subglottically as well as supraglottically A Riverden needle is introduced externally in the medium line of the neck, through the crico-thyroid membrane into the subglottic portion of the larynx, the silver wire is fed into the slot of the Riverden needle and the needle is then withdrawn externally bringing with it the silver wire This same procedure is used by inserting the Riverden needle through the thyro-hyoid membrane The position of the polyethylene tubing is carefully checked, and if found to be satisfactory, the silver wires are anchored externally by appropriate metal buttons and lead shot The polyethylene tube is left in place for a period of six weeks to two months There is no interference with respiration, tracheotomy is not necessary and as a rule the tube is well tolerated The patient is examined at intervals, and when epithelization has occurred, the larynx is again exposed with the aid of the suspension laryngoscope The tube is grasped from within the larynx, the wires are cut externally and the tube is removed The web is still present and attached to both vocal cords, but there is now an epithelized opening at the anterior commissure The web is excised along the edges of the cord, care being taken not to injure the true cord There is no raw surface at the anterior commissure, hence, the web does not recur

We are indebted to Justo Alonso for first describing use of the Riverden needle in the insertion of an acrylic mold in the larynx for correction of stenosis and webs Since then we have used the polyethylene tube with excellent results in a limited number of cases with either thin or moderately thick web formation between the cords

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

The 34th convention of the Central Illinois Society of Ophthalmology and Otolaryngology will be held at the Illinois Hotel in Bloomington, Ill, on April 22nd, 23rd and 24th

Our guest speakers are Dr John W Henderson, Rochester, Minn, Ophthalmology, Dr Z Z Godlowski, Chicago, Ill, Otolaryngology Member participants are Dr. W A McNichols, Dixon, Ill, Ophthalmology, Dr George H Woodruff, Joliet, Ill, Otolaryngology

Prof Arthur W Watterson, of the Geography Department of the Illinois State Normal School, Bloomington, Ill, will be the speaker at the Saturday evening banquet

For further information please address Dr C L Pannabecker, Secretary-Treasurer, 434 Jefferson Bldg, Peoria, Ill

SEQUELAE OF TRACHEOBRONCHIAL ASPIRATION

CARL J BRIDGE, M D ,

Gardner, Mass

A small series of cases is presented for demonstration of short and long range pathologic changes associated with suctioning of the tracheobronchial tree through a tracheotomy stoma

Tracheotomy is performed if the upper respiratory tract is a primary source of obstruction, if the patient is unable to aid at all in removal of secretions and exudates, or if suction will be required over a long period of time It is obvious that the extent to which suction is required is dependent upon the patient's ability to cough When the cough reflex is depressed, the introduction of a catheter or bronchoscopy tube aids in its stimulation, and secretions in the lower portions of the respiratory tract can be expelled, providing there is some air in the alveoli distal to them

Blind suction of the trachea and primary bronchi is thought to be almost as effective as direct vision suctioning through a bronchoscope, when liquid secretions or exudates are involved Blind suctioning is also believed to be relatively atraumatic when done with a No 16 French soft rubber catheter with a hole in the end and one in the side, just proximal to the end, under a negative pressure of from five to ten pounds If less than five pounds, it is ineffective, if more than ten pounds, the catheter collapses, thus preventing the bronchial and tracheal mucosa from becoming unduly traumatized At times a curved tipped catheter with a hole in the end has been used, in the belief that it aids in the removal of material from secondary bronchi When there are indications that blind suction is inadequate for removal of material from the tracheobronchial tree, we resort to direct vision bronchoscopy

A series of 18 cases of poliomyelitis, from the Boston epidemic of 1955, treated at the Massachusetts General Hospital,

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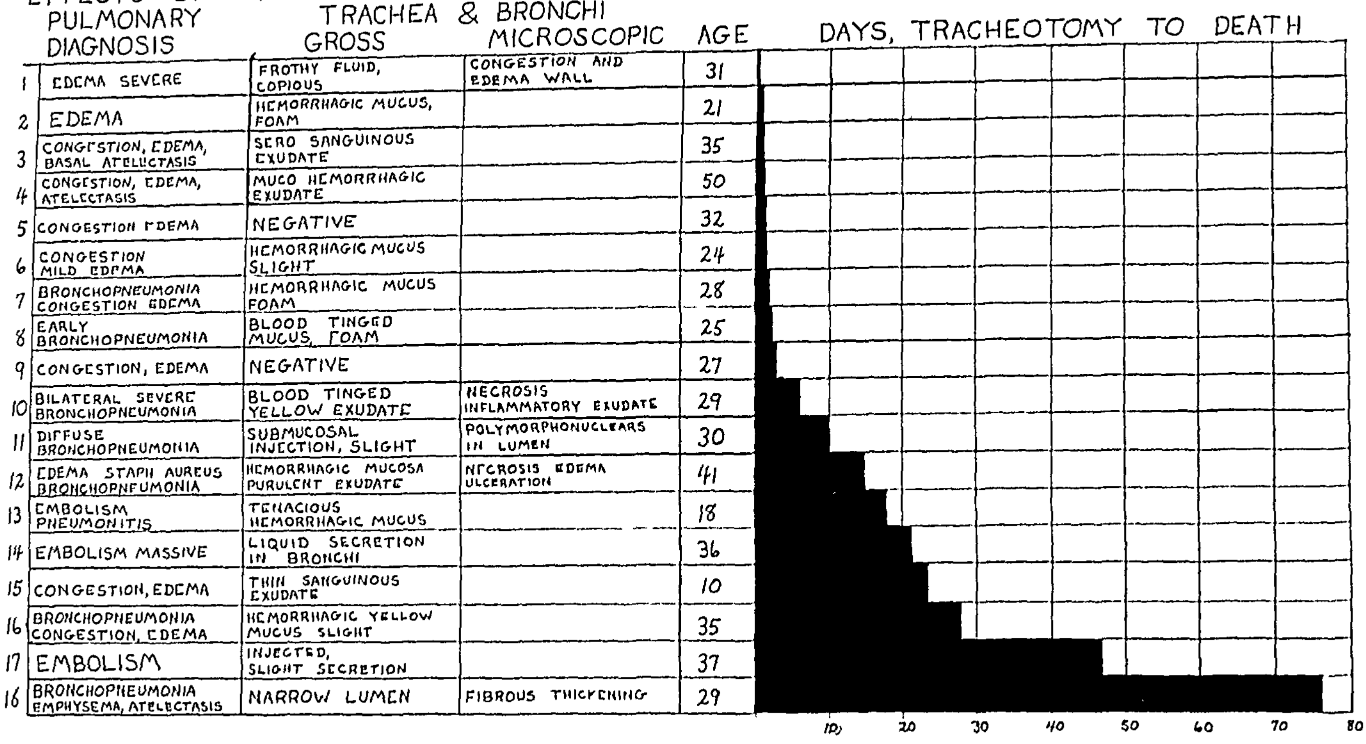
Editor s Note This manuscript received in The Laryngoscope Office and accepted for publication Jan 20 1960

FIGURE 1

EFFECTS OF TRACHEOBRONCHIAL ASPIRATION IN FATAL POLIOMYELITIS CASES



	PULMONARY DIAGNOSIS	TRACHEA & BRONCHI GROSS	TRACHEA & BRONCHI MICROSCOPIC	AGE
1	EDEMA SEVERE	FROTHY FLUID, COPIOUS	CONGESTION AND EDEMA WALL	31
2	EDEMA	HEMORRHAGIC MUCUS, FOAM		21
3	CONGESTION, EDEMA, BASAL ATELECTASIS	SERO SANGUINOUS EXUDATE		35
4	CONGESTION, EDEMA, ATELECTASIS	MUCO HEMORRHAGIC EXUDATE		50
5	CONGESTION EDEMA	NEGATIVE		32
6	CONGESTION MILD EDEMA	HEMORRHAGIC MUCUS SLIGHT		24
7	BRONCHOPNEUMONIA CONGESTION EDEMA	HEMORRHAGIC MUCUS FOAM		28
8	EARLY BRONCHOPNEUMONIA	BLOOD TINGED MUCUS, FOAM		25
9	CONGESTION, EDEMA	NEGATIVE		27
10	BILATERAL SEVERE BRONCHOPNEUMONIA	BLOOD TINGED YELLOW EXUDATE	NECROSIS INFLAMMATORY EXUDATE	29
11	DIFFUSE BRONCHOPNEUMONIA	SUBMUCOSAL INJECTION, SLIGHT	POLYMORPHONUCLEARS IN LUMEN	30
12	EDEMA STAPH AUREUS BRONCHOPNEUMONIA	HEMORRHAGIC MUCOSA PURULENT EXUDATE	NECROSIS EDEMA ULCERATION	41
13	EMBOLISM PNEUMONITIS	TENACIOUS HEMORRHAGIC MUCUS		18
14	EMBOLISM MASSIVE	LIQUID SECRETION IN BRONCHI		36
15	CONGESTION, EDEMA	THIN SANGUINOUS EXUDATE		10
16	BRONCHOPNEUMONIA CONGESTION, EDEMA	HEMORRHAGIC YELLOW MUCUS SLIGHT		35
17	EMBOLISM	INJECTED, SLIGHT SECRETION		37
16	BRONCHOPNEUMONIA EMPHYSEMA, ATELECTASIS	NARROW LUMEN	FIBROUS THICKENING	29

is reviewed, and the pertinent respiratory findings are charted on Fig 1 Apparently in all the cases, except No 18, where death was due to chronic tracheal and bronchial obstruction, and two others, where death was due to pulmonary embolism, death was due to circulatory collapse of central origin All the cases listed had a tracheotomy, and they underwent numerous instances of blind suctioning with soft rubber catheters,

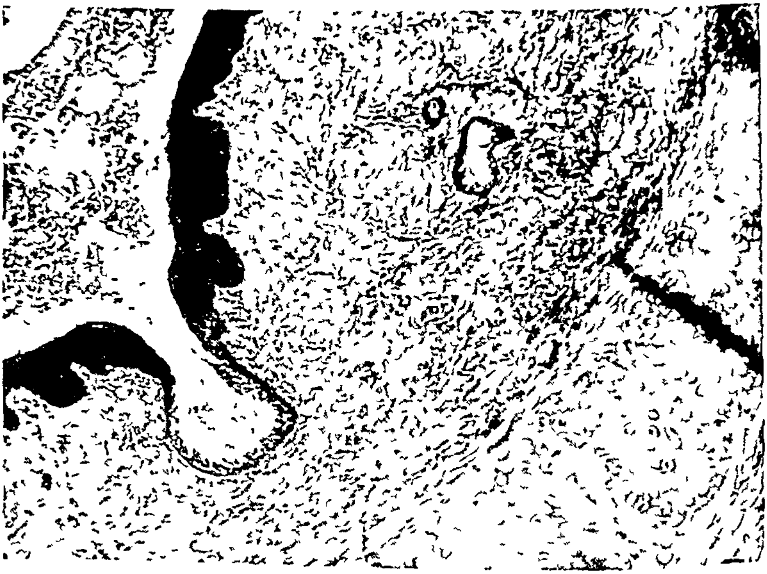


Fig 2 Inferior extent of cicatricial scar in Case 18 showing debris in the lumen of the trachea stratified squamous epithelium, and underlying scar tissue

as well as bronchoscopy, however, it is believed that these procedures were quite frequent and that the incidence of suctioning procedures was roughly proportional to the time span involved It is also assumed that the pathologic changes in the tracheobronchial tree are due principally to direct trauma, but they may be contributed to by pulmonary complications of respiratory paralysis or to pulmonary embolism where the latter has been shown to occur

Case No 18 on the above chart, which is of great interest in this study, is reviewed briefly

YKE (MGH No 904807), a 29 year old housewife was admitted on September 5, 1955, with a diagnosis of questionable poliomyelitis, following a four day history of sore throat, which had also been present transiently in other members of the family, stiff neck of three days duration with back pain and headache appearing shortly after the stiff neck Her only significant physical findings were marked stiffness of the neck and bilateral limitation of straight leg raising Her temperature was 101 0° F Spinal fluid was under initial pressure of 114 mm of water, and it contained 260 wbc/mm^3 80 per cent of which were lymphocytes and 20 per cent polymorphonuclears The next day she developed diplopia and the following day weakness of voice, dyspnea and weakness of the face and all extremities She was then placed in a respirator On September 9th she had complete quadriplegia Tracheotomy was performed On that day her temperature was 103° F

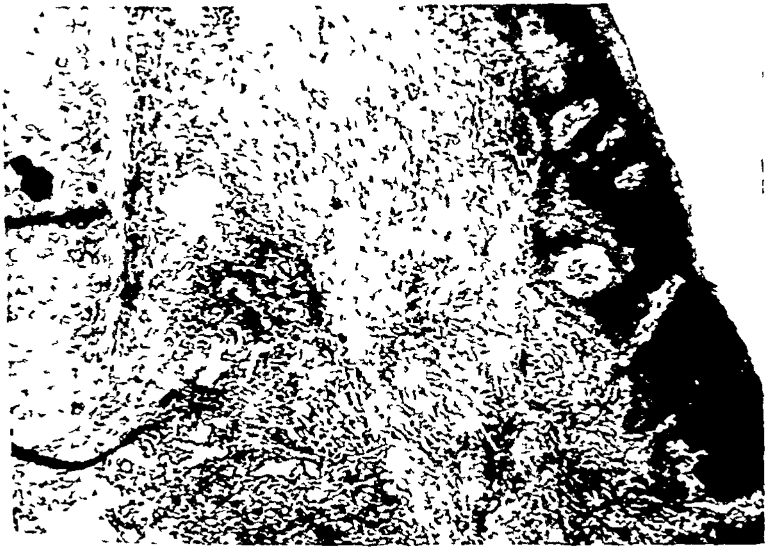


Fig 3 Central portion of cicitricial scar in Case 18 showing thick stratified squamous epithelium and underlying scar tissue which contains some inflammatory cells Tracheal glandular tissue is also seen

During the next 15 days the patient had several episodes of cyanosis, necessitating repeated catheter suctioning and a bronchoscopy, the latter for removal of dried mucous plugs Fever persisted despite multiple antibiotic therapy On September 20th tidal volume was very low, 100 cc Bronchoscopy revealed crusted blood narrowing all bronchi, it could not be detached without producing bleeding points Tidal volume remained low, but it could be brought to 250 cc at times by repeated bronchoscopy and positive and negative pressure breathing Portable chest roentgenograms were negative She became very disoriented at times On September 24th it was felt that she was improving Bronchoscopy revealed no narrowing, but a grayish mucosa was seen During the next month her course remained intermittently febrile This was thought due to Staphylococcus aureus, alpha hemolytic Streptococcus and Hemophilus influenzae infection, these organisms having been cultured from material aspirated by bronchoscopy.

The patient also underwent several episodes of hypotension and sinus tachycardia. Blind aspirations and bronchoscopies were still frequent. On November 7th, on bronchoscopy it was noted that two inches below the tracheotomy there was a concentric narrowing, where the mucosa was injected and edematous, and it bled easily. On November 19th it was stated that she had been doing well. Although paralyzed completely below the neck, she was placed for a few minutes each day on a rocking bed. On November 22nd she developed cyanosis, which required very frequent suctioning and bag breathing. It was stated that the bronchi would not admit a catheter. Tidal volume was kept at 250 cc., however, she became unresponsive and died ten hours later.

The post mortem report, in part, is as follows:

The tracheostomy opening is located in the upper end of the trachea and measures 1 cm. in diameter. The opening is situated above the thyroid isthmus. The proximal 5 cm. below the tracheotomy orifice is unremarkable. Beginning 5 cm. below the tracheotomy orifice an almost abrupt narrowing of the lumen is noted. This narrowing extends for a distance of 4.5 cm. down to the carina. The narrowed portion measures 0.8 cm. in circumference at the narrowest point. The wall of the trachea in this region is thickened and fibrotic, with a purplish red discoloration. The mucosal surface of the narrowed portion is covered by a considerable amount of fibrinous exudate, while the lumen is almost completely occluded by a mucoid tenacious and sometimes purulent material. The bifurcation is also slightly narrowed for a distance of 1.5 cm. on each side, gradually merging into a normal bronchus on each side. The wall is similarly thickened, and the mucosal surface is covered by a fibrinous exudate. Beyond 1.5 cm. from the bifurcation the lumen appears normal; however, all the ramifications of the bronchial tree in either side reveal a large amount of mucoid and fibrinous exudate within the lumen, almost completely filling these structures. This exudate extends into the bronchi and bronchioles and can be expressed easily at the terminal portions of the lungs.

Microscopically, there is thickening of the mucosa by dense fibrous tissue infiltrated by chronic inflammatory cells. Squamous metaplasia of the epithelium and mild vascularization of the tunica propria are present. The tracheal glands show periglandular inflammatory reaction and dilatation of the ducts, or even of the glands themselves, and are filled by mucinous material. Foci of ulceration covered by fibrin are noted on the epithelial surface. The mucosal thickening is worse in the distal than in the proximal trachea. The fibrosis sometimes extends beyond the cartilage rings. The lumen contains cellular debris, mucus, inflammatory cells, red cells, and fibrin.

The prosector commented that this patient died of upper respiratory tract obstruction resulting from a complication, probably brought about by frequent bronchoscopic examination and chronic infection. The cicatricial contraction produced further difficuty in the aspiration of mucus, initiating a vicious cycle. Chronic irritation further down the bronchial tree has apparently produced bronchiolitis with subsequent organization of the exudate. This process was apparently not diffuse, so that it could not have contributed significantly to the obstruction

ANATOMIC DIAGNOSES

Poliomyelitis, spinal

Tracheitis and bronchitis chronic with tracheobronchial stenosis and obstruction

Bronchiolitis fibrosa obliterans, focal

Bronchopneumonia, mild

Pulmonary atelectasis superficial, bilateral

Pulmonary emphysema, focal mild

Phlebothrombosis old, right femoral vein

Hydrothorax, left (50 ml)

Cortical atrophy adrenal secondary to hydrocortisone therapy

Focal necrosis of liver, old with bile pigmentation of Kupffer Cells (Thorazine therapy)

Operation Appendectomy, ten years

Caesarean sections, three-and-a-half and two years

Tracheostomy two-and-a-half months

Of those patients who survived severe poliomyelitis it is not known that prolonged suction through a tracheotomy caused any permanent disability associated with injury to the tracheobronchial tree

A six-year-old boy, a poliomyelitis patient had a tracheotomy tube in place for 114 days During the five months after closure of the tracheotomy wound, he attained, gradually, a nearly normal existence for a person of his age including participation in competitive games, however, he died at home very suddenly, presumably because of aspiration of a pea, due to weak throat muscles Unfortunately a post mortem examination was not performed and Hemophylus influenzae infection, these organisms having been cultured from material aspirated by bronchoscopy

The patient also underwent several episodes of hypotension and sinus tachycardia Blind aspirations and bronchoscopies were still frequent On November 7th, on bronchoscopy it was noted that two inches below the tracheotomy there was a concentric narrowing, where the mucosa was injected and edematous, and it bled easily On November 19th it was stated that she had been doing well Although paralyzed completely below the neck, she was placed for a few minutes each day on a rocking bed On November 22nd she developed cyanosis, which required very frequent suctioning and bag breathing It was stated that the bronchi would not admit a catheter Tidal volume was kept at 250 cc , however, she became unresponsive and died ten hours later

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Gardner State Hospital

THE GOULD AWARD

The International Committee for the Gould Award announces the selection of the recipient for the year 1959—Professor Joel J Pressman, M D, University of California at Los Angeles, for his anatomic and radio-isotope studies of laryngeal compartments

The Gould Award is presented annually for basic research in laryngology to encourage and reward fundamental investigations in this field The International Committee for the Gould Award for the year 1960 includes Sir Victor Negus, London, England, Professor Cotoji Satta, M D Tokyo, Japan, Professor Dr Luzius Ruedi, Zurich, Switzerland, and Hans von Leden, M D, Medical Director, The William and Harriet Gould Foundation, 30 North Michigan Avenue, Chicago 2, Ill, U S A

The Gould Award consists of a plaque and an honorarium of $300 00

DISCUSSION

It should be emphasized that the last case on the chart is the only one which showed scar tissue grossly

It is also noteworthy that in several cases at necropsy, little material was found in the air passages, this fact attesting to the effectiveness of suctioning procedures Under direct visualization, through tracheal windows, in five fresh cadavers, catheter suctioning was done under the usual pressures The ability of the catheter to remove all visible material in the vicinity was striking, and it was noted also that the mucosa of the air passages was not drawn into the holes of the catheter

SUMMARY AND CONCLUSIONS

1 Suctioning procedures in the tracheobronchial tree cause acute and chronic inflammatory reactions in the trachea and primary bronchi, as seen in a series of 16 of 18 fatal poliomyelitis cases

2 The degree of traumatic effect of suctioning does not seem to be clearly directly proportional to the length of time over which frequently performed suctioning is carried out

3 One fatality in this series can be attributed to the mechanical effect of frequent suctioning over a very long period of time, a cicatricial tracheal scar

4 In rehabilitated poliomyelitis cases, there is no commonly known morbidity due to prolonged suctioning of the tracheobronchial tree

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or aqueous Dionosil® (50 per cent propyliodone, n-propyl ester of 3 5 diiodo-4-pyridone-n-acetic acid, Glaxco Laboratories Ltd) Negative pressure in the tympanum is created by drawing the air from it with a Bruening Zeiss pneumatic otoscope, the speculum of which fits air tight into the external auditory canal Air drawn from the tympanum bubbles through the fluid in the external auditory canal The procedure is continued until bubbles cease to appear The fluid in the external auditory canal runs into the middle ear because of the negative pressure A cotton plug is inserted into the canal Roentgenography is carried out immediately, in the positions commonly used for temporal bone study These include the Law position with the tube tilted 15 and 35 degrees, the submento-vertex and the Towne positions, the last named made in stereo

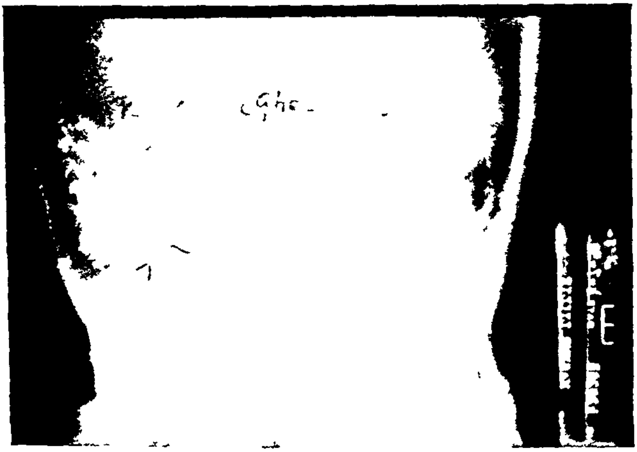

Fig 1

STATISTICS

Ten patients were studied The ages ranged from five to 68 years Three persons were white, seven colored, six were male, four female Some middle ears were free of discharge at the time of examination, whereas others revealed varying degrees of mucosal disease and ossicular destruction Two

TEMPOROGRAM*

LESTER A BROWN, M D ,
and
DAVID T SMILEY, M D ,
(By Invitation),
Atlanta, Ga

Tympanotubal lavage is used in the conservative treatment of middle ear infections especially when the tympanic membrane perforation is central Medications used include broad spectrum antibiotics, steroid preparations and other medicaments in powder, liquid and semi-solid states

In order to determine to what extent the medication was reaching the middle ear and connecting areas, several patients were studied radiographically after using a radiopaque dye for tympanotubal lavage

SELECTION OF CASES

The patients selected had perforations of the tympanic membranes, but otherwise were selected at random with two exceptions *a* where there had been no aural discharge for a long period of time, and *b* young children whose lack of cooperation prevented obtaining satisfactory X-ray films

TECHNIQUE

The external auditory canal is carefully cleansed The patient is instructed to lean his head toward the shoulder opposite the involved side, and to incline the body likewise until the vertical axis of the head is nearly horizontal The face is turned upward and the head tilted backward The upper external auditory canal is half filled with Lipiodol® (iodized oil—28 per cent iodine, Andre Guerbet and Company),

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*Read at the meeting of the Southern Section of the American Laryngological Rhinological and Otological Society Inc New Orleans La Jan 15 1960

Editor s Note This manuscript received in The Laryngoscope Office and accepted for publication Jan 22 1960

2 The location of the perforation

3 The presence and extent of cholesteatoma or granulation tissue

4 The time spent in performing the procedure

5 The position of the patient's head during the instillation

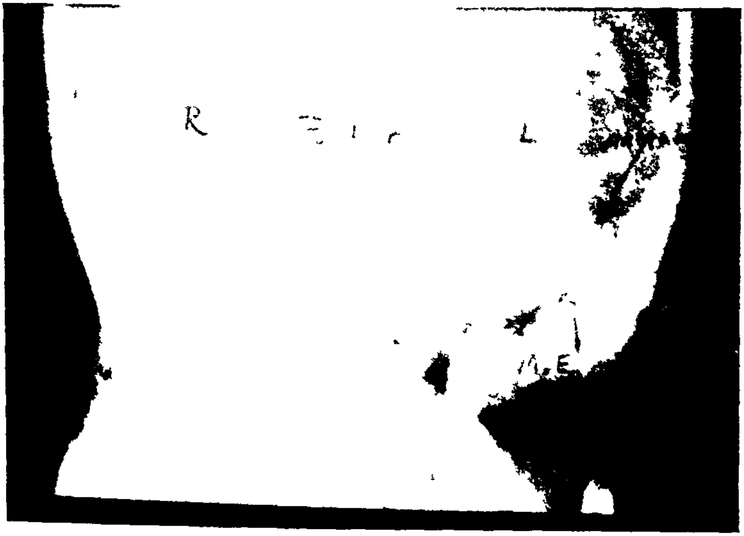

Fig 2

No indication of residual dye was seen in the roentgenograms of three patients, which were repeated ten days after the original instillation

REVIEW OF LITERATURE

Dye contrast roentgenography of the air spaces of the temporal bone is not brand new In 1928 Ruttin[1] was able to demonstrate the attic and mastoid antrum using 40 per cent *Iodopin* as contrast media The following year, in this country, Mandelbaum[2] published his findings He had been using *Lipiodol* for local treatment of chronic middle ear disease and found unexpectedly that the dye was useful as a diagnostic aid in outlining soft tissue masses Unger,[3] in New York, emears contained cholesteatoma, clinically Two perforations were of the attic type, the others central The quantity of dye required in each case was proportional to the extent of mastoid pneumatization and averaged 1 6 cc The time required for satisfactory instillation ranged from two minutes to eight minutes There was no difference between the oily (Lipiodol) and aqueous (Dionosil) contrast media in ease of instillation or ability to fill the air spaces, if the Dionosil was diluted by adding 3 cc of water to the 20 cc vial Table I shows the distribution of the dye

TABLE I

Type of Perforation	No of Cases	Dye Present in				
		Tympanum	Epitympanum	Eustach Tube	Antrum	(Mastoid) Cel
Central	8	8	7	6	6	
Attic	2	2	2	1	2	

*Four others were acellular
†One other was acellular

COMMENT

Scattered incomplete mastoid filling occurred in the first case, presumably because of insufficient time spent in the procedure, and an unusually long delay in obtaining the X-ray films, however, when the procedure was correctly performed, there was fairly complete filling of the mastoid cells with the dye, whether the cells were many or few

In each case where the perforation involved Shrapnell's membrane, and there was cholesteatoma present, the mastoid process filled poorly

The purpose of the study originally was to determine to what extent the medication reached the diseased areas It becomes obvious that there are ramifications For example, this procedure may be valuable in determining to what extent granulation or other soft tissue fills the aerated cavities of the temporal bone

It would seem that the factors influencing the effectiveness of tympanotubal lavage include

1 The size of the perforation

In Memoriam

FELIX ROBERT NAGER

1877 - 1959

ployed the technique of tympanotubal lavage to differentiate between pneumatic and cancellous bone, noting that the dye filled the former but not the latter He used living subjects and cadaver specimens In 1951 Faccini[4] in Italy, demonstrated roentgenographically the normal anatomy of the middle ear and adnexa by instilling *Joduron B* through a Eustachian catheter and the auditory tube Shortly thereafter, Papshitskii,[5] in Russia, demonstrated cholesteatoma by means of iodine, barium, and bismuth compounds injected through an attic cannula Hungary was represented in an article by Bollobás[6] in which he reported filling of the middle ear system with contrast media

SUMMARY

1 It is possible to apply medication directly to the diseased areas of the aerated parts of the temporal bone through a perforation in the tympanic membrane

2 It is possible to demonstrate the distribution of radiopaque dye by roentgenography

3 The presence of filling defects may become diagnostically significant

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4 FACCINI, M Visualizzazione con Mezzi di Contrasto Opachi della Tuba Auditiva e delle Cavitá dell Orecchio Medio nell'uomo *Radiol Med, Tor*, 37 11 908 916, Nov 1951

5 PAPSHITSKII, IU A Primenenie Kontrastnykyh Veshchestv pri Rentgenologicheskom Issledovanii Visochnykh Kostei *Vest Otorinolarygol* 16 6 43 46, Nov-Dec 1954

6 BOLLOBAS, B Das Auffullen des Mittelohrhohlensystems mit Rontgen Kontrastmitteln *Fortsch Rontgenstrahl*, 83 3 323 329, Sept, 1955

developmental anomalies of the ear with or without association with facial-cranial and skeletal malformations, and on etiologies of forms of deafness Many of these studies have enduring significance

Professor Nager founded a histopathological laboratory which now houses a unique collection of temporal bone pathology Visitors from the entire world were welcomed here, and in this laboratory many scientists were introduced to the morbid anatomy of the ear A large number of them made important contributions during their stay in Zurich or later as Directors or Departments in the United States and elsewhere This international exchange of ideas and close team work on scientific problems of mutual interest started many wonderful friendships, created close bonds between Zurich and many centers of the Anglo-Saxon world and in its own way contributed much to the resumption of friendly cooperations after the disruptive effects of two world wars

Following the tradition of his masters, Bezold and Siebenmann, Felix R Nager created his own well-known school of otolaryngology which produced excellent chairmen of departments, teachers, scientists and numerous outstanding specialists throughout his country, and elsewhere

At the time of the 25th anniversary of his clinic and also on his 70th birthday, pupils, colleagues, and friends throughout the otolaryngic world honored Professor Nager with a Festschrift of Scientific papers

His early recognition of the important scientific activity in the New World promoted his journey to America in the mid-twenties and his return visit in 1937 He often said that these trips added much to the knowledge of the visitor They gave him some of his dearest friends and brought him a sincere admiration for American otolaryngology

He was elected to numerous honorary memberships In the United States he was an Honorary Member of the American Academy of Ophthalmology and Otolaryngology (1925), of the American Otological Society, Inc (1937), of the American Laryngological Society (1937) and of the Harvey Society of

Felix Robert Nagel was born on December 9, 1877, in Lucerne, Switzerland. His father, Dr. Gustav Nagel, was a well-known Otolaryngologist, devoted to the welfare and rehabilitation of the hard of hearing. Felix R. Nagel attended the public schools in that city and later went to the Universities of Basle, Geneva, Heidelberg and Zurich, where he was given his Medical Doctor degree in 1904.

He began his post-graduate training in Pathology, as an assistant to Professor P. Ernst in Zurich. Subsequently, he worked with Hofrat Professor Frederick Bezold in Munich and from 1904-1906 with Professor Frederick Siebenmann in Basle, the "founder" of Otolaryngology in Switzerland.

He began private practice in Zurich and in 1908 started a personally conducted clinic in the evenings for indigent patients. In 1915 he became Professor of Otolaryngology and in 1919 was appointed Director and Chairman of the newly created independent Ear, Nose and Throat Clinic and Ear, Nose and Throat Out Patient Department of the Zurich University School of Medicine. Until then, Ear, Nose and Throat Departments in Switzerland existed only in Basle, Berne and Lausanne. From 1940-1942 he was Dean of the Medical School, and in 1948 he was retired as Emeritus Professor.

Professor Nagel made great and extensive contributions to the literature of otolaryngology. Almost 200 scientific papers, in several languages, bear evidence of his profound interest in every aspect of otolaryngology and in every problem that arose during his clinical activity. Many articles are concerned with clinical and anatomical pathology of the nose, paranasal sinuses, nasopharynx, throat, larynx, trachea, bronchial tree and the esophagus. His surgical interests were manifold, thus he became a pioneer and expert in the paranasal approach to intrasellar tumors of the hypophysis, and bronchoesophagology and laryngeal surgery were greatly developed. His principal interest, however, was the morbid anatomy of the temporal bone. Numerous are the monographic publications on bone diseases involving the otic capsule, on inflammatory disease, specific and non-specific, as they affect the ear, on

ROYAL SOCIETY OF MEDICINE

The Sections of Laryngology and Otology of the Royal Society of Medicine, London, will be meeting on May 6 and July 14-16, 1960. The programs follow.

May 6 Meeting

Otology 10 A.M.—Discussion on the Effects of Noise Upon Hearing
  Dr. A. Glorig, Los Angeles, Calif.

Laryngology 2.30 P.M.—"Dysphagia in Adults (non-malignant), Its Diagnosis and Management."
  Mr. H. D. Brown Kelly
  "Management of Chemical Burns of the Oesophagus."
  Professor Truls Leegaard (Oslo)
  Mr. Norman Barrett will open the discussion.

July Meeting

The Sections will hold a combined meeting at Oxford and the program follows.

15th, 9.30 A.M.—"The Innervation of the Inner Ear."
  Professor Arslan (Padua)
  Also papers by members of the Otological Research Unit, Medical Research Council, Queen Square, London.
  2.30 P.M.—Visit to Department of Plastic Surgery, Churchill Hospital.

16th, 9.30 A.M.—The Management of Frontal Sinusitis and its Complications."
  Mr. Desmond Dawes
  "The Radiography of the Lower Lateral Food Channels."
  Dr. F. H. Kemp and Dr. G. M. Ardran

Members of the American Laryngological Association, the American Triological Society and the American Otological Society who will be in England at these times will be welcome. If they wish to come they should write to E. H. Miles Foxen, F.R.C.S., Secretary for Laryngology, Charles Gledhill, M.B.E., F.R.C.S., Secretary for Otology.

New York (1925) In 1939 he delivered the Sir Felix Semon Lecture at the University of London He became an Honorary member of the Otological and Laryngological sections of the Royal Society and of the Royal Society of Medicine In 1949 he was elected an Honorary Fellow of the Royal College of Surgeons of England He was one of the founders of the Collegium Oto-Rhino-Laryngologicum

On April 15, 1948, Professor Nager retired as chairman of the department After retirement, he continued a limited private practice, and devoted much time to the social and personal welfare of the hard of hearing and to his research A progressive illness finally wrested his beloved activities from his hands

His last address to the students and faculty was at the end of his 79th semester and had the title "After Forty Years" In this address he cast a retrospective glance at the development of our specialty and he outlined the problems that will challenge otolaryngology in the future In a philosophical manner he discussed dignity, virtues and ethics of the physician and reveals his personal philosophy or way of life to the coming medical generation from which he is departing His valedictory address will always be remembered by those who were in the audience

Professor Nager died at home on September 7, 1959, in his 82nd year He is survived by his widow, the former Emily Reinhart, by three daughters and three sons

External Otitis Specific Therapy Based on Accurate Diagnosis

Edley H Jones, Vicksburg, Miss

Address of Guest of Honor

Louis H Clerf, St Petersburg 2, Fla

Nasal Tip Surgery

Sam H Sanders, Memphis 4, Tenn

Address of Section Chairman

Paul H Holinger, Chicago 11, Ill

Problems and Complications in Head and Neck Surgery

George A Sisson, Syracuse 10, N Y , and Norman E Johnson

The Long-Term Care of the Laryngectomized Patient

George F Reed, Massachusetts Eye and Ear Infirmary, Boston, Mass

Friday, June 17, 1960

Cystadenoma of the Parotid and of the Larynx

Arthur J Kuhn, Hammond, Ind

Effect of Premarin on the Ground Substance

Maurice Schiff, Capt (MC) USN, U S Naval Hospital, Oak Knoll, Calif

Cervico-Facial Congenital Anomalies

Richard T Farrior, Farrior Clinic, Tampa 6, Fla

Incidence of Hearing Impairment in a Medical Center Population

Edmund P Fowler, Jr , M D , and Thomas H Fay, Ph D , New York 32, N Y

Otometric Operations

George D Albers, Grand Rapids 3, Mich

PROGRAM FOR AMERICAN MEDICAL ASSOCIATION SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY, 109th ANNUAL MEETING

June 15-17, 1960, Miami Beach, Fla

Wednesday, June 15, 1960

Joint Session of the Section on Laryngology, Otology and Rhinology, Section on the Diseases of the Chest, and Section on Radiology

1 Panel Discussion on Tumors of the Trachea and Bronchi

Moderator Dr Paul H Holinger, Chicago 11, Ill

Endoscopist Dr Herbert W Schmidt, Rochester, Minn

Radiologists Dr Barton R Young, Philadelphia, Pa,
Dr Wendell G Scott, St Louis, Mo

Surgeons Dr Robert J Jensik, Chicago 4, Ill,
Dr Robert W Jamplis, Palo Alto, Calif

2 Panel on Disseminated Diseases of the Chest

Moderator Dr Harold O Peterson, Minneapolis, Minn

Internist Dr James A Wier, Denver, Colo

Chest Physician Dr David Radner, Chicago, Ill

Endoscopist Dr Walter H Maloney, Cleveland 6, O

Radiologists Dr Eugene F Van Epps, Iowa City, Ia,
Dr Clyde A Stevenson, Spokane, Wash

Surgeons Dr Donald L Paulson, Dallas, Tex,
Dr William Tuttle, Detroit, Mich

Thursday, June 16, 1960

Audiology Its Progress and Problems

Kenneth O Johnson, Ph D, Executive Secretary, American Speech and Hearing Association, Washington 6, D C, (By Invitation)

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES

(Secretaries of the various societies are requested to keep this information up to date)

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President Dr Erling W Hansen, 90 So Ninth St, Minneapolis, Minn
Executive Secretary Dr William L Benedict, Mayo Clinic, Rochester, Minn
Meeting Palmer House, Chicago, Ill, October, 1960

AMERICAN ASSOCIATION FOR CLEFT PALATE REHABILITATION

President Dr J J Longacre, 1503 Carew Tower Cincinnati, O
President-Elect Dr D C Samuel Pruzansky, D D S, 840 So Wood St, Chicago, Ill
Secretary-Treasurer Dr Spriestersbach, Ph D, Department of Otolaryngology, University Hospital Iowa City, Ia
Meeting Palace Hotel, Denver, Colo, May 12 14, 1960

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Secretary Dr Dean M Lierle, University Hospital Iowa City, Ia
Meeting Palmer House Chicago, Ill, October, 1960

AMERICAN BRONCHO ESOPHAGOLOGICAL ASSOCIATION

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THE
LARYNGOSCOPE.

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Workshop on the Inner Ear.

THE HISTOCHEMISTRY OF THE ENZYMES OF OXYGEN METABOLISM IN THE INNER EAR*†

K H VOSTEEN, M D,
Hamburg, Germany

One of the most interesting problems in the biology of the cochlea is the study of chemical sources of energy From the transformation of sound in the cochlea, it is known that the energy which is transferred to the nervous system is greater than that received from the cochlea The only possibility for this energy increase is a chemical metabolic process Almost all metabolic reactions receive their energy from the oxygen metabolism (respiration of the cell) This metabolism depends again upon the activity of its enzyme systems

Using histochemical techniques we can investigate the respiratory function of a cell and so demonstrate the sites of energy production, therefore, histochemistry can supply valuable insight into the transformation of energy in the cochlea

Cell respiration is a chain reaction, during which individual organic substrates loose their hydrogen atom by specific enzymatic processes The hydrogen is then carried by the coenzymes DPN and TPN, with the assistance of diaphorase, to the cytochromes, or is directly delivered by means of succinodehydrogenase By reoxydation of the cytochromes and

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*Read at the meeting of the Middle Section American Laryngological Rhinological and Otological Society Inc Chicago Ill Jan 18 1960

†From the Department of Otolaryngology University of Hamburg

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of dehydrating enzymes to the colored formazan. By using succinic acid as a substrate and by elimination of the other hydrogen donors this test can be made specific. The blue-red formazan crystals can also be found in areas where great amounts of succinodehydrogenase are concentrated, for example in the stria vascularis. A specific control can be achieved by pre-treatment of the slides with sodium malonate.

The method for the demonstration of diaphorase uses the same principles. A tetrazolium salt again serves as an indicator. The use of the coenzymes DPN and TPN in the medium makes this test specific. According to Farber, Steinberg and Dunlap[2] other substrates have been added to get more pronounced effects.

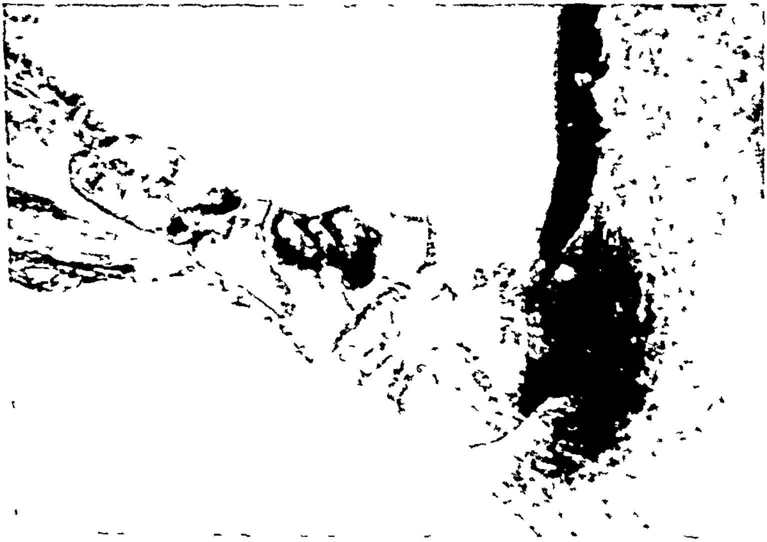

Fig. 2. Ductus cochlearis: succinodehydrogenase.

The respiratory enzymes are very sensitive and, therefore, the histochemical reactions can be carried out only with fresh and unfixed material. Immediately after death of the animal—we have used guinea pigs exclusively—one must separate parts of the ductus cochlearis by microdissection and cut frozen sections.[19]

with the assistance of the cytochromoxydase the hydrogen can finally be eliminated as water

The most important respiratory enzymes, namely the cytochromoxydase, the succinodehydrogenase and the diaphorases can be histochemically demonstrated

The method for cytochromoxydase is one of the oldest histochemical procedures More than 40 years ago Graeff showed

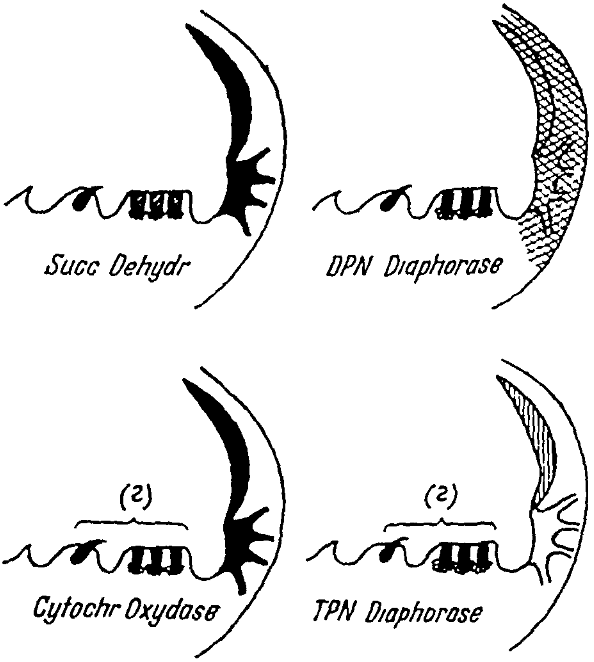

Fig 1 Distribution of the enzymes of oxygen metabolism

that a mixture of x-naphthol and dimethylparaphenylendiamin can be oxydized by the oxydase to indophenol-blue This indophenol-blue appears in histochemical preparations only where considerable amounts of oxydase are localized By blocking with potassium cyanide we can specifically test for cytochromoxydase

The histochemical procedure for succinodehydrogenase was worked out by Rutenburg and Seligmann[12] It is based on the fact that a colorless tetrazolium salt is reduced in the presence

A very strong accumulation is to be found in the hair cells also, but in the stria vascularis only the DPN-diaphorase is especially active, and the areas behind the sulcus externus show no strong staining reaction

All these enzymes are links of the same chain reaction and therefore, it is surprising that they are not concentrated in the same location The dehydrogenase and oxydase are always found in the same spot In other organs, for example the liver[13] and the kidney,[5] it has been demonstrated that the diaphorase shows a different pattern of distribution An explanation for this phenomenon can not be offered at this time Comparative histochemical and biochemical investigations have shown that the intensity of respiratory processes always runs parallel with the activity of succinodehydrogenase and cytochromoxydase

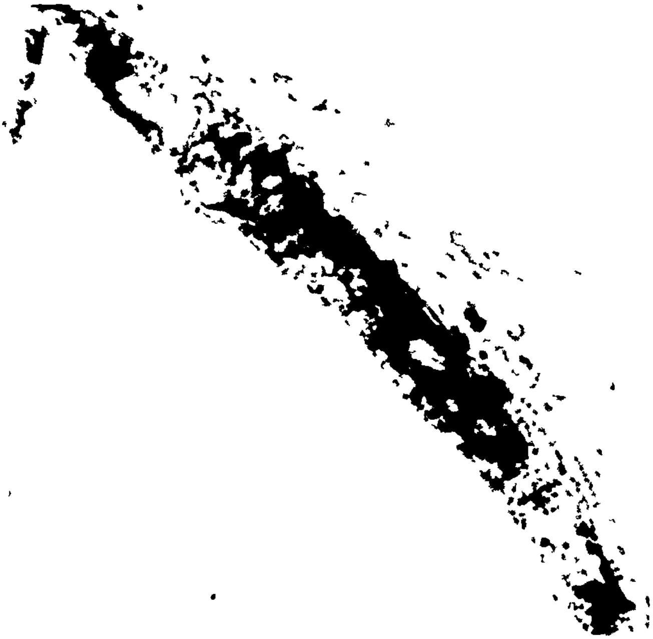

Fig 4 Stria vascularis TPN-diaphorase

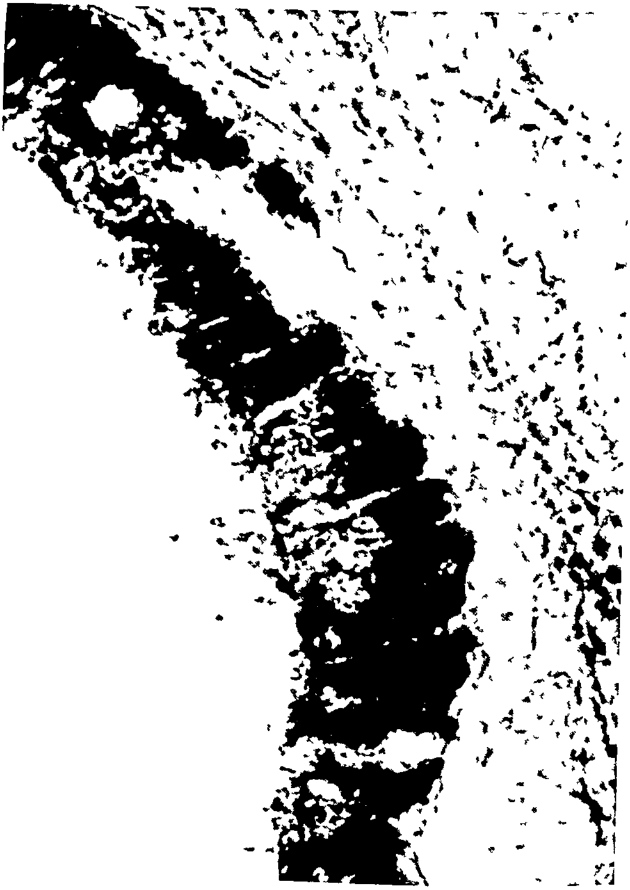

Fig 3 Stria vascularis succinodehydrogenase

The distribution of the enzymes can be recognized in such a frozen section through the ductus cochlearis The succinodehydrogenase shows, for example, its strongest concentration in the stria vascularis and in a part of the ligamentum spirale behind the sulcus externus and in the hair cells

The typical patterns of distribution of the other respiratory enzymes are shown in a diagram Cytochrome oxydase and succinodehydrogenase are concentrated in the same place the stria vascularis, behind the sulcus externus and the hair cells

The diaphorases show a different pattern of distribution

Fig 6 Corti organ Succinodehydrogenase

cal preparations we can recognize that the areas outside the hair-cells represent the nerve endings One may, therefore conclude that one of the energy sources for the sound transformation is to be found here because of the strong concentration of respiratory enzymes in these areas

From observations of other organs we know that an increased functional strain can cause a change of the activity of the respiratory enzymes, similar changes can be readily examined in the inner ear There we are in a rather unique position for evaluating changes of different parts, some of which react selectively to the applied stimulus, whereas other

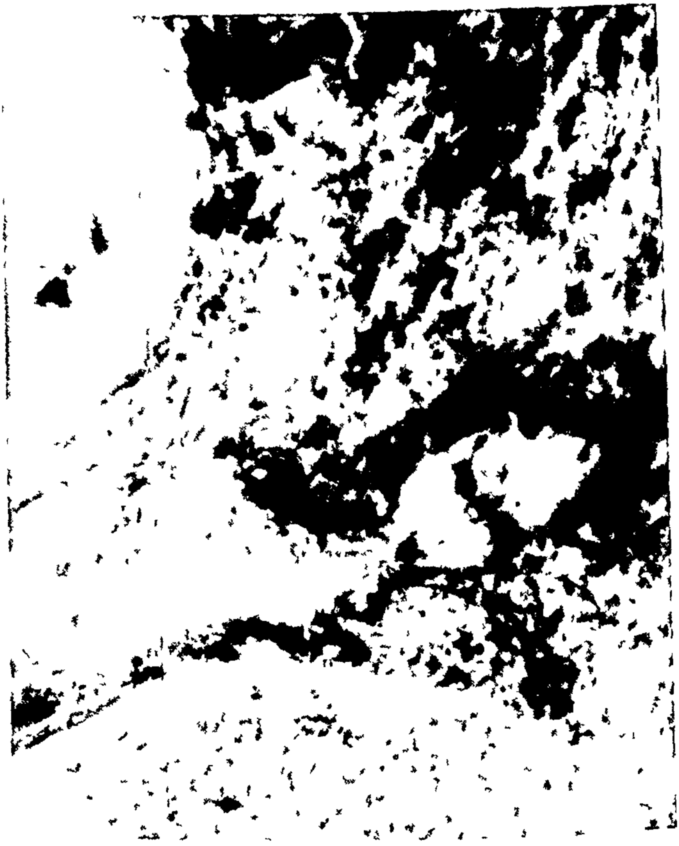

Fig 5 Sulcus externus with a glandlike canal Succinodehydrogenase

In the sensory cells the diaphorase also is distributed differently from the dehydrogenases In the case of DPN-diaphorase for example, one sees that the whole cytoplasm of the cells is filled with enzyme while the surrounding areas remain free The succinodehydrogenase on the other hand is very strongly concentrated in the basis of the outer hair cells and also in the areas surrounding these bases

From electronmicroscopic pictures we know that the basis of the outer hair cells contains many mitochondria’ The nerve endings of the cochlear nerve which surround the bases of the outer hair cells are also filled with mitochondria If we compare such electronmicroscopic pictures with histochemiinstances the sensory cells also have lost their enzymatic activity which is always accompanied by the aforementioned changes of the nerve endings After approximately eight to ten days finally all external hair cells in the exposed area are destroyed, and only the inner hair cells remain unchanged

If one allows an animal to live four to six weeks after termination of the exposure, the same histochemical changes can be observed as in animals which have been sacrificed immediately after exposure

These observations indicate the following time sequence in the development of the damage to hair cells The first histochemical findings after experimental exposure to sound represent a decrease of the respiratory enzyme in the nerve endings In later stages the entire outer hair cells are also affected, and the complete destruction of these specific cellular elements finally follows

These changes occur only after the exposure to a continuous tone If this tone is rhythmically interrupted, so that after an impulse which lasts 250 milliseconds we allow a 250 millisecond interval, the described effect then can be avoided The interpretation of the findings is not very difficult The destruction of the outer hair cells following an acoustic trauma under these conditions is not the result of a mechanical injury but rather the result of a complete exhaustion of the metabolites The disappearance of respiratory enzyme precedes the dissolving of the cell structure, and it is possible to avoid this phenomenon by short recovery periods

The respiratory enzymes are bound to membrane structures in the mitochondria (cristae mitochondriae) Spondlin[16] by electronmicroscopy showed a breakdown of these membrane structures in the nerve endings and in the bases of hair cells following acoustic trauma, therefore, a recovery of the enzymatic activity is no longer possible

Equally interesting as the ratio in the organ of Corti is the distribution of succinodehydrogenase in the ligamentum spirale This enzyme is especially active in the epithelial cells of the stria vascularis For many years it was believed that areas, which are not affected by treatment, can serve also as controls in the same organ of the same animal

Actually after stimulation with pure tones with an intensity range of approximately 75 to 85 db for two days we can recognize the first changes in the enzyme profile of the Corti organ [22] In the responding areas there can be found numerous outer hair cells with decreased dehydrogenase activity,

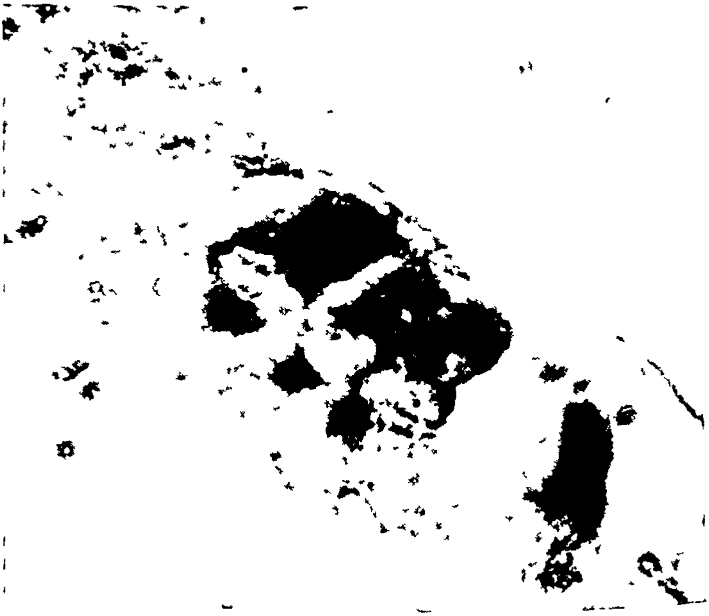

Fig 7 Outer hair cells DPN-diaphorase

especially in the areas of the cell bases and the immediate structures, the nerve endings With increasing duration or intensity these findings become more pronounced At first one sees a few destroyed sensory cells in the center of the respective area, the number of which later will increase considerably Many sensory cells remain unchanged when evaluated by conventional light microscopy Histochemically, however, a few changes are already noticeable Sometimes the enzyme reaction fails only in the area of the nerve endings while the hair cells themselves are nicely stained In other tion of the oxygen by inhibition of the respiratory enzymes A short time after such an injection there is a decrease of the amplitude of the microphonics Gisselsson believed that the respiratory enzymes in the hair cells would be inhibited by injecting potassium cyanide[17] When we examine the experimental animals histochemically we find that the activity of the respiratory enzymes in the organ of Corti is unchanged or blocked in the stria vascularis I believe, therefore, that histochemical methods supplement the physiological investigations of Davis,[2] Tasaki,[18] Misrahy,[10] and other investigators, on source of endocochlear potentials

The properties of the endolymph and of the perilymph, especially their potassium and sodium content, has been known for a long time During the last year we were very much interested in the investigations of Rauch,[11] who showed that the ratio of sodium to potassium in the fluid between the membrana reticularis and the basilar membrane is inversely related to the endolymph These findings are definitely of great importance for the biology of the inner ear On the other hand, we now know some facts about the chemistry of the stimulated nerve membrane of the peripheral nerve and the meaning of the potassiumion pump We know that the energy for the potassiumion pump comes from ATP, and thereby forms cell respiration One can, therefore, assume that sound-transformation in the inner ear follows a similar mechanism

Answering the question raised in the beginning regarding the increase of energy during the transformation of sound, I should say that the histochemistry of the respiratory enzymes points to the fact that the main energy sources are located in the stria vascularis and in the sensory cells

The stria vascularis, which has for many years been the battery of the endocochlear potential, shows histochemically the most intensive concentration of respiratory enzymes According to Davis[3] the sensory cells modulate the endocochlear potentials According to newly acquired knowledge of the properties of the fluid surrounding the hair cells, the sensory cells can perform this modulation by an exchange of ions, in the endolymph is originated from the stria vascularis In earlier times it was assumed that similar to the perilymph production the vessels of the stria vascularis were also involved in the production of endolymph The chemical properties of the endolymph differ from all other extracellular fluids, in that the concentration of potassiumions is much higher than the concentration of sodiumions[15], therefore, it seems very unlikely that the endolymph is a blood filtrate, because this would require that somewhere the potassium must be separated from the blood plasma and the inflow of sodium must be prevented These two functions can be achieved only by an active metabolic process Only the epithelial cells in the stria vascularis are equipped for such an active metabolic process because of their content of respiratory enzymes

Another area of the ligamentum spirale shows also considerable concentration of enzymes This area is located behind the sulcus externus Rather long processes of the epithelium have been observed in this area[9] Some sections show that these extensions of the epithelial cells have been intensively stained by the indicator Approximately 50 years ago Shambaugh[14] saw in these areas, gland-like canals which were lined with epithelium, and he believed that these epithelial cells also had secretory functions It is very interesting that we found identical canals in our sections

Not too many years ago Borghesan[1] showed that in these areas of the ligamentum spirale most of the blood vessels are concentrated, and he believed that the actual secretion of the endolymph takes place here and that the electrolytes were secreted in the stria vascularis This conclusion is equally as difficult to prove as to disprove

Histochemically it is known that the amplitude of the microphonics decreases when there is lack of oxygen It is also known that the microphonics depend upon the presence of an endocochlear potential, and, finally, we know the relations between the content of potassium ions in the endolymph and the endocochlear potential

Gisselsson[6] produced lack of oxygen by injecting potassium cyanide into the carotid artery thereby blocking the utilization of the oxygen by inhibition of the respiratory enzymes. A short time after such an injection there is a decrease of the amplitude of the microphonics. Gisselsson believed that the respiratory enzymes in the hair cells would be inhibited by injecting potassium cyanide.[17] When we examine the experimental animals histochemically we find that the activity of the respiratory enzymes in the organ of Corti is unchanged or blocked in the stria vascularis. I believe, therefore, that histochemical methods supplement the physiological investigations of Davis,[2] Tasaki,[18] Misrahy,[10] and other investigators, on source of endocochlear potentials.

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SIXTH INTERNATIONAL CONGRESS ON DISEASES OF THE CHEST

The Sixth International Congress on Diseases of the Chest will be held at the University of Vienna from August 29 to September 1, 1960

INNERVATION OF THE COCHLEA IN RELATION TO HEARING LOSS[*†]

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(By Invitation),
Chicago, Ill

The report reviews briefly the present information regarding hearing loss associated with disorders of nerve supply of the cochlea, and considers the three following systems of fibers autonomic, efferent and afferent

A The Autonomic Innervation

The current knowledge about autonomic innervation of the cochlea and its physiologic correlates is fragmentary and often contradictory

It is believed that the sympathetic innervation is represented by postganglionic fibers of the stellate ganglion The fibers would follow successively the vertebral, basilar, anterior inferior cerebellar and internal auditory arteries A supply of sympathetic fibers from the internal carotid artery and/or segmental contribution from other sources has been postulated[1] Postganglionic fibers from the cervical ganglia reach the cochlear nerve via tympanic plexus, geniculate ganglion and facial nerve[2]

The sympathetic fibers innervate the internal auditory artery and its primary and secondary tributaries situated in the modiolus Arterioles and capillaries of both spiral lamina and spiral ligament including stria vascularis, seem to be entirely without innervation[3,4] Studies of the stria vascularis by Smith and Engstrom, et al,[5] did not mention the presence of nerve fibers in its vascular bed

The contribution of the parasympathetic division to the

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cochlea is unknown. Since Rasmussen[7,8] described the olivocochlear bundle it is quoted often that this represents a preganglionic parasympathetic innervation; however, this opinion seems untenable after further investigations by Rasmussen[9] and other authors.[10-14]

It is assumed that neurovascular disorders of the inner ear are associated with imbalance of the autonomic nervous system. The etiology of Ménière's disease and sudden deafness are attributed to arterial vasospasm of inner ear blood vessels. This theory has been elaborated and expanded despite little anatomical, physiological and pathological evidence.

The surgical procedures which aim to release the inner ear vascular supply from sympathetic action[15-25] in man, are not satisfactory. Little and sometimes contradictory results are reported on stimulation and ablation of the autonomic nervous system of the inner ear;[26-28] similarly, contradictory and often paradoxical results have been obtained with certain drugs which supposedly act upon the sympathetic and parasympathetic systems.[1,29,30]

There are several possible explanations for the failures and discrepancies in the surgical and experimental results mentioned above. The failure of the surgical procedure may, in some cases, be due either to the fact that the operation was performed in an irreversible lesion, or that the disorder was primarily not of autonomic origin. In other cases the failure may be caused by both incomplete denervation and sensitization. The clinical observations in sympathectomy in other organs[31] gave rise to two important conclusions which have a bearing on sympathectomy of the inner ear: first, in order to insure the result, the organ or structure must be denervated completely; second, to obtain maximal reduction of tone in arteries and minimal sensitization of the organ denervated, the sympathetic pathways must be interrupted at the preganglionic neuron.

The failure of sympathetic stimulation to induce physiologic and structural changes in the inner ear may be brought about by several factors. First as pointed out by Fulton,[32] the action of the autonomic nervous system varies considerably from one species to another, and also among organs, furthermore, in the same organ the action varies according to the physiologic state of the tissue at the time of the experiment. Another important factor is that the internal auditory artery belongs to the central nervous system vascular supply which is known to be little affected by sympathetic stimulation.[33] Arteries larger than 50 microns show vasoconstriction of about 8 per cent, while there is no apparent change in smaller vessels. These factors must at least partially account for the results with drugs acting upon sympathetic and parasympathetic systems.

It is assumed that the autonomic imbalance may produce several degrees of vasoconstriction in the inner ear, but the corresponding functional and structural correlates are matters of much speculation. There are some experimental observations[34, 35, 36] supporting the opinion that if the autonomic imbalance produces arterial block, then a striking functional impairment must follow. In these experiments a mechanical temporary sudden block of the internal auditory artery in the guinea pig was produced. The blood flow of the capillary bed in the stria vascularis was used as an index of complete obstruction. Cochlear potentials were observed and measured before, during and after the arterial block. The survival time of neural components was about 45 seconds while that of aerobic cochlear microphonics was twice as long. The revival of cochlear function was related to duration of the occlusion. Complete recovery may occur after a blocking of no more than 30 minutes, between 30 and 50 minutes the recovery was partial and a permanent depression followed an occlusion of 60 minutes or more. The latter produced severe and irreversible changes in sensory hair cells and afferent neurons.

These experimental observations strongly suggest that some human cases of sudden deafness may be due to occlusion of vascular supply, however there is little evidence whether the condition is brought about by vasospasm embolus or other mechanisms.

The extensive literature on the anatomy, physiology and physiopathology of autonomic nervous system of the inner ear can be found in the comprehensive work of Montandon,[1] Williams,[25] Bocca[37] and others [38,39]

B The Efferent Innervation

Rasmussen[7,8,9] demonstrated that the crossed limb of the so-called olivary peduncle originating in the superior olivary complex enters the cochlea of the opposite side This olivo cochlear bundle passes into the inner meatus between superior and inferior division of the vestibular nerve, it reaches as far as the saccular ganglion, and crosses toward the cochlear nerve forming the fasciculus of Oort The fibers enter the cochlea by two or three fasciculi which run along the cochlea as a spiral intraganglionic bundle [3,9,10,11,40] The observations indicate that not all components of the spiral intraganglionic bundle are efferent fibers, some of them probably belong to the afferent innervation [3,9] Rasmussen[9] suggests that in addition to the olivo-cochlear bundle other efferent systems to the cochlea may exist

The nerve endings of the olivo-cochlear bundle still remain unknown although some authors[10,11,12] proposed that the bundle innervates the internal hair cells, however, recent investigations[38,41-45] suggest that the olivo-cochlear bundle innervates both inner and outer hair cells The bundle seems to represent the final pathway of a descending system which extends from the cortex to the cochlea [46] The system makes synaptic connections with many and probably all auditory nuclei According to Galambos,[46] activation of the descending system may represent a neural mechanism acting as modulator of the incoming auditory signals So far, his experimental observations show that activation of the olivo-cochlear bundle inhibits the cochlear neural components The mechanism of this phenomenon and its effect upon hearing in normal and pathologic conditions are still speculative matters

C The Afferent Innervation

In man the cochlea is innervated by about 35,000 afferent neurons [47,48] The central axons terminate in the primary cochlear nucleus [49,50,51] The dendritic processes are distributed among 23,000 sensory hair cells in the organ of Corti The anatomical pattern of nerve fibers among hair cells have been described by several authors (von Ebner,[52] Ramón y Cajal,[53] Held,[54] Lorente de Nó,[3] Polyak,[55] and others). The internal hair cells are innervated mainly by radial fibers while the external hair cells are supplied by spiral fibers. The latter extend considerably along the organ of Corti. The radial fibers innervate one or a few sensory cells while many hair cells are innervated by one spiral fiber. The electron microscope shows two types of nerve endings on the sensory hair cells,[38,43,44] large and small terminations containing more or less granules or vesicles. The question whether one corresponds to an afferent nerve and the other to an efferent fiber has not been settled. The density of afferent neurons along the ganglion of Corti is uniform except at both the basal and apical end of the Rosenthal's canal, where the density is diminished.

The significance of this multiple innervation is unknown. The evidence from human material and experimental animals, however, suggests that this multiple innervation is not necessary for coding auditory signals of simple nature such as threshold of pure tones. Dandy[56] reported that section of as much as two-thirds of the cochlear nerve in some cases of Ménière's disease, did not modify significantly the pre-operatory audiometric thresholds. In other cases the section produced a small or pronounced threshold shift, mainly for high frequencies.

Several investigations in laboratory animals[57-61] revealed that an atrophy of as much as 75 per cent of neurons may be associated with thresholds within normal limits. Schuknecht and Woellner[60] showed in the cat that nerve degeneration localized at the basal coil produced larger threshold shift (high frequencies) than that arising from a lesion of similar magnitude localized at the apex (low frequencies). The explanation may be due to basalward spread of the response-area for low frequencies as intensity of stimulus increases.

The multiple cochlear innervation, however, seems to be necessary for recording complex auditory signals, such as speech or even loudness of a simple tone. Loudness seems to be a function of the total number of nerve impulses per unit time This is determined by the number of fibers activated and number of nerve impulses in each fiber Since the latter is limited by the refractory period it is apparent that the number of fibers activated is critical for carrying loudness function, thus, a diminution of neurons will be associated with impairment of loudness function It is known that diseases involving only the cochlear nerve generally present no recruitment while this is associated with lesions of the sensory cells Dix[62] reported two cases of acoustic neurofibroma presenting loudness recruitment which disappeared after surgical removal of the tumor This finding was attributed to impairment of sensory cell function resulting from decreased vascular supply This explanation is questionable because impairment of circulation should affect nerve cells equally if not more strongly than sensory hair cells

The multiple innervation seems to play an important role in coding speech Lesions affecting the cochlear nerve, such as acoustic neurinoma, and presbycusis of "neural atrophy" type produce severe loss in auditory discrimination

SUMMARY

The mammalian cochlea is innervated by three systems of fibers, *ie*, autonomic, efferent and afferent

There is little evidence that imbalance of the autonomic nervous system produces vascular disorders resulting in hearing loss The theory of vasospasm as a manifestation of autonomic imbalance requires a thorough investigation

The information about the efferent system is still fragmentary It is not known whether and how disorders of this system affect hearing function

The significance of multiple innervation in the cochlea is unknown, however, for coding auditory signals of simple nature, such as pure tones at threshold, it is not necessary Several observations in man and animals revealed that a large reduction of nerve population is compatible with thresholds within normal limits On the other hand, coding of complex auditory signals such as loudness function and speech, requires integrity of cochlear innervation A typical example is absence of loudness recruitment and severe loss of auditory discrimination associated with an acoustic neurinoma

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ANNUAL OTOLARYNGOLOGIC ASSEMBLY

September 24-30, 1960

The University of Illinois College of Medicine Department of Otolaryngology will offer an intensive postgraduate basic and clinical program for practicing otolaryngologists The Assembly offers a compact program of one week of daytime and evening sessions It is designed to bring to specialists a wide variety of current advances in management, therapy and philosophies Review of basic morphologic features is also included by means of laboratory demonstrations, dissection and prosection, all augmented by visual aids

Panel programs have been designed to bring out special features of otologic and reconstructive surgery and tumors of the head and neck Luncheon chats are an important part of the daily program

Interested physicians should write direct to the Department of Otolaryngology, University of Illinois College of Medicine 1853 West Polk Street, Chicago 12, Ill

CLINICAL IMPLICATIONS OF RECENT RESEARCH ON THE INNER EAR[*][†]

DONALD H ELDREDGE, M D ,
(By Invitation),
St Louis, Mo

Recently I have been doing some worrying about two distantly related problems The first was the technical problem of relating changes in electrophysiological responses from the cochlea of the guinea pig following acoustic trauma to the anatomical changes that Dr Walter Covell and I could see in the microscopic sections of the same cochleas The second problem was, "What guides do we have for the causal relations between exposure to noise and loss of hearing?" The latter question became especially important in Missouri with the enactment of new sections on Occupational Hearing Loss in the Missouri Workmen's Compensation Law[1]

I plan to suggest that the evidence arising from the technical problem should help the clinician to divide the problem of causal relations into more manageable packages This by no means solves the whole problem of causal relations, but I believe it will help to mark off two extreme areas of greater certainty from a middle area of great *un*certainty In general outline the hypothesis is not new I will suggest that acoustic trauma can be considered to depend upon a pathologic physiology that is different from that of noise-induced hearing loss I should emphasize here that I do *not* use the terms "acoustic trauma" and "noise-induced hearing loss" synonymously I believe acoustic trauma to be an acute process, more truly traumatic, and noise-induced-hearing loss to be a chronic process that is not, strictly speaking, traumatic Under certain

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[*] Read at the meeting of the Middle Section American Laryngological Rhinological and Otological Society Inc Chicago Ill Jan 18-19 1960

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extreme but common conditions of exposure to noise we can exclude one or the other of the two processes Whenever these conditions are met, I believe the possibilities to be considered with respect to causal relations are greatly simplified

STATUTORY OCCUPATIONAL LOSS OF HEARING

The expert testimony of the otolaryngologist is usually required to establish the hearing status of the claimant and also to establish the cause of any defective hearing To demonstrate the problem I will refer to the new statutes for Missouri Other statutes may differ in detail, but the general problem is universal In Missouri the pertinent paragraphs of statute to be implemented by testimony begin as follows

"An employee shall be conclusively deemed to have been exposed to the hazards of an occupational disease when for any length of time, however short, he is employed in an occupation or process in which the hazard of the disease exists "

The next paragraph is legally complex but concludes

" and, in such cases," (silicosis, asbestosis, loss of hearing due to industrial noise, and radiation disability) "an exposure during a period of less than ninety (90) days after the effective date of this section shall be held not to be a last exposure "

The paragraph implies that the named occupational diseases are not immediately manifest upon exposure but require at least ninety days for an significant manifestation

Occupational disease is defined both by exclusion and by inclusion in another section

" ordinary diseases of life to which the general public is exposed outside of the employment shall not be compensable, except where the said diseases follow as an incident of an occupational disease as defined in this section A disease shall be deemed to arise out of employment only if there is apparent to the rational mind upon consideration of all the circumstances a direct causal connection between the conditions under which the work is performed and the occupational disease, and which can be seen to have followed as a natural incident of the work as a result of the exposure occasioned by the nature of the employment as the proximate cause, and which does not come from a hazard to which workmen would have been equally exposed outside of the employment"

Finally, we have in the next paragraph

"Loss of hearing due to industrial noise is recognized as an occupational disease for the purposes of this chapter and is hereby defined to be a loss of hearing in one or both ears due to prolonged exposure to harmful noise in employment Harmful noise means sound capable of producing occupational deafness"

This definition of harmful noise makes a closed circle in only two sentences This is little help for the expert witness To a very considerable degree he must have his own responsible opinion in each case The statutes could not be written to define a harmful exposure to noise more accurately because enough information to write a good statute simply is not available Accordingly the wisdom of the otolaryngologist must be substituted for the information that is not available

ACOUSTIC TRAUMA IN THE GUINEA PIG

The past history and present problems of experiments in acoustic trauma on the guinea pig[2 3 4 5 6] and other laboratory animals now seem to provide some clues that may fortify the otologist's wisdom The comparisons I make to man can only be qualitative This is because I believe the ear of the guinea pig to be 20 db to 25 db more sensitive to noise than the ear of man

If I expose the ear of the guinea pig for one to four minutes to a tone in excess of 140 db SPL and examine the ear a few days later, the destruction seen at the site of worst injury is often complete A few scattered fragments of cells of the organ of Corti are apparent, but a low epithelium will have replaced the familiar structures

If the exposure is reduced to 140 db for the same duration, the worst injury is still very great, but enough of the supporting cells may remain in place to preserve a recognizable structure Reissner's membrane may be broken The rods of the tunnel of Corti may be fractured, allowing collapse and fracture of the reticular lamina The hair cells are usually pretty well destroyed No definite cochlear microphonic (CM) can be recorded from such areas Occasionally one may record CM from adjacent less injured areas

When the exposure is reduced to about 135 db and lasts a few minutes, rather than one or two, the overall structure of the organ of Corti is even better preserved Occasionally there may be rupture of Reissner's membrane, but usually the tunnel of Corti and the reticular lamina remain intact Many of the hair cells and their Deiters' cells will be severely injured Often, but not always, there will be enough intact cells to give a small CM Adjacent areas are less severely injured and may yield fairly good CM

The character of the worst injury changes again for exposures to 125 db lasting from 20 to 30 minutes Here we may find hair cells normal or almost normal in appearance, but Deiters' cells may be rather badly broken apart Pieces of these cells may even be seen scattered in the tunnel of Corti The threshold for a three microvolt CM response from one of these areas may be elevated 50 db, however, the cells do give a true response that may grow to considerable voltages with strong test tones

Throughout the sequence that I have been tracing, the sound pressure level for each successive example has been reduced Longer exposures were required for the lower sound pressures in order to produce visible injuries All exposures clearly injured the ears but the *character of the worst injury in each ear changed progressively* Now, if we reduce the sound pressure level to about 115 db and increase the duration of the exposure to about two hours, the anatomic changes become even less pronounced Some small lesions involving hair cells and supporting cells occur There are small to moderately large losses in CM and in the nerve action potentials (AP) recorded from these ears Unfortunately, the degree of loss of cochlear potentials does not regularly match the severity of the injuries seen under the microscope Too often substantial losses of cochlear potentials are paired with a relatively undisturbed organ of Corti Similarly, an organ of Corti that shows a moderate number of apparently injured hair cells may show very little loss of cochlear potentials These contradictory relations appear whether the ear specimens are taken immediately after exposure or not until three months after the the exposure

The evidence that the hair cells are the source of cochlear microphonics is so overwhelming that we can only conclude that the usual histologic techniques of examination are not adequate to evaluate these injuries properly I personally believe that some of the newer histochemical methods, such as those described by Dr Vosteen, and the electron microscope will help us to understand why For the discussion here, however, it is the changing nature of the injuries that is important and should be remembered

NOISE-INDUCED HEARING LOSS AND ACOUSTIC TRAUMA

In man we know noise-induced hearing loss and acoustic trauma much better by the threshold shifts produced than we do by the details of the pathology of the organ of Corti I like to reserve the term "acoustic trauma" for the permanent threshold shift and injury that follows either a single exposure to an explosive episode or a brief exposure to high level noise The constant feature of the environment is the presence of peak sound pressures in excess of 135 db SPL It does not matter whether these peaks are impulsive or occur in a continuous noise By analogy to the experiments on the guinea pig, acoustic trauma should really include any exposure that immediately and directly injures cells of the organ of Corti In man there is characteristically a rather large threshold shift, most of which is temporary

I would like to use the term "noise-induced hearing loss" for the losses that are observed only following years of exposure to relatively steady machinery noise The overall levels may be about 105 db SPL and the levels in the octave bands from 300 cps to 2,400 cps are usually between 85 db and 95 db Such noises are relatively intense compared to noises encountered in everyday life

The threshold shift that follows exposure for a single day to these noises is temporary Recovery is apparently complete For some people recovery from these exposures is always complete even after a lifetime of repeated exposure From these observed relations I infer that these exposures do not produce a direct, permanent anatomic injury The functional defect is more likely to be of the nature of a temporary biochemical depletion of necessary metabolites, the temporary accumulation of toxic by-products of metabolism, or temporary physical distortions that require metabolic activity for restoration Some ears, however, eventually fail to recover if these exposures are repeated, and develop a permanent threshold shift From this we infer that the repetition of the temporary functional defect can lead to anatomical degeneration The end result may not differ materially from that produced by acoustic trauma, but the processes that lead to the result differ significantly

Even though I have emphasized the differences between acoustic trauma and noise-induced hearing loss I do not at all mean to imply that they are necessarily mutually exclusive processes They surely can both be present in the same ear I emphasize the differences because whenever we have reason to believe that only one of the processes is present we can better understand the events that could have taken place within a particular ear

DISCUSSION

In this discussion I shall be concerned primarily with several hypothetical patients presenting permanent threshold shifts following some kind of exposure to noise The hearing is presumed, *but not definitely known*, to have been normal prior to exposure The usual medical conditions producing threshold shifts have been ruled out The question that remains is, "Could the threshold shift have been produced by the reported exposure to noise, or is it much more likely to be a threshold shift of unknown etiology that would have been present in the absence of the exposure?" The answers I am giving are consistent with the general scheme I have been developing and represent my own opinions based on this scheme

First, let us consider exposures to steady continuous noise without unusual pressure peaks The sound pressure level in the octave bands 300 cps to 2,400 cps may equal but on the average not exceed 95 db A casual visitor spends the better part of a day in the work area On leaving he complains of tinnitus and later appears with a permanent threshold shift Or an electrician has been installing new wiring in the general work area for a week Regular production and noise schedules have been maintained The electrician complains that the noise is "deafening" and later appears with a permanent threshold shift These have both been relatively short exposures to the kind of noise we associate with noise-induced hearing loss and not with acoustic trauma The exposure to noise should not be considered responsible for their threshold shifts even if we assume these ears to be especially sensitive to noise I believe an otologist can fairly testify that these exposures could not possibly be causally related to the threshold shifts

If we consider the case of a regular employee who has been on the job three months to a year, the problem is somewhat more difficult We do know that some individuals are more sensitive to noise than others and must accept the *possibility* that this exposure could be responsible for a very small permanent threshold shift A large threshold shift would be distinctly unreasonable Accordingly, the larger the threshold shift, the more certain it is that the exposure to noise was not responsible We expect a small percentage of regular employees to show large permanent threshold shifts at 4,000 cps and modest threshold shifts for lower frequencies after five or more years of exposure for eight hours a day Without other evidence from prior audiometry one would have to testify that such exposures could have produced the threshold shifts

Now let us jump to a set of exposures that we may expect to produce acoustic trauma This kind of sound field will commonly include impulsive or explosive noises with peak pressures that regularly exceed 135 db SPL The larger drop forges, jet engines and gunfire are three examples of sources for this kind of noise Under these circumstances it is possible, *but not common,* for our casual visitor to show a large temporary threshold shift with a small permanent residual If the equivalent of the electrician is assumed to have sensitive ears, he also may show some permanent threshold shift that can be attributed to his exposure The possibilities for regular employees are obvious Even so, many persons will not show permanent threshold shifts until they have been exposed for years

The rules I have been using for the above cases are not so helpful for the range of exposures from 95 db per octave band to frequent peaks of 135 db In this range we must assume the possibility that the acute processes of acoustic trauma and the chronic processes of noise-induced hearing loss can be present together One useful generalization is possible These sound pressures are not great enough to produce acoustic trauma unless the exposures last for several hours Accordingly, it should not be possible for one or a few 15-minute exposures to peak pressures of 125 db to be responsible for a permanent threshold shift There will be many other combinations of duration and level of exposure that are universally safe, but most of these are harder to identify with certainty Under these circumstances statements about the possibilities for causal relations are less positive and a larger element of judgment is required

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INTERNATIONAL POSTGRADUATE COURSE IN RECONSTRUCTIVE SURGERY OF THE EXTERNAL NASAL PYRAMID AND NASAL SEPTUM

An international course in Reconstructive Surgery of the External Nasal Pyramid and Nasal Septum will be given in Mexico City July 4-15, 1960, under the auspices of the University of Mexico Medical School and its Postgraduate Department, in cooperation with the American Rhinologic Society, and under the direction of Dr Andres Bustamante Gurria, of Mexico City The guest professor will be Dr Maurice H Cottle, Chicago Medical School He will be assisted by a faculty of otorhinolaryngologists from the United States and Mexico

Class membership is limited Apply for registration information to Dr Robert M Hansen, Secretary, American Rhinologic Society, 1735 North Wheeler Ave, Portland 17, Ore

HISTOPATHOLOGIC INNER EAR CHANGES IN DEAFNESS DUE TO NEOMYCIN IN A HUMAN *†‡

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The toxic effect of certain antibiotics on the sense organs in the inner ear has caused concern since streptomycin first came into clinical use The known offenders have been streptomycin, dihydrostreptomycin, neomycin and kanamycin, all of which belong to the "useful but unruly family of basic streptomyces antibiotics "[1]

This concern has been recently intensified with the increasing incidence of ototoxicity following relatively small doses of these drugs The widespread use of dihydrostreptomycin in combination with other antibiotics as recently reported by Shambaugh, et al ,[2] undoubtedly has been related to this increase The common occurrence of hearing impairment and often severe deafness after prolonged administration of these drugs with high dosage has been well documented (Streptomycin,[3,4,6,7,8] Dihydrostreptomycin,[2,5,7,8,9,10] Neomycin,[11,12,13,14,15,16,17] Kanamycin[1,18]) The toxicity of streptomycin for the vestibular system as compared with the auditory system has been generally recognized In contrast, dihydrostreptomycin, neomycin and kanamycin has shown greater toxicity for the auditory system A greater tendency to ototoxicity in the

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†From the University of Chicago Medical School and the University of California Medical School

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presence of high blood concentrations has been reported by certain authors,[1,11 12,15 19 20 21] but denied by at least one author [22]

A toxic effect on the kidney has been reported from neomycin and kanamycin,[1,15,23 24 25] but less frequently from dihydrostreptomycin and streptomycin [26 27 28] This has been thought to be a factor in increasing the blood levels and the tendency to ototoxicity

In recent years cases of auditory damage have occurred after a low total dosage of these drugs, administered within several days only [1 17 29 30] Impaired renal function has been evident in some of these cases, but is not known to have existed in all

Onset of the hearing impairment as long as one month after cessation of therapy has been noted in many instances[1 5 9 10 22] and has been reported after as long as five months

The toxic effect of these drugs on the cochlear and vestibular systems in animals has been well demonstrated Pathologic changes in the cochlear nuclei in dogs and in the vestibular and cochlear nuclei in humans were reported by Stevenson, et al ,[31] from streptomycin Winston, et al [32 33] reported changes in the cerebellar cortex, medial and lateral vestibular nuclei and other areas in the brain stem of cats from streptomycin Christensen[34 35] reported degeneration in the central vestibular and cochlear nuclei after streptomycin in chronic experiments in guinea pigs Floberg, et al ,[36] reported inhibition of intracellular production of nucleic acids in vestibular nerve cells and Deiters' nucleus

Degenerative changes in the peripheral receptor organs in animals have been demonstrated by Causse,[37] Berg,[38 39] Ruedi,[40 41,42] Hawkins and Lurie,[43] Hawkins, et al ,[1] Schuknecht,[44] Riskaer,[45] and Nakamura ,[46] also less marked ascending degenerative changes in the nerve have been described [34 35 36 37 38 39 40 42 43 45 46] Atrophy and degenerative changes in the stria vascularis have been reported in several,[40 41 42 45 46] but were not noted in other reports [37 43] Loss of auditory function also has been confirmed by recording of the electric potentials [1 42 43 46 47]

In the human, atrophy of the sensory cells in the organ of Corti was first demonstrated in 1951 by K Graf[48] in cases of tuberculous meningitis, which became deaf during streptomycin therapy No other definitive description of histopathologic changes in the sensory receptors in the human ear due to antibiotics has been found in the literature Temporal bones from human cases of tuberculous meningitis were also examined by Whetnall and Lucas,[49] but they did not permit a differentiation between the inflammatory effects and possible drug toxicity

The present report presents histopathologic findings in a human which followed neomycin therapy The case report is as follows

The patient (H K O) was a 50 year old white male who was receiving treatment for subacute bacterial endocarditis When first hospitalized he received five million units of penicillin and one gram of streptomycin daily by injection for four days Blood culture grew aerobacter aerogenes which were inhibited by less than 10 micrograms per cc of neomycin and less than five milligrams of polymyxin B Fifty milligrams of polymyxin B was given daily by injection for seven days and ½ gram of neomycin was given concurrently twice daily by injection and continued for 19 days, except for two days when he received ½ gram daily

Although no hearing loss had been noted following the streptomycin therapy, it was now discovered that the patient was completely deaf No dizziness was present, although the patient was nauseated and vomited on two or three occasions

There was no history of disease of the ears, nose, or throat, and on entry into the Armed Services on June 24, 1942, the patient had had 20/20 hearing bilaterally Examination on December 6, 1957, revealed grossly complete deafness and otherwise essentially normal ears, nose and throat A caloric test employing 40 seconds of irrigation with 50 cc of water at 40° C produced nystagmus directed away from the tested ear with a duration of two minutes and 30 seconds for both ears

AUDIOGRAM—December 13 1957

	125	250	500	1000	2000	4000	8000
Right Ear	NR	NR	90	95	NR	NR	NR
Left Ear	NR	65	85	90	80	90	NR

Bone Conduction No response at any frequency

Speech Detection Right ear 90 Left ear 80

The patient expired on February 12 1959 at 1 00 P M The temporal bones were removed the following day and placed in 10 per cent Formalin Decalcification and mounting of the specimen were carried out in the routine manner

HISTOPATHOLOGICAL FINDINGS

Left Ear Labyrinthine Capsule and Middle Ear Spaces The left temporal bone was in a good general state of preservation The mastoid was well pneumatized, with a tract of large air cells extending posteromedially to the labyrinth as far as the internal meatus No inflammatory or other changes were present in the middle ear, mastoid, or internal acoustic meatus

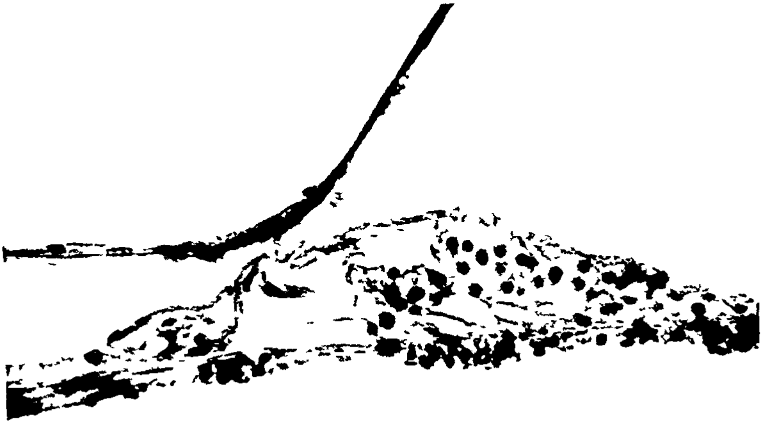

Fig 1 Microphotograph X580 showing organ of Corti left side at junction of upper and middle coil The inner hair cell is absent Outer hair cells show a combination of postmortem degeneration and toxic effects The outer pillar shows partial degeneration Depression of Reissner s membrane was localized and probably artifact

Inner Ear There were early post mortem degenerative changes of mild degree affecting the receptor organs These, however, were not great enough to obscure the pathologic changes to be described The vestibular sensory epithelium showed no changes which were not attributable to early post mortem degeneration The vestibular nerve fibers and Scarpa's ganglion appeared normal

The organ of Corti showed an absence of the inner hair cell which was nearly complete throughout (see Fig 1) Only an occasional cell remained The outer hair cells also showed degeneration throughout, but of lesser degree The estimated percentage of degenerated cells varied from 100 per cent to about 60 per cent The distribution of the degeneration was irregular Areas of maximum loss were found both in the apical coil and in the basal coil

The pillars also showed degenerative change In limited areas both in the apical and basal coils the external pillar was absent or markedly degenerated, while in some areas it showed little definite change The inner pillars showed a lesser degree of change than the outer

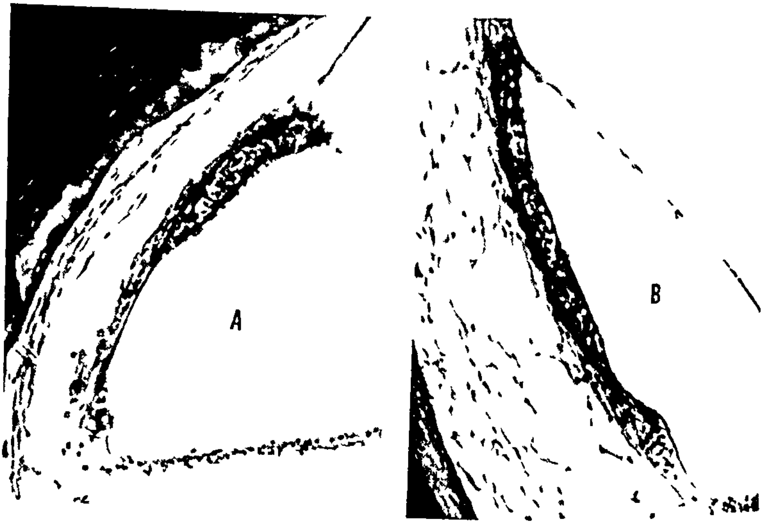

Fig 2 Microphotographs X225 showing the stria vascularis in the apical coil A and in the middle coil B of the left ear The stria in A is considered as partially atrophied but normal in extent in B The condition varied throughout the cochlea with A representing the extreme degree of loss

The stria vascularis was present throughout In some areas it was absent immediately above the spiral prominence for one-third or more of the distance to Reissner's membrane (see Fig 2) In other areas it extended down to the spiral prominence The peripheral cochlear neuron and the spiral ganglion appeared normal throughout Distortions of Reissner's membrane, the tectorial membrane and the inner pillar were present which can be ascribed to fixation artifact

Some loss of Deiters' cells was evident in areas where degeneration of pillars was most marked

Hensen's cells and the outer and inner sulcus cells were relatively well preserved

Right Ear

The labyrinthine capsule and middle ear spaces were normal throughout Pneumatization was extensive in the area posteromedial to the labyrinth The internal meatus was round

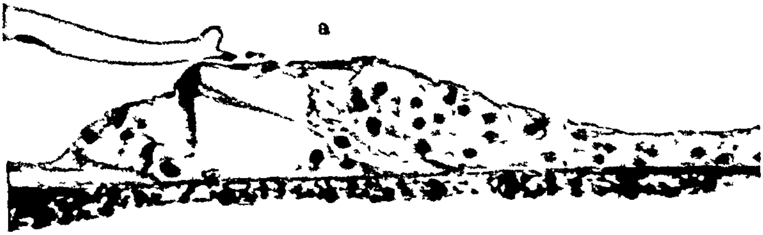

Fig 3 Microphotograph X580 right cochlea section through the junction of middle and basal coils The inner hair cell is absent Outer hair cells are mostly degenerated The pillars show early degenerative changes at their bases

Inner Ear

The state of preservation corresponded to the left ear Early post mortem degenerative changes were present but could usually be distinguished from toxic degenerative effects The vestibular receptor organs were well preserved and showed no evidence of toxic change The nerves and ganglion cells appeared normal

The organ of Corti showed changes which were almost identical with the left ear The inner hair cells were almost all absent The degree and distribution of degeneration of outer hair cells, pillars and Deiters' cells corresponded fairly well to the opposite side (see Fig 3) In one area at the beginning of the apical coil (see Fig 4) the inner and outer hair cells, and the outer pillar were absent In the middle part of the basal coil, the inner and outer hair cells, the pillars and Deiters' cells and Hensen cells all showed much degeneration (see Fig 5) The peripheral cochlear neurons were well preserved

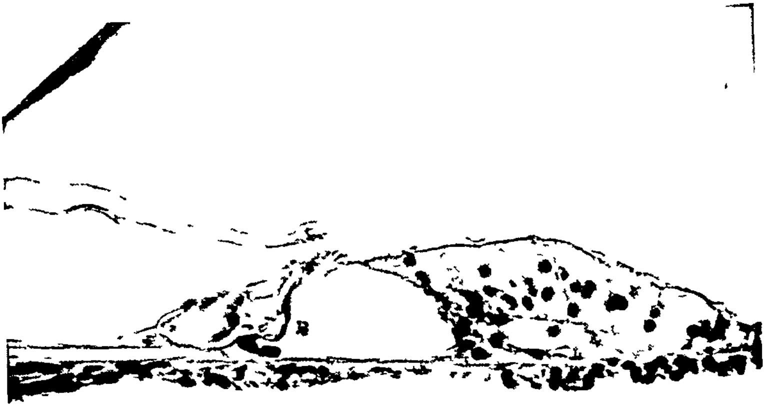

Fig 4 Microphotograph X580 right ear showing organ of Corti at junction of apical and middle coils The inner hair cell is absent Outer hair cells are absent except for possible remnants The outer pillar is absent and the inner pillar degenerated Deiters cells are seen only as distorted remnants

Reissner's membrane was depressed in some areas, as on the opposite side and the tectorial membrane and inner pillar showed the distortion typical of fixation artifact

SUMMARY OF HISTOPATHOLOGIC FINDINGS

The degenerative changes which are of significance are similar in the two ears, but more advanced in two particular areas on the right (see Figs 4, 5)

An estimate of the percentage loss of hair cells throughout indicated a slightly greater loss of both inner and outer cells on the right The distribution of loss was similar in that it was irregular, but the loss of the inner cells on the right was almost total

The outer pillar showed greater degenerative changes than the inner The loss of pillar cells was less marked than loss of hair cells Deiters' cells showed less degeneration than the pillars

The stria vascularis might be interpreted as showing some degree of atrophy in some areas (see Fig 2), but it is unlikely that this was of functional significance There was no evidence that changes in the stria were locally related to toxic degeneration of the hair cells

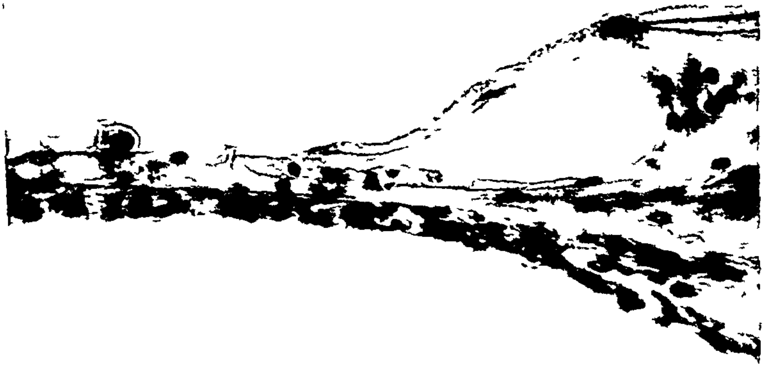

Fig 5 Microphotograph X580 right cochlea section through the middle part of the basal coil showing advanced degeneration of hair cells pillar cells Deiters and Hansen's cells

DISCUSSION

In reviewing the history of this case, the total dosage of streptomycin (four grams in four days) and of polymixin B (50 milligrams per day for seven days) could not be considered the cause of deafness The dosage of neomycin, 18 grams in 19 days, definitely appears to have been responsible

The toxic effect of the drug on the inner ears in this case has been evidenced mainly by degeneration of the inner hair cells, and to a lesser extent, the outer hair cells Lesser degrees of degeneration were evident in the pillars, and to a minor degree in Deiters' cells While it is not possible to exclude a toxic effect on the cochlear nuclei and central pathways in this case, it is perhaps significant that the peripheral cochlear neurons were well preserved The degree of degeneration of the hair cells and to a lesser degree of the supporting cells provides an adequate explanation for the loss of hearing

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REVERSIBLE HEARING LOSS IN HUMAN HYPOTHYROIDISM AND CORRELATED CHANGES IN THE CHICK INNER EAR[*,†]

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Deafness has been associated with disease of the thyroid gland by many observers, and although this deafness has generally been reported as of the perceptive type, the exact cause has not been demonstrated One of the reasons for this is that the changes associated with hypothyroidism depend upon whether the thyroid failure occurs during the embryonic development of the individual or occurs at later stages in life

The congenital type of hypothyroidism results in cretinism in which deafness is most frequently found This deafness is progressive throughout life and usually ends in total loss of hearing[1,2] Although cretinism seems to cause a perceptive deafness there has not always been support for this view Nager[3] in reporting the histology of the cretin temporal bone describes it as smaller than normal with marked hyperostosis on the medial wall of the cavum tympani which leads to narrowness and occlusion of the window niches The ossicles are deformed and enlarged, but there are only slight changes in the organ of Corti On the basis of this anatomical evidence the deafness would appear to be conductive rather than perceptive

Deafness has also been observed in acquired hypothyroidism

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[†] From the Physiological Acoustics Laboratory Department of Otolaryngology School of Medicine The University of Michigan Medical Center This investigation was supported in part by the Research and Development Division Office of the Surgeon General Department of the Army under contract No DA 49-007-MD-634

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Howarth and Lloyd[4] record five such cases Barnes[5] treated cases of hypothyroidism and associated deafness, noting hearing improvement in many instances Moehlig,[6] McLaurin,[7] and Barlow[8] have also reported deafness as occurring in cases of hypothyroidism The deafness in these cases does not seem to be as severe as that in cretins, and the evidence for either a conductive or perceptive type of lesion is no clearer

According to McMahon[9] the deafness may be conductive, if edema of middle ear or Eustachian tube is present, or perceptive from some obscure effect of lack of thyroid on the inner ear Marquet[10] states that the auditory disturbances appear late in hypothyroidism and can be attributed to intercellular edema of the tissues Both middle ear and labyrinthine lesions occur In the latter, the membranous labyrinth shows sclerotic changes and the cuboidal epithelial cells become atrophic These are found in the vestibule and constitute examples of atypical degeneration He further cites four causes of this deafness *1* a congenital process similar to that of hereditary labyrinthine deafness, *2* lesions of the osseous capsule of the labyrinth (edema and possibly hemorrhagic suffusions), *3* blockage of the two windows and *4* true intralabyrinthine hypertension based on myxedematous infiltration affecting the endolymphatic circulation

It appears then, that the aural anatomical changes present in thyroid disease vary with the degree of the disease present, and it is conceivable that slight changes in hearing could occur with subtle changes in thyroid activity, furthermore, from the reports, it seems that the changes in hearing can occasionally be reversed upon treatment of the hypothyroidism

Two cases of hypothyroidism and associated hearing loss have recently been seen in the University of Michigan Otology clinic These have been carefully examined audiometrically to determine whether the deafness is conductive or perceptive The hearing was followed in each case after treatment Following this clinical and audiometric study, an experiment was set up to see whether the anatomical locus of changes producing the hearing loss could be determined Because of the ease with which developing chick embryos can be made hypothyroid the studies were made on the developed chick ear even though it anatomically is somewhat different from the human

CASE REPORTS

Case 1 Mr R C a 52 year old male was known to have had hyper tension 17 years prior to admission to the University Hospital in February, 1959 Five years prior to admission he had a severe chest pain with a diagnosis of a myocardial infarction and at that time he was placed on potassium thiocyanate, four tablets daily This dosage was maintained for two years then decreased to three tablets daily and more recently prior to admission in February, 1959, to two tablets daily In November, 1958, he had noticed decreased hearing and gradual enlargement of his thyroid gland Concomitantly, he noticed also puffy eyelids and a dry skin

Examination in February, 1959, showed a blood pressure of 105/70, and a pulse of 84 The skin was dry warm and doughy The thyroid gland was found to be diffusely enlarged without nodules or tenderness He was referred to the ear nose, and throat clinic where the additional history of nine years' work in a very noisy environment was obtained The examination of the ears, nose and throat was essentially within the limits of normal except that the nodular thyroid was also noticed Audiograms shown in Fig 1-a reveal a perceptive hearing loss The average loss for the speech frequencies in the left ear was approximately 25 decibels and in the right ear approximately 35 decibels There is a decrease in sensitivity to the high frequencies typical of a noise induced hearing impairment A discrimination loss was recorded at 48 per cent in the right ear and 36 per cent for the left ear

The laboratory findings showed normal urine and blood picture A BMR was found to be minus 23 per cent serum cholesterol 220 mg per cent PBI 1 6 gamma per cent, and the 24 hour iodine 131 uptake was 9 8 per cent The serum thiocyanate was 11 mg per cent These findings as recorded on February 24 1959, were consistent with hypothyroidism

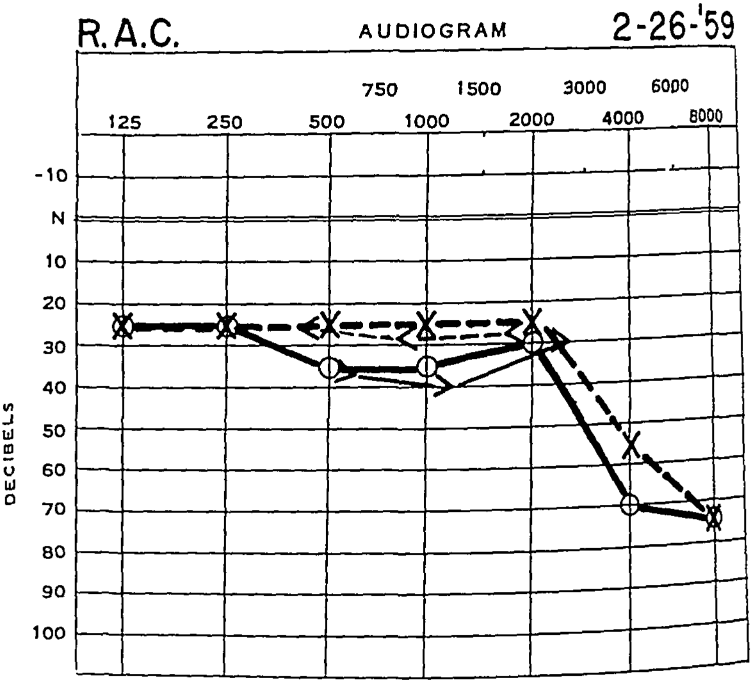
The patient was taken off his thiocyanate and treated with digitalis and dicoumarol because of his heart disease and was discharged on March 7 1959

He was readmitted March 15, 1959 because of chest pain cough and fever of 101 6° F In the interim, he stated that he had been feeling much better and that his appetite and hearing had improved Examination showed the thyroid to be slightly enlarged as before

Audiograms were again taken and are shown in Fig 1-b There is a marked improvement in his hearing The bilateral perceptive hearing loss for the high tones specifically in the region of 4,000 and 8,000 cps persists but for the speech frequency of 1 000 cps the loss is only 5 decibels

Laboratory findings on March 19, 1959 showed a 24 hour iodine uptake of 43 6 per cent which was interpreted as showing a low hyperthyroid range of uptake compatible with a gland which has been previously depleted of thyroid by potassium thiocyanate and is now enjoying a rebound hyperthyroid uptake following the withdrawal of the potassium thiocyanate In addition the serum cholesterol was now 187 mg per cent, thiocyanate 2 2 mg per cent, and the PBI 5 2 gamma per cent These findings, recorded on March 26 1959 are within the limits of normal

It is the impression that this is a case of hypothyroidism and associated



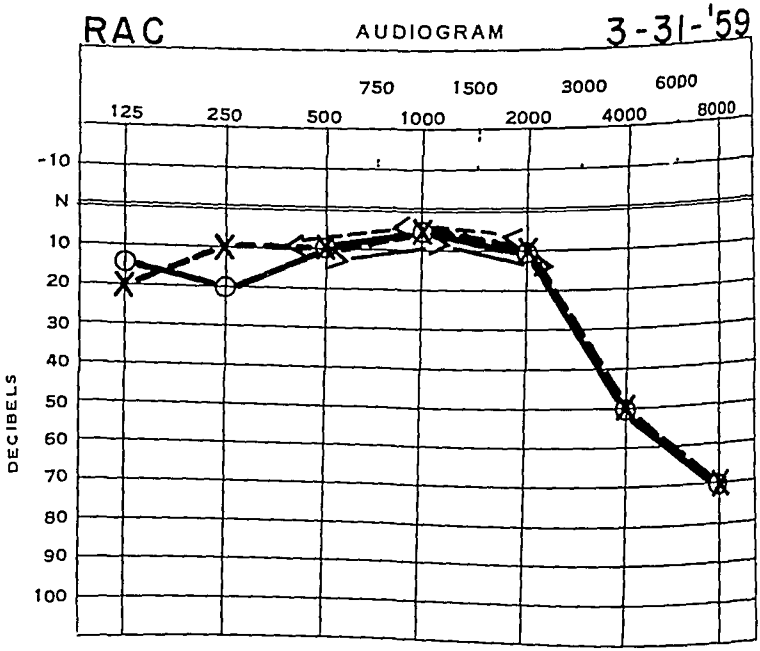


Fig 1-a Audiogram of patient with myxedema (Case 1) showing bilateral perceptive hearing loss

Fig 1-b Audiogram of patient (Case 1) showing improvement after withdrawal of potassium thiocyanate

perceptive hearing loss which has recovered following withdrawal of potassium thiocyanate

Case 2 Mrs J L, a 65 year old housewife was admitted to the University Hospital on August 2 1959, with the chief complaint of general sluggishness and fatigue Until 18 months ago she had always been in good health operating her own dry cleaning establishment but then she began to slow down and feel tired Four months prior to admission she had a heart attack and was hospitalized for six weeks Three months prior to admission she had a cholecystectomy for cholelithiasis and following this operation she never quite regained her strength At this time she could not stay up for longer than 30 minutes before lying down Concomitantly she has also noticed intolerance to cold a very puffy face dry skin and decreased hearing

Examination showed an obese lady with fine scaly, dry skin that is typical of myxedema Her blood pressure was 110/70, pulse 64 and respirations 10 Examination of her eyes showed that they were nearly closed because of the puffy eyelids

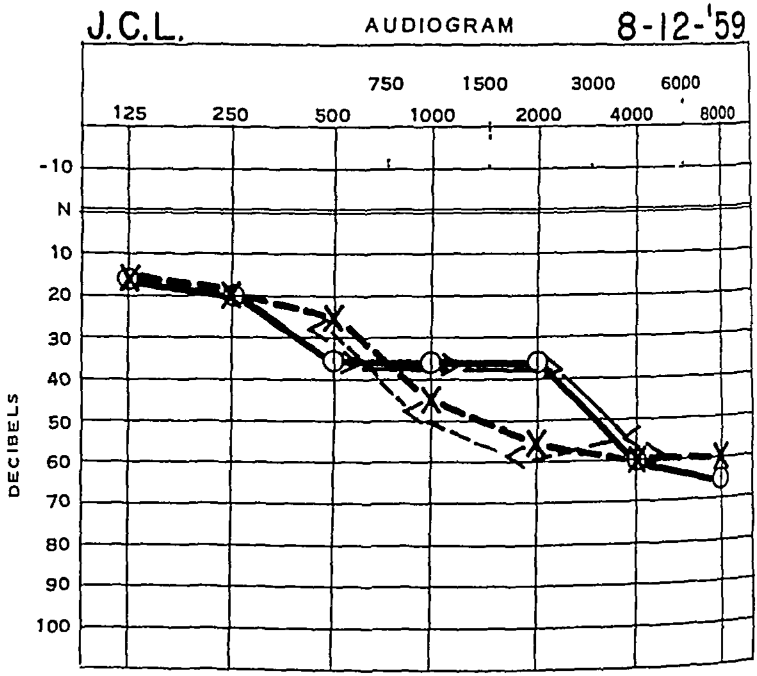
The laboratory findings on admission revealed normal blood and urine The total cholesterol was 456 mg per cent The BMR was minus 24 per cent the 24 hour iodine 131 uptake was 1 5 per cent and scintiscan of the thyroid showed complete absence of concentration of iodine 131 The PBI was 1 9 gamma per cent Because of her hearing loss she was referred to the ear, nose and throat clinic where examination of the ears nose and throat revealed them to be essentially normal The thyroid gland was not enlarged Audiograms as shown in Fig 2 a showed a bilateral perceptive type of hearing loss, greater for the high than for the low tones She had a bilateral discrimination loss of 32 per cent at 90 decibels

The patient was placed on 8 mgs of thyroid extract daily which was increased to 16 mgs daily within one week She began to feel better, have more energy and was discharged on August 30 1959

After several weeks while at home it was noted by her daughter that the patient became more ambitious and was able to stay up for longer periods of time In addition she also noticed a gain in her hearing, and on November 25, 1959, audiograms were again taken (see Fig 2 b) A noticeable improvement in hearing was recorded Again the bilateral perceptive type of hearing loss prevails the higher tones being involved more than the lower tones but the degree of depression is not so great as that during hospital stay

At the present time the patient is on 1½ gr of thyroid per day her weight is about 16 pounds less than at the time of her discharge in August 1959 her pulse is 80 and her blood pressure 140/100 Because of her cardiac condition the amount of thyroid given her is being increased very slowly to prevent any precipitation of cardiac damage

The deafness in the two cases reported above is unquestionably of the perceptive type, and more than likely the changes have occurred in the inner ear Whatever these changes are they are certainly not so drastic as to be permanent In order to determine histologically what these changes might be, a series of experiments on hypothyroid chick embryos was carried out The hypothyroidism was produced by chemical



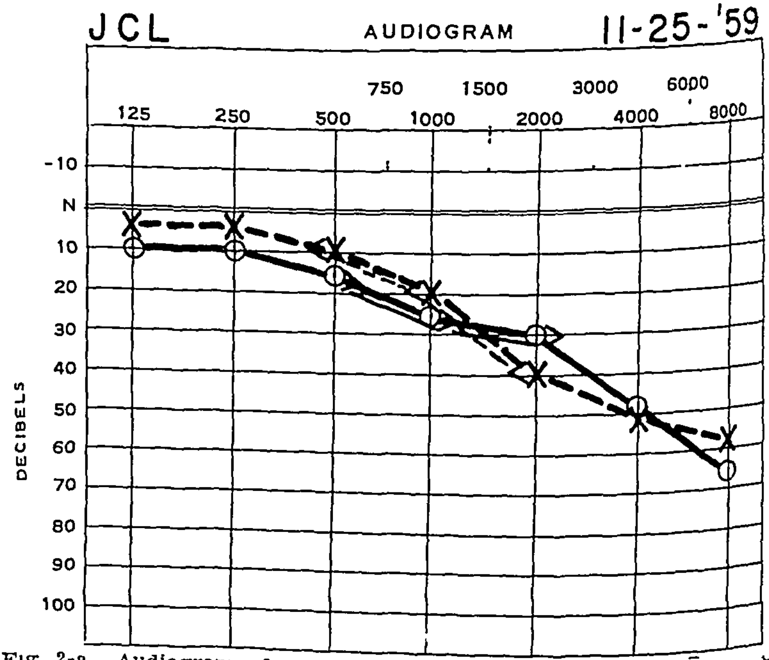


Fig 2-a Audiogram of patient with myxedema (Case 2) showing bilateral perceptive hearing loss

Fig 2-b Audiogram of patient (Case 2) after treatment with dessicated thyroid

means, and the temporal bones of the chicks were examined at the normal time of hatching

PROCEDURE

A total of 123 supposedly fertile eggs was placed in an incubator which was maintained at 37 5° C and 70 per cent humidity At various intervals the eggs were turned from their last resting position Because of incubator mishaps, non-fertility, or early death of the embryo, only 89 eggs survived to be used experimentally Of these, 17 were kept as normal controls, most of them hatching on the twenty-first day of incubation Of those hatching four were sacrificed for histological study of the normal temporal bone

The remaining 72 eggs were divided into three groups, depending upon the age at which injection with thiourea or plain chicken ringers was to be injected The times selected were fourth, ninth and fourteenth days of incubation

It has been shown[11] that 2 mg of thiourea injected into a developing chick embryo has a destructive effect upon the function of the thyroid gland and produces a marked degree of hypothyroidism For our purposes thiourea was dissolved in sterile chicken ringers[12] (2 mg per ½ cc) and refrigerated until used Before this solution, or the plain ringers, was injected each was heated to 37 5° C Injections were made under sterile conditions into the albumin in the narrow end of the egg after it had been cleaned with Lugol's solution The injection sites were then sealed with liquid paraffin

In the first group 20 eggs were injected on the fourth day of incubation Seventeen were injected with ½ cc thiourea in chicken ringers and three with ½ cc plain chicken ringers Of the 17 thiourea injected eggs, eight embryos died during incubation, one was sacrificed on the fourteenth day and eight on the twenty-first day Of the three ringers injected eggs two died during incubation and one was sacrificed on the twenty-first day

In the second group 49 eggs were injected on the ninth day of incubation, 43 were injected with ½ cc thiourea in chicken ringers and six with ½ cc plain chicken ringers Of the 43 thiourea injected eggs, 20 embryos died during incubation, two were sacrificed on the fourteenth, fifteenth, sixteenth, seventeenth, eighteenth, nineteenth and twentieth days of incubation and six on the twenty-first day Hatching is invariably delayed in the thiourea injected chicks so a delayed hatching embryo was sacrificed on the twenty-second, twenty-third and twenty-fourth day Of the six ringers injected eggs four died during subsequent incubation, and two were sacrificed at the end of 21 days of incubation

In the third group three were injected on the fourteenth day of incubation All three were injected with thiourea in chicken ringers Of these, two died and one was sacrificed on the twenty-second day

Upon sacrificing, each embryo was fixed in Bouin's solution, decalcified, and eventually imbedded in paraffin for sectioning The sections were cut at seven micra, mounted serially, stained with hematoxylin and eosin, and examined microscopically

RESULTS

Because the ears of the embryos injected on the ninth day of incubation failed to show any abnormality, the eggs injected with 2 mg of thiourea on the fourteenth day of incubation were not studied further All the embryos of eggs injected with chicken ringers, regardless of date of injection, failed to show any abnormality

General changes were noted in the chick embryos injected with thiourea at four days' incubation These chicks were all delayed in hatching and if hatching occurred they were underdeveloped The yolk sac in each chick was large, and incompletely absorbed

Fig 3-a is a photomicrograph from a normal 21-day-old chick embryo showing a section of the root of the neck The picture showing the root of the neck in Fig 3-b is that of a 21-day-old chick embryo injected on the fourth day of incubation with 2 mg of thiourea in ½ cc chicken ringers By

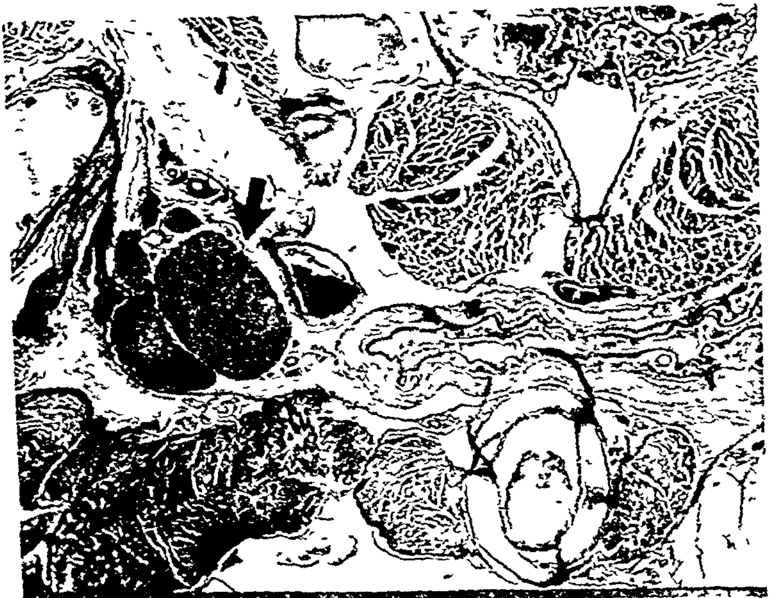


Fig. 3-a Cross section of neck of 21-day chick embryo showing normal structures. Normal thyroid gland is indicated by arrow. (Mag. 50X.)

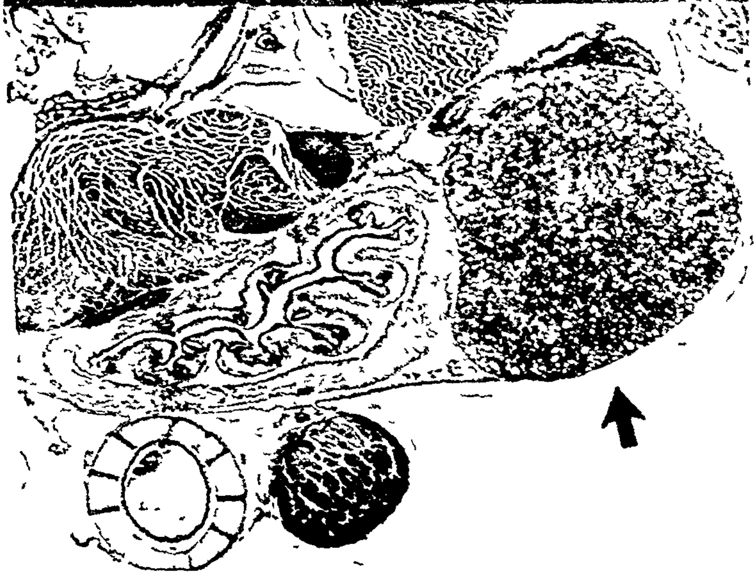


Fig. 3-b Cross section of the neck of a 21-day chick embryo injected with thiourea on the fourth day of incubation. The thyroid gland (arrow) is enlarged and the follicles contain more colloid than the normal chick embryo. (Mag. 50X.)

ringers and six with ½ cc plain chicken ringers Of the 43 thiourea injected eggs, 20 embryos died during incubation, two were sacrificed on the fourteenth, fifteenth, sixteenth, seventeenth, eighteenth, nineteenth and twentieth days of incubation and six on the twenty-first day Hatching is invariably delayed in the thiourea injected chicks so a delayed-hatching embryo was sacrificed on the twenty-second, twenty-third and twenty-fourth day Of the six ringers injected eggs four died during subsequent incubation, and two were sacrificed at the end of 21 days of incubation

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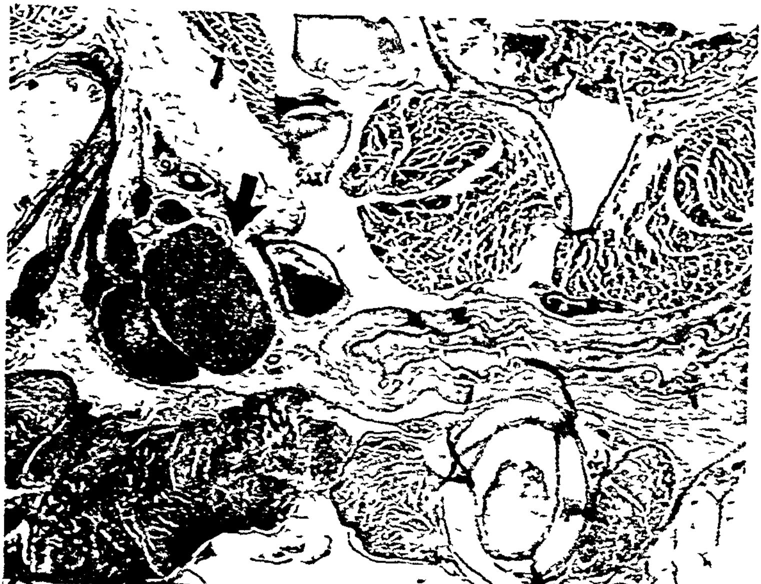


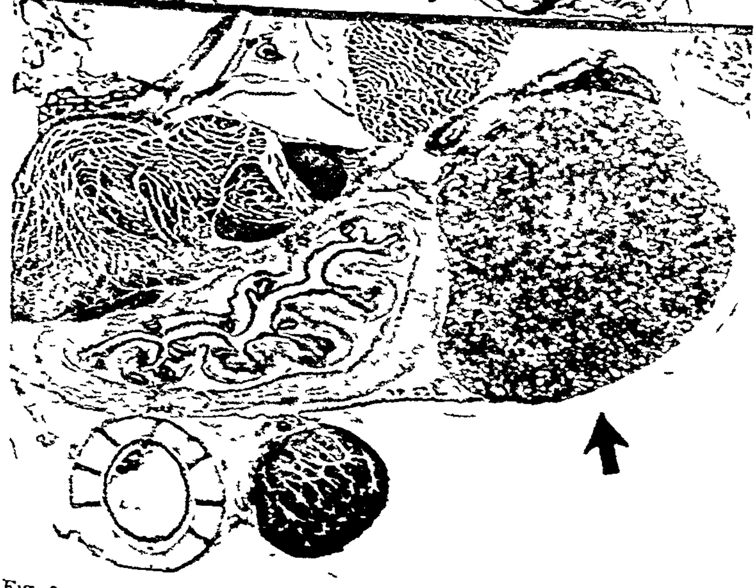
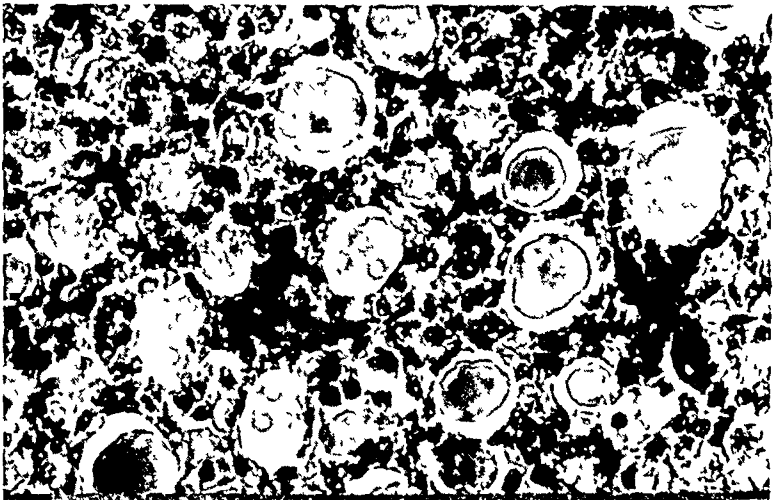


Fig 3-a Cross section of neck of 21-day chick embryo showing normal structures Normal thyroid gland is indicated by arrow (Mag 50X.)

Fig 3-b Cross section of the neck of a 21-day chick embryo injected with thiourea on the fourth day of incubation The thyroid gland (arrow) is enlarged and the follicles contain more colloid than the normal chick embryo (Mag 50X.)



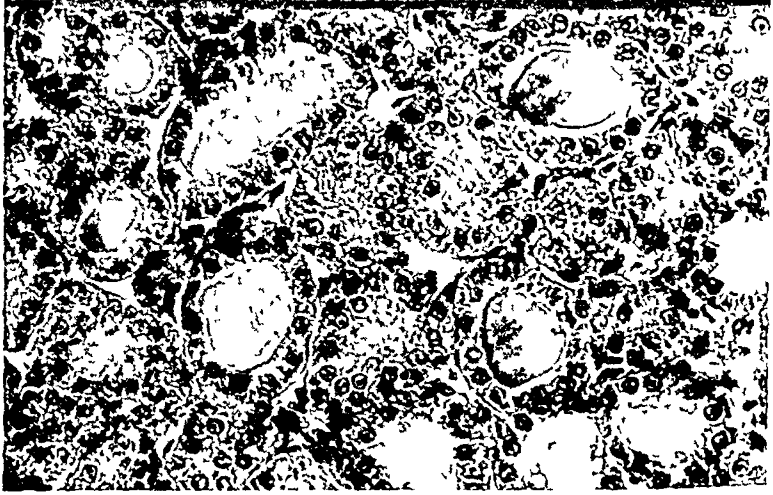
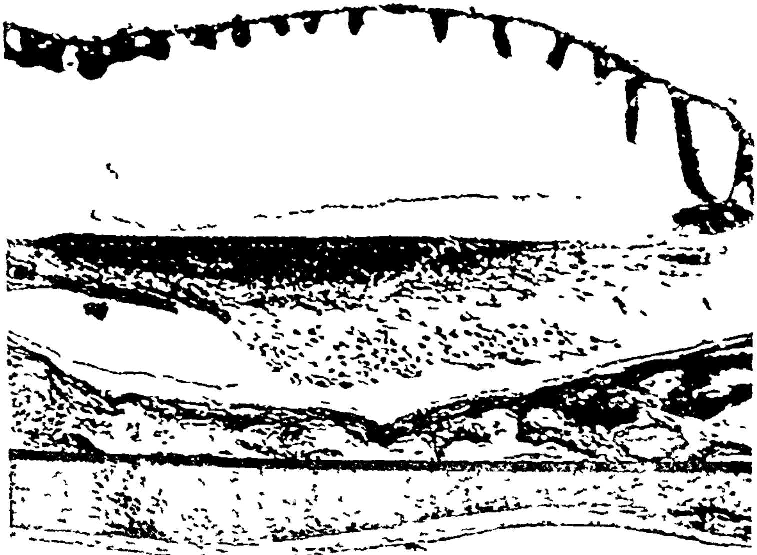


Fig 4-a The thyroid gland of the normal 21-day chick of Fig 3-a (*Mag 100X*)

Fig 4-b The thyroid gland of the chick of Fig 3-b The follicles are larger than normal their lining is of a high cuboidal type and the amount *of stored colloid is* increased in amount compared to the normal (Mag 100X)

comparing the two figures, 3-a and 3-b, it is possible to note the hypertrophy of the thyroid gland in the thiourea injected chick The cervical muscles are hypertrophied and the cornification of the esophagus is low These changes would be compatible with the findings of myxedema in which a goiter is present and the tissues edematous Figs 4-a and 4-b are



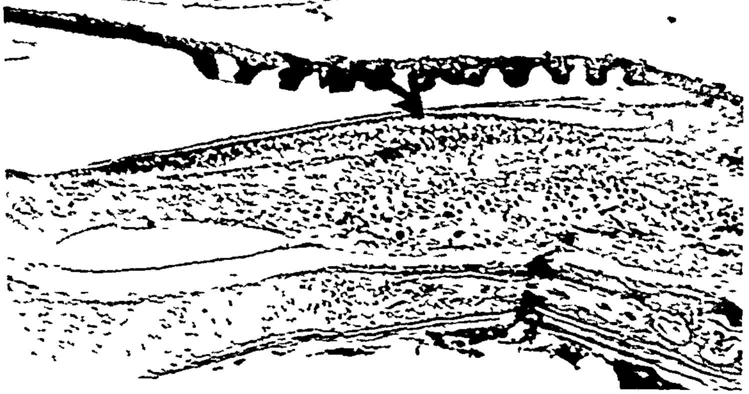


Fig 5-a The papilla of a normal 21-day-old chick embryo The normal tegmen vasculosum tectorial membrane hair cells and ganglion cells are shown (Mag 50X.)

Fig 5-b The papilla of a 21-day-old chick embryo injected with thiourea on fourth day of incubation The tegmen vasculosum is present and the tectorial membrane overlies the hair cells Apparent is the single distinct row of hair cells separated from the supporting tissue by a clear layer of edema (arrow) which extends below the sensory epithelium for its entire length (Mag 50X.)

high magnification photomicrographs of the chick thyroid glands seen in Figs 3-a and 3-b The gland is normal in Fig 4-a, but Fig 4-b shows stored colloid in many follicles, hypertrophy of the follicular lining with a high cuboidal epithelium

The changes noted in the ears of the thiourea injected chick embryos were limited chiefly to the inner ear The only evidence of middle ear abnormality was some delay in conversion of the stapes from cartilage to bone The stapes was present but not malformed No hypertrophy of the middle ear mucous membrane, or closure of either the round or oval windows was seen No fluid was present in these middle ears

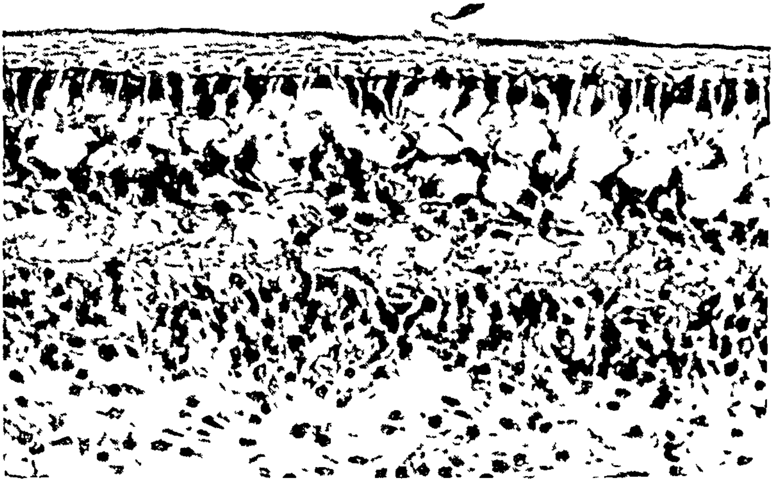


Fig 6 The papilla of a 21-day-old chick embryo, injected on fourth day with thiourea The hair cells are canine shaped and the protoplasm is increased in amount and watery in appearance The distinct layer of edema is just below the hair cells and separates the hair cells from the supporting tissues (Mag 200X)

Fig 5 a shows the normal organ of Corti of a 21-day-old chick embryo One notes here the artifact of the tectorial membrane separated from the hair cells, but the hair cells are normal, with ovoid shape, likewise are the supporting tissues, ganglion cells and auditory nerve all normal

Fig 5-b shows the inner ear of a 21-day-old chick embryo injected on the fourth day of incubation with 2 mg of thiourea in ½ cc chicken ringers Here the tectorial membrane is in contact with the hair cells, but the hair cells are separated from the supporting tissues by a clear layer of edema, several cell thicknesses in width This edema has separated the hair cells from the supporting cells completely in some areas

Fig 6 is a high magnification photomicrograph of the organ of Corti of the chick shown in Fig 5-b This shows again the edematous area separating the hair cells from the supporting tissues, also the supporting tissues appear thinned and stretched out The hair cells themselves have changed from a normal ovoid shape with scant protoplasm, to a columnar, tooth-like shape with increased amount of watery protoplasm This layer of edema and the tooth-like hair cells were present in each chick that was injected with 2 mg of thiourea at the fourth day of incubation They were not present in any chicks injected after nine days of incubation or in any control or normal chicks

DISCUSSION

The two clinical cases have shown a direct relationship between hypothyroidism and deafness They have also shown that this hearing loss can be reversed upon either the administration of the proper amount of thyroid or removal of the agent which inhibits thyroid activity

The chick experiments demonstrate a direct relationship between edema in the developed sensory papilla and injection into the egg at a very early stage of development of the antithyroid compound, thiourea The evidence is indirect but fairly conclusive that this change in the sensory area is the result of inhibited thyroid activity and not of the direct influence of thiourea As Romanoff and Laufer[11] have pointed out the effect of the thiourea depends upon the stage of development of the thyroid gland at the time of injection In our experiments injection of the antithyroid compound had no effect upon the auditory organ unless injected at least earlier than nine days of incubation, and the changes that were produced were always accompanied by hypertrophy of the thyroid gland

The chick organ of Corti differs considerably from that of the human There are no Deiters' cells nor rods of Corti The cochlea consists of only one-half turn with the sensory cells supported by columnar cells The chick auditory sensory area is more logically called the acoustic papilla It may be unwarranted to assume that because hypothyroidism in the developmental stages of the chick results in edema of the sensory area by the time of hatching, something similar could occur in the human, yet it is not inconceivable, and the reversibility of the effect in the human supports its plausibility Perhaps occasional changes in the inner ear occur with slight abnormality of thyroid activity, and it is possible that these might, therefore, be reversible with treatment of the thyroid abnormality

SUMMARY

Two cases of hypothyroidism with associated perceptive deafness have been described Treatment with thyroid in one instance and cessation of the administration of a drug inhibiting thyroid activity in another reversed this hearing loss

Experiments conducted by injecting thiourea into fertile chick eggs at a very early stage of development showed, at the age of hatching (21 days of incubation), marked hypertrophy of the thyroid gland and edema in the area of the sensory supporting cells of the acoustic papilla

The possible relationship between the occurrence of this edema and the reversibility of the hearing loss is discussed

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We are indebted to Miss Maxine Clapper for her tireless care of the incubating eggs and for her skillful preparation of the overwhelming amount of histological material

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THERAPY OF SUDDEN LABYRINTHINE DEAFNESS*

O. ERIK HALLBERG, M.D.,†

Rochester, Minn.

As the name implies, sudden deafness can and does strike like a bolt from the blue and, like it, is almost always associated with a roaring tinnitus. It may or may not be associated with dizziness.

The etiology of sudden deafness of labyrinthine origin is still obscure. Information about it is next to impossible to obtain because the deafness is very seldom associated with events that kill the patient.

REVIEW OF LITERATURE

Treatment for sudden deafness has varied greatly all over the world, depending mainly upon the attending otologist's opinion as to the etiologic factors at play. Certain circumstances associated with the onset may indicate or at least lead one to suspect the cause in some cases. Before 1950, very little had been written on sudden deafness and its treatment. Surgical treatment such as the fenestration operation has been confined mostly to Europe.

Opheim[1] in 1950 expressed the view that sudden deafness is caused by an acute rise of pressure in the labyrinth. This rise, he said, opens a road to rational intervention against this condition, which otherwise has such an unfavorable prognosis. He performed fenestration operations on three of his patients for their decompression effect. At operation he attempted to leave the ossicular chain intact. In all these patients the hearing improved somewhat, but not to a serviceable level. On puncture of the labyrinth the fluid was found

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†Section of Otolaryngology and Rhinology, Mayo Clinic and Mayo Foundation. The Mayo Foundation, Rochester, Minn., is a part of the Graduate School of the University of Minnesota.

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to be under pressure in one patient, but not in the other two Opheim stipulated the following conditions for operation

1 The loss of hearing must be complete to the extent that such a condition is recognizable, the function of the other ear being normal

2 The patient must be available for treatment early enough in the course of the disease that it may be supposed that damage to the neurosensory mechanism has not become irreparable

Berchert,[2] working with Zollner, offered a rather unique classification made up of two groups

Group 1 consisted of patients whose deafness could be attributed to psychoemotional factors that work through the vegetoneural system upon the neurosensory mechanism His five patients in this group had dizziness associated with the deafness One of these patients underwent a fenestration operation by Zollner

This patient a 38 year old woman experienced sudden total loss of hearing in the right ear associated with severe dizziness Zollner used a postauricular incision leaving the ossicles intact The inner ear was filled with blood As one would expect the deafness which was total before operation remained so

The other patients in this group were treated as though they had Ménière's disease

Group 2 consisted of patients whose deafness was thought to be caused by a mechanical effect on the cervical sympathetic nervous system or on the nerves surrounding the vertebral artery by osteochondrosis of the cervical part of the spinal column Sudden deafness and roaring tinnitus were the only symptoms None of the patients had dizziness It seemed that all developed their symptoms while at work, and X-ray examinations of the cervical spine showed arthritic changes One patient, a 39-year-old man, is reported on in detail

Sudden loss of hearing with tinnitus occurred one morning The patient had picked cherries all day the day before The results of a complete general examination were negative except for arthritic changes of the cervical spine on X ray examination An audiogram however showed marked deafness on the left (between 50 and 70 decibels) Treat ment with rest in bed and stretching of the neck was started on the seventh day Five days later, hearing had returned to normal except for moderate loss in the high frequencies

To me this approach seems a little farfetched, since many patients with arthritic changes in their cervical spine have no history of sudden deafness

Svane-Knudsen,[3] of Denmark, performed a fenestration operation on one of his patients six days after the onset of deafness What could be seen of the labyrinth looked normal Hearing did not return

In the United States, many of the publications on this subject indicate that the patients were treated as though they had Ménière's disease, in the hope that irreversible changes had not already taken place in the cochlea

A different approach to therapy might possibly come from investigations by Bocca and Giordano[4] in 1956 They reported 28 cases in which the deafness occurred after recent exposure to a draft or cold or in the course of an infection such as influenza or the common cold They presumed that the process must be very similar to that causing Bell's palsy

Van Dishoeck and Bierman[5] studied 100 cases of sudden deafness In 40 of these they found evidence that a virus infection was the cause of the sudden perceptive deafness They specifically blamed the mumps virus, but admitted, however, that vascular disorders may be common They had no specific treatment to offer

Recently Saunders and Lippy[6] examined nine patients who had sudden severe deafness Four of these definitely, and two probably, had mumps Mumps and influenza antibodies were determined on blood specimens from these nine patients, from 10 additional patients with Bell's palsy, and from 370 unselected patients (the control group) They concluded that subclinical mumps is a common cause of sudden deafness and Bell's palsy

It certainly seems possible that sudden deafness might be of virus origin in many of the patients in the younger age group, but even then, as has been stated by Fowler,[7] the hearing loss might be basically vascular in origin.

I still feel that in most patients, at least in the middle and older age groups, the hearing loss is of vascular origin. It seems that there are at least two objections to the mumps virus theory. *1.* Clinical mumps seldom recurs. *2.* Mumps is such a widespread disease that few individuals escape it during childhood.

If mumps were suspected, I would still try the effect of vasodilation, although if the patient were seen right after the onset of deafness, I might also give mumps vaccine. The recommended dosage for children as well as for adults is two injections of 1.0 cc. each. The interval for optimal immunization is not known, but one week between injections is suggested.

The results of treatment of sudden deafness have, all in all, been disappointing; the main reason, I hope, is that most patients come late for treatment. The obvious reason, of course, is that the exact cause is not known in each case. Fowler[7] mentioned five patients with severe sudden hearing loss of the neurosensory type who had spontaneous and gratifying recoveries. This certainly was unexpected and, as Fowler states, it makes the rationale of hospitalization for intravenous injections of procaine and for heroic anticoagulation therapy, or any treatment for that matter, difficult to defend.

Fowler treated 30 patients who had hearing loss of three weeks' duration or less. Fifteen patients received both nicotinic acid, 50 to 100 mg. three times a day, and injections of 250 cc. of 0.1 per cent solution of procaine hydrochloride; three of these manifested gratifying improvement. Five received nicotinic acid alone; one was much improved. Ten received anticoagulant drugs in addition to nicotinic acid and procaine, but most of them received the anticoagulation therapy so late that it could hardly be expected to have much effect on the small blood vessels that must be involved in labyrinthine apoplexy. In two patients in whom the treatment was started early, the result was very encouraging.

Fowler gives the patient heparin for immediate effect He injects 50 to 75 mg deep into the subcutaneous tissue every six hours for five or six days He then administers dicumarol or warfarin (coumadin), which can be taken in tablet form Prothrombin time has to be checked regularly If this type of treatment is used, I suggest close supervision and guidance by the internist

Several investigators say that practically every patient having arteriosclerosis obliterans and acute vasospastic disease presents some degree of sludging and intravascular abnormality, as seen in the bulbar conjunctival vessels Most patients also appear to have a clinically significant element of vasospasm

PATIENTS STUDIED AT THE MAYO CLINIC

Previously, I reported on a study of 178 patients with sudden deafness[8] Patients whose deafness was from more or less obvious causes, such as from injuries and local infections, were not included All patients with sudden deafness, so far as my colleagues and I have seen them, have a perceptive deafness, mostly of end-organ type, with different degrees of recruitment The deafness may vary greatly from a total loss of hearing to a slight dip involving only one or two frequencies

The following illustrations present possible etiologic factors at play As you see, most of them come under the classification "vascular accident" (89 patients) or "Ménière's disease" (56 patients) I use the term "vascular accident" because it implies vascular damage but does not specify type The majority of the 178 patients studied were spread over the older age groups, corresponding to those in which coronary occlusion is common Parker and associates[9] estimated that 85 per cent of 3400 patients with angina were in the age group 40 to 69 years Of the 89 patients with deafness due to vascular accident in my series, 64 1 per cent were in this age group, however, there was one significant discrepancy 3 6 per cent of patients with angina were in the age group 20 to 39, whereas 30 3 per cent of the 89 with deafness were in this age range

This is difficult to explain, as the blood supply to the labyrinth is such that even minor vascular changes might cause early symptoms I was able to recheck 20 of the 27 patients (30 3 per cent) in the age group 20 to 39 years Three of these subsequently had vascular accidents elsewhere in the body This may or may not be significant, however, I began to suspect that early atherosclerotic changes may be responsible for the sudden deafness, at least in some patients Four patients with sudden deafness were checked more thoroughly, including determination of cholesterol, fatty acids and total lipids Below are reports on two of these patients

Case 1 A 40 year old mechanic developed sudden vertigo while firing his furnace in 1952 It was a rather severe spinning vertigo with nausea and vomiting Ten days later, he had sudden deafness in the right ear and also severe dizzy spells Then he was well until 1958 when he came to the Mayo Clinic because of pain across his chest The ears looked normal with normal hearing on the left and total deafness on the right The corneal reflexes were intact and he had no nystagmus The caloric test revealed a hypoactive right equilibratory labyrinth

The results of general examination were not remarkable The cardiac findings including the electrocardiographic picture were normal The blood pressure was 110 mm of mercury systolic and 70 diastolic The ocular fundi showed minimal narrowing and sclerosis of the arterioles Blood cholesterol measured 305 mg per 100 cc on two occasions (normal 150 to 250 mg) fatty acids 398 and 444 mg (normal 350 to 400) and total lipids 703 and 749 mg (normal 400 to 659) Because of these findings the internist thought the patient had atherosclerotic disease

Case 2 A physician aged 46 had sudden blurring of hearing on the left with roaring tinnitus on February 15 1957 The ear felt as though it had fluid in it He had a strong family history of coronary disease On examination the following day, the ears including the drum membranes looked normal The audiogram showed a sharp island loss of hearing at 2000 cps The results of general examination were negative except for rather high levels of blood fats Cholesterol measured 190 fatty acids 405 and total lipids 595 He was immediately started on treatment with a low fat diet and 200 mg of nicotinic acid given 20 minutes before meals and at bedtime The hearing returned to normal in two weeks When I talked to him recently his hearing seemed still to be normal I might add that he has decided to stay permanently on a low fat diet

RESULTS OF TREATMENT

With the sludging and vasospasm in mind, my colleagues and I concentrated more on using one of the vasodilators, and for several years nicotinic acid has been the drug of choice

At first we started with nicotinic acid tablets, 100 mg before breakfast and 100 mg before supper, on an empty stomach If primarily we have suspected that the level of blood fats might be high, we have tended to increase the dosage substantially According to Achor and Berge,[10] and later Parsons and Flinn,[11] blood cholesterol has been decreased a significant degree by using 1 gm of nicotinic acid three times daily with or after meals This is five times the usual dose Patients should also go on a low fat diet and stay on it for the rest of their lives

Other methods, now more or less investigative, are at hand, which make it possible to lower the concentration of blood cholesterol in man Favorable responses in the blood lipid pattern can be effected by a diet low in fat, by the addition of unsaturated vegetable oils to the diet, and by the administration of such agents as plant sterols, estrogens and heparin These regimens have been reported as improving the survival rate for persons who previously had had coronary occlusion

So far, the results of treatment have been very disappointing, to say the least, and one of the main reasons could be that patients come so late for treatment In such a fine mechanism as that of the end organ of hearing, irreversible changes may take place quickly

If the hearing loss has lasted more than three weeks and the hearing does not fluctuate, one must conclude that the damage to the cochlea is permanent and that treatment directed toward improving the hearing is useless

In checking over the records of these patients, it was surprising and disconcerting to see how many of them were treated for a long time with inflation of the Eustachian tube

SUMMARY

Since nothing definite is known about the etiology of sudden deafness, the results of treatment are difficult to evaluate I believe, however, that the cause in most instances is to be considered a vascular accident and that treatment should consist of large doses of nicotinic acid and a diet low in saturated fats Treatment should be directed toward the patient as a whole, since the vascular accident in the ear might be a forerunner of pathologic changes in more vital parts elsewhere in the body

In conclusion, may I say that we are still playing with the problem, like a cat with a mouse, but so far we "cats" have not yet had the pleasure of swallowing the morsel

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PHASE-CONTRAST PHOTOGRAPHY OF THE DIFFERENTIATED LIVING CELLS OF THE INNER EAR

TAWFIK GIRGIS, M D,
(By Invitation),
Chicago, Ill

(This was a Movie Presentation)

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AUDIOLOGICAL MANIFESTATIONS OF LESIONS IN THE AUDITORY NERVOUS SYSTEM*

JAMES JERGER, Ph D,
(By Invitation),
Evanston, Ill

For the purpose of this discussion, it is useful to divide the auditory nervous system into four parts, as follows

1 Cochlea
2 VIIIth Nerve
3 Brain Stem
4 Auditory Cortex

Experience gained over the past three years leads us to believe that lesions at each of these four sites are characterized by distinctly different patterns of response to a diversified battery of auditory tests There seems to be a unique patterning of response corresponding to each of these four sites of lesion

The basis for this patterning seems to be at least four-fold first, the diseased cochlea produces a number of unusual symptoms not encountered in any other part of the system. It manifests, for example, loudness recruitment, non-linear distortion, and a rather astonishing capacity to appreciate small changes in sound intensity It may be fruitful to consider all of these manifestations as symptoms of a basic *disorder in the "biological amplifier"* of the auditory system, as described by Davis

Second, the diseased VIIIth nerve produces a quite unique phenomenon encountered nowhere else in the system, a rather marked inability to maintain response under sustained stimulation During sustained stimulation by a pure tone, for

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*Read at the meeting of the Middle Section American Laryngological Rhinological and Otological Society Inc Chicago Ill Jan 18-19 1960

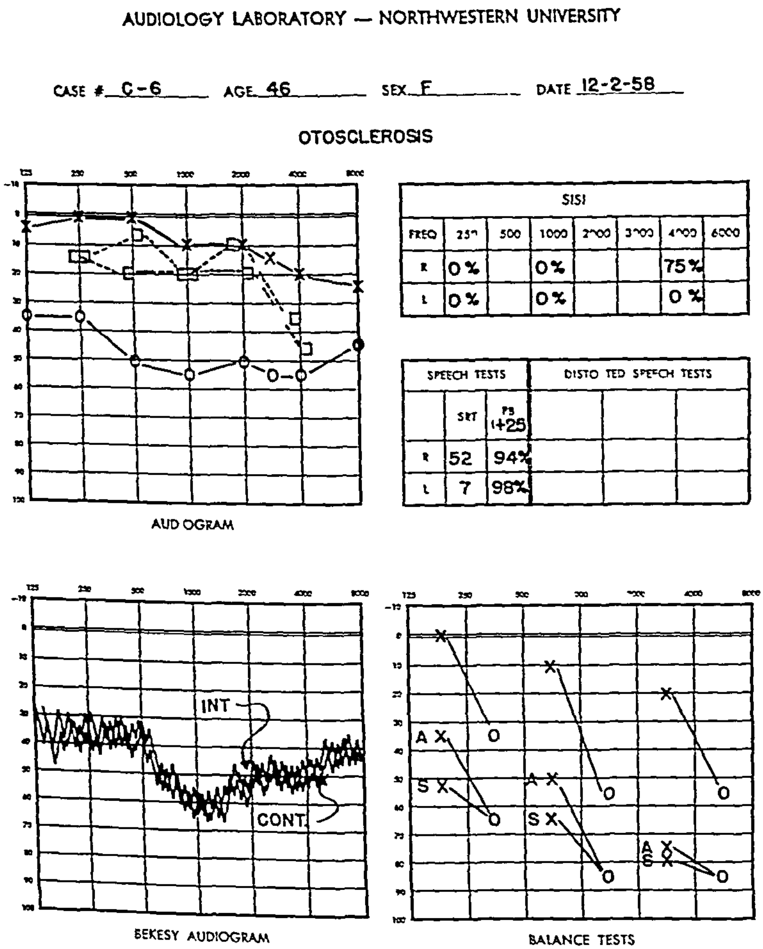
Editor s Note This manuscript received in The Laryngoscope Office and accepted for publication Feb 8 1960

example, the threshold intensity may rise 40-50 db in as little as 60 seconds. The effect is so dramatic that many of our colleagues fail to appreciate its reality until they have observed it personally. In a previous publication, we have referred to this as the phenomenon of "*abnormal auditory adaptation*." In some respects, however, the term "*relapse*" might be more appropriate.

In addition to these localized sets of symptoms, there appear to be two rather general principles. One of these states that *the subtlety of the auditory manifestation increases as the site of lesion progresses from peripheral to central*. An illustration of this principle is the audiogram itself. Thresholds for pure tones represent relatively simple judgments in any hierarchy of auditory tasks. In accordance with the "subtlety principle," therefore, they are most affected by lesions of the peripheral apparatus (*i.e.*, the middle ear and cochlea), less affected by VIIIth nerve lesion, and affected only slightly, if at all, by lesions within the central nervous system. Another way of viewing this principle is in terms of the subtlety of the auditory manifestations of lesions at progressively more central sites. At the peripheral level the presence of a lesion may be demonstrated with relatively simple tests, such as threshold for pure tones or response to isolated monosyllabic words. At more central sites, however, the presence of a lesion can often be demonstrated only by means of considerably more subtle auditory tasks.

The second general point concerns what we consider the "*bottleneck principle*." This states, in effect, that the process of transmitting the informational content of a complex auditory stimulus (*i.e.*, speech) encounters a very real bottleneck in the VIIIth nerve and lower brain stem. Lesions at these sites limit the ability to understand speech drastically. One may observe, for example, cases of VIIIth nerve tumor with a very poor speech discrimination score in the face of only slight reduction in the audiogram. Brain stem lesions may produce severe discrimination problems with *no depression* in the audiogram whatsoever.

Lesions either more peripheral or more central than the



AUDIOLOGY LABORATORY — NORTHWESTERN UNIVERSITY

CASE # C-6 AGE 46 SEX F DATE 12-2-58

OTOSCLEROSIS

SISI							
FREQ	250	500	1000	2000	3000	4000	6000
R	0%		0%			75%	
L	0%		0%			0%	

SPEECH TESTS			DISTORTED SPEECH TESTS		
	SRT	PB (+25)			
R	52	94%			
L	7	98%			

Fig 1

VIIIth nerve-brain stem region apparently have much less effect on the successful transmission of complex stimuli A possible exception to this generalization exists in the case of Ménière's disease during its active phase when speech discrimination may be severely involved

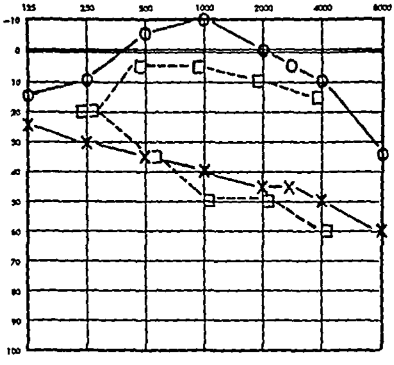
In general, however, neither cochlear nor cortical lesions seriously impair the ordinary speech discrimination score

The following five cases illustrate these principles more

AUDIOLOGY LABORATORY — NORTHWESTERN UNIVERSITY

CASE # S-8 AGE 57 SEX F DATE 5 1 59

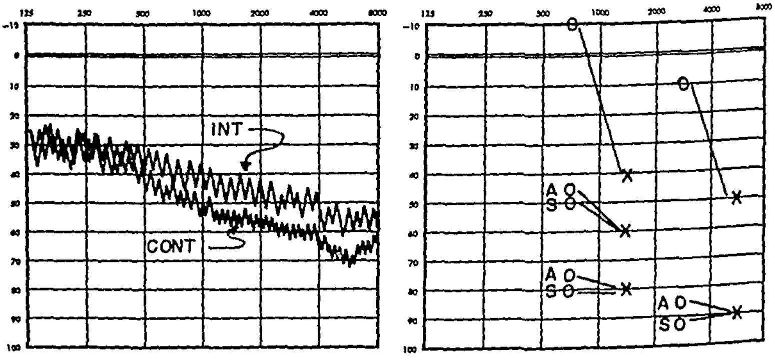
LEFT LABYRINTHINE HYDROPS



AUDIOGRAM

SISI							
FREQ	250	500	1000	2000	3000	4000	6000
R	0%		0%			0%	
L	0%		90%			85%	

SPEECH TESTS			DISTORTED SPEECH TESTS		
	SRT	PB +25			
R	3	92%			
L	45	50%			



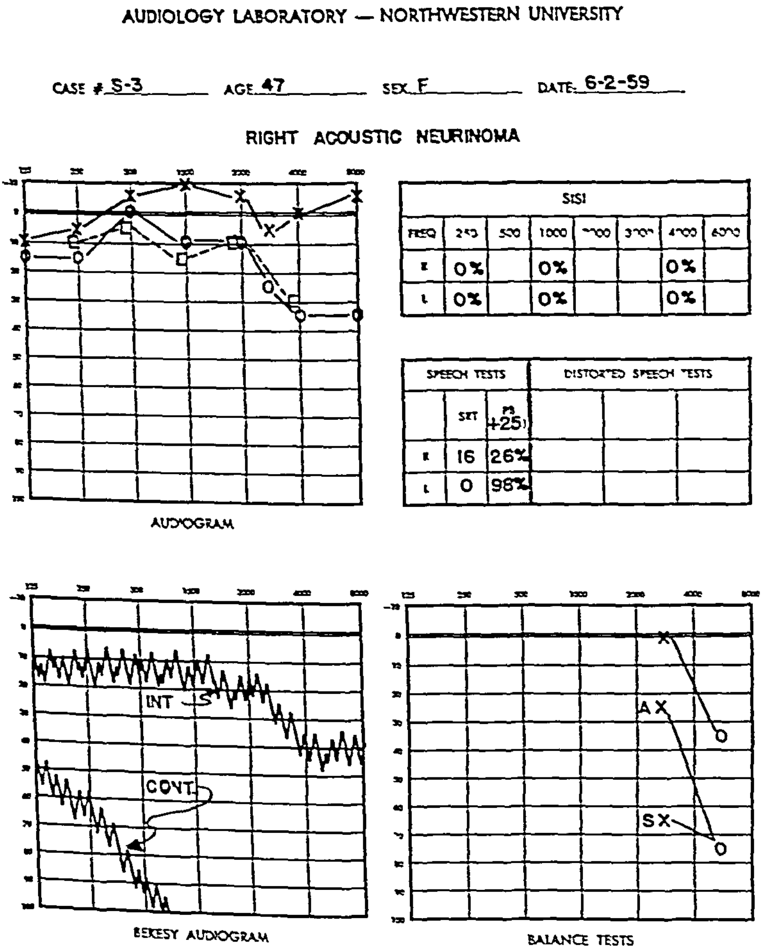
BEKESY AUDIOGRAM

BALANCE TESTS

Fig 2

concretely They represent, respectively, unilateral lesions in the middle ear, cochlea, VIIIth nerve, brain stem, and temporal cortex

Fig 1 shows auditory test results in a case of left otosclerosis Note the low SISI scores, absence of loudness recruitment, and normal Bekesy audiograms, as well as the excellent speech discrimination score The only audiological manifestation of a hearing loss is the loss in threshold sen-



AUDIOLOGY LABORATORY — NORTHWESTERN UNIVERSITY

CASE # S-3 AGE 47 SEX F DATE 6-2-59

RIGHT ACOUSTIC NEURINOMA

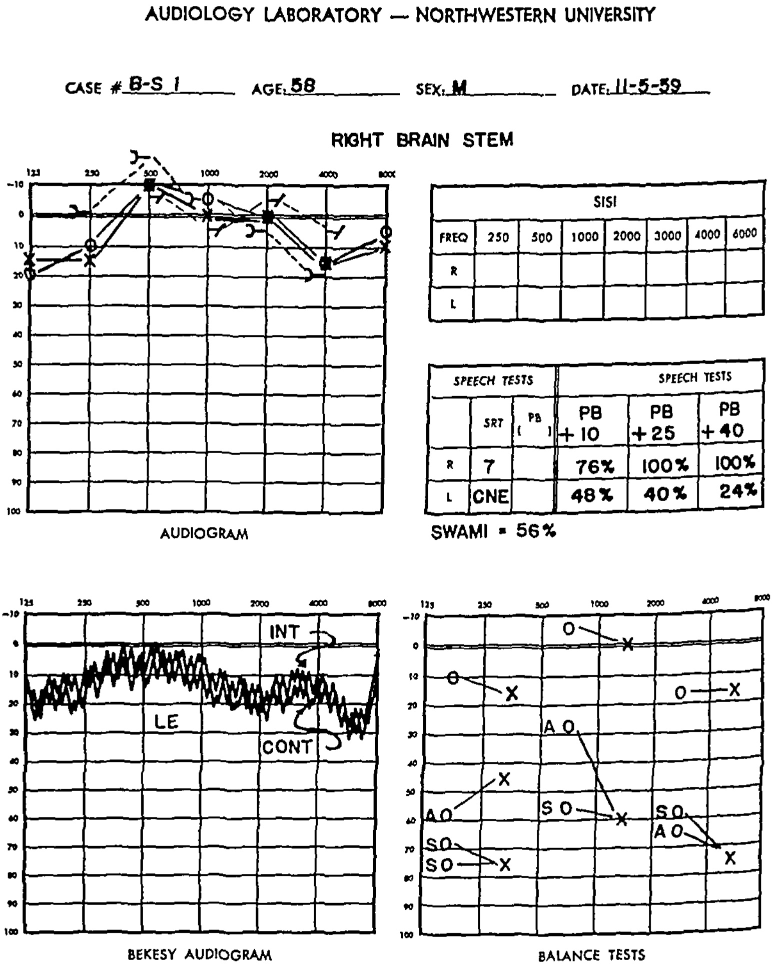
SISI							
FREQ	250	500	1000	2000	3000	4000	6000
R	0%		0%			0%	
L	0%		0%			0%	

SPEECH TESTS			DISTORTED SPEECH TESTS		
	SRT	PB (+25)			
R	16	26%			
L	0	98%			

Fig

sitivity on the affected ear On all of the other auditory tests this ear behaves exactly like a normal ear

Fig 2 shows test results in a case of unilateral Ménière's disease Now we see all the signs of a diseased cochlea, high SISI scores, loudness recruitment, and the Category II Bekesy audiogram, so characteristic of inner ear lesion The speech discrimination score is reduced, but not too much in view



AUDIOLOGY LABORATORY — NORTHWESTERN UNIVERSITY

CASE # B-S I AGE: 58 SEX: M DATE: 11-5-59

RIGHT BRAIN STEM

SISI							
FREQ	250	500	1000	2000	3000	4000	6000
R							
L							

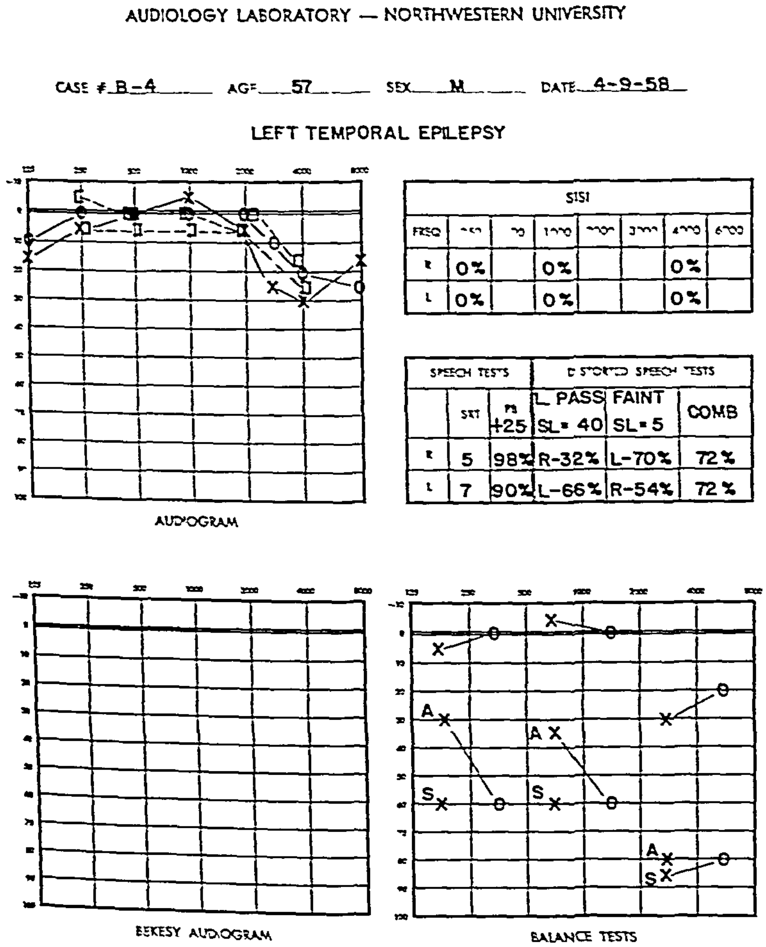
SPEECH TESTS			SPEECH TESTS		
	SRT	PB ( )	PB +10	PB +25	PB +40
R	7		76%	100%	100%
L	CNE		48%	40%	24%

SWAMI = 56%

Fig 4

of the unfortunate state of the sensory receptor mechanism In any event these three signs, high SISI score, loudness recruitment, and Category II Bekesy audiogram are unique to lesions affecting the cochlea

Fig 3 shows a quite different picture in the case of acoustic neurinoma Here the SISI score is low, loudness recruitment is absent, and the Bekesy audiogram is Category III, exemplifying the rather marked adaptation or relapse under sustained stimulation, a symptom apparently quite unique to



AUDIOLOGY LABORATORY — NORTHWESTERN UNIVERSITY

CASE # B-4 AGE 57 SEX M DATE 4-9-58

LEFT TEMPORAL EPILEPSY

SISI							
FREQ	250	500	1000	2000	3000	4000	6000
R	0%		0%			0%	
L	0%		0%			0%	

SPEECH TESTS			DISTORTED SPEECH TESTS		
	SRT	PB +25	L PASS SL = 40	FAINT SL = 5	COMB
R	5	98%	R-32%	L-70%	72%
L	7	90%	L-66%	R-54%	72%

Fig 5

lesions of the VIIIth nerve Notice, also, the astonishingly low speech discrimination score despite only a mild loss in pure-tone threshold sensitivity

Now we are in the bottleneck region, and one is led to suspect that the loss of a substantial number of VIIIth nerve fibers has a much more profound effect on the capacity to transmit speech than on the capacity to transmit faint pure tones

Fig 4 shows test results in a case of right-sided brain stem lesion consequent on vascular insult Pure tone thresholds are relatively normal on both ears, but, on the ear opposite the affected side of the brain stem, the ordinary speech discrimination score is quite low Now this is really a very remarkable situation, a markedly reduced speech discrimination score in the face of a quite normal audiogram, and the defect shows up on the opposite side only

Finally, in Fig 5, we see a case of left temporal epilepsy Here, both pure-tone thresholds and speech discrimination scores are quite normal There is really no evidence of a "hearing loss" in the ordinary sense The involvement of the

	Relative Subtlety of Auditory Task		
Site of Lesion	Pure-tone Acuity	PB Discrim	Distorted Speech
Middle Ear	P	O	O
Inner Ear	P	S	—
VIIIth Nerve	S	P	—
Brain Stem	O	P	—
Temporal Cortex	O	O	P

O = Not affected P = Primarily affected S = Secondarily affected
— = Cannot be assessed

Fig 6

auditory system can be demonstrated, but only if the auditory task is made sufficiently difficult One method is to pass speech through a low-pass filter before delivering it to the patient's ear This has the effect of attenuating the consonantal sounds relative to the vowels and makes the speech "muffled" and rather difficult to understand In this auditory task the unilateral defect shows up quite clearly The discrimination score is now considerably lower on the ear opposite the affected temporal lobe

These cases illustrate what we believe to be the salient audiological manifestations of lesions in the auditory nervous system At the risk of oversimplification, it seems to us that the entire picture is quite orderly

Fig 6 summarizes our present view We believe that the auditory manifestations of lesions at various sites within the auditory nervous system are rather meaningfully related to the subtlety of the auditory demands made by various kinds of tests

Acuity for pure tones, for example, is maximally affected by peripheral lesions and progressively less affected as the site of lesion moves centrally Ordinary speech discrimination is maximally affected in the bottleneck region of the VIIIth nerve and brain stem, less affected by cochlear lesion, and not at all by either middle ear or cortical lesion

Finally, at the extreme central level only the ability to perform relatively subtle and difficult listening tasks is demonstrably affected

The principles and generalizations presented here are based on very incomplete evidence They should be interpreted as guideposts for further research rather than fully documented conclusions

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SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY OF SOUTHERN MEDICAL ASSOCIATION

The Section of Ophthalmology and Otolaryngology of the Southern Medical Association announces that they are now accepting papers by physicians of either specialty living in the area of the Southern Medical Association for consideration for presentation at the next annual meeting to be held in St Louis, Mo , from October 31 to November 3, 1960

The paper or an abstract of the paper should be sent directly to the Secretary, Dr Albert C Esposito, Suite 1212, First Huntington National Bank Building, Huntington, W Va , as soon as possible

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Finally, in Fig 5, we see a case of left temporal epilepsy Here, both pure-tone thresholds and speech discrimination scores are quite normal There is really no evidence of a "hearing loss" in the ordinary sense The involvement of the auditory system can be demonstrated, but only if the auditory task is made sufficiently difficult One method is to pass speech through a low-pass filter before delivering it to the patient's ear This has the effect of attenuating the consonantal sounds relative to the vowels and makes the speech "muffled" and rather difficult to understand In this auditory task the unilateral defect shows up quite clearly The discrimination score is now considerably lower on the ear opposite the affected temporal lobe

	Relative Subtlety of Auditory Task		
Site of Lesion	Pure-tone Acuity	PB Discrim	Distorted Speech
Middle Ear	P	O	O
Inner Ear	P	S	—
VIIIth Nerve	S	P	—
Brain Stem	O	P	—
Temporal Cortex	O	O	P

O = Not affected P = Primarily affected S = Secondarily affected
— = Cannot be assessed

Fig 6

These cases illustrate what we believe to be the salient audiological manifestations of lesions in the auditory nervous system At the risk of oversimplification, it seems to us that the entire picture is quite orderly

Fig 6 summarizes our present view We believe that the auditory manifestations of lesions at various sites within the auditory nervous system are rather meaningfully related to the subtlety of the auditory demands made by various kinds of tests

Acuity for pure tones, for example, is maximally affected by peripheral lesions and progressively less affected as the site of lesion moves centrally Ordinary speech discrimination is maximally affected in the bottleneck region of the VIIIth nerve and brain stem, less affected by cochlear lesion, and not at all by either middle ear or cortical lesion

Finally, at the extreme central level only the ability to perform relatively subtle and difficult listening tasks is demonstrably affected

The principles and generalizations presented here are based on very incomplete evidence They should be interpreted as guideposts for further research rather than fully documented conclusions

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SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY OF SOUTHERN MEDICAL ASSOCIATION

The Section of Ophthalmology and Otolaryngology of the Southern Medical Association announces that they are now accepting papers by physicians of either specialty living in the area of the Southern Medical Association for consideration for presentation at the next annual meeting to be held in St Louis, Mo , from October 31 to November 3, 1960

The paper or an abstract of the paper should be sent directly to the Secretary, Dr Albert C Esposito, Suite 1212, First Huntington National Bank Building, Huntington, W Va , as soon as possible

NEUROVASCULAR DYSFUNCTIONS OF THE EIGHTH NERVE*†

SAM E ROBERTS, M D ,

Kansas City, Mo

My presentation in the workshop today is entirely clinical It is not statistical as there were no controls It is a metabolic study in which *hypometabolism* will be stressed Acoustic trauma, bacterial, viral and other etiologic factors have been excluded Opinions and conclusions are hypothecated upon the investigations of 157 patients with adequate physical, serologic and other examinations

Positive statements obviously cannot, and should not, be made until clinical and basic research are better correlated

Historians of otology will probably record the years of 1950 to 1960 as the decade of greatest surgical achievement on the bony capsule of the labyrinth

Cardiovascular history will, I am sure, also record that in this same period more has been done to crystalize our knowledge of cardiovascular diseases than in all the preceding centuries We must recall that James B Herrick of Chicago made cardiac history when he gave his first complete clinical description of coronary occlusion as recently as 1912 This was 254 years after Wepfer, a Swiss physician, demonstrated the fact that apoplexy is due to cerebral hemorrhage

I believe the next ten years, 1960 to 1970, will show a significant advance in the understanding, prevention and treatment of neurovascular dysfunctions of the entire VIIIth nerve We are now in the thinking stages only

Such men as Fowler, Sr , Fowler, Jr , Hallberg, Kimura,

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*Read at the meeting of the Middle Section of the American Laryngological Rhinological and Otological Society Inc Chicago Ill Jan 18 1960

†From the Department of Otolaryngology University of Kansas Medical Center Kansas City Kans

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Perlman and many others are doing superior jobs of creative and provocative thinking on this subject

Fowler, Sr ,[1] for example, when discussing presbycusis states, "the basic causes in both the young and the aging are similar insofar as they are dependent upon a disordered metabolism, particularly a disordered oxygen supply" He further emphasizes, "even when there appears to be no general hypometabolism or hypothyroidism there may be local hypometabolism"

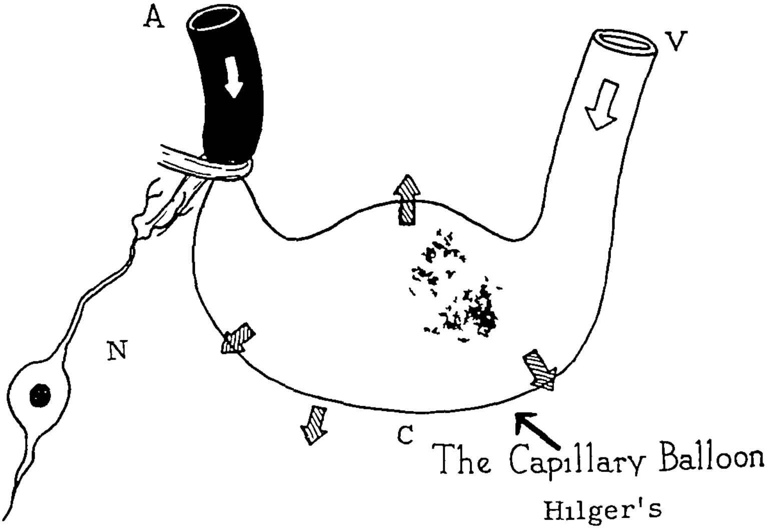
We have far too long attempted to visualize otologic pathology through a tiny speculum When we are able to construct mentally an ear speculum the size of the human body, otology will advance even more rapidly

Since there is no definitive laboratory proof of an organic lesion in hypometabolism, the clinical diagnosis depends entirely upon the awareness of the physician and a careful and thoughtful history The history usually reveals a vague borderline state of ill health Symptoms are often so bizarre and puzzling even to the erudite physician that these unfortunate patients will be told, "there is absolutely nothing physically wrong with you," leaving the distinct connotation that the condition is nervous or mental This often starts the trek to our over-crowded institutions for nervous and mental illnesses

Fatigue is a common symptom of these patients who suffer complete exhaustion by mid-day due to hypoglycemia The fact that these persons frequently have had no breakfast, or an inadequate breakfast, renders them hypoglycemic before they start the day's activities!

Hypometabolism chiefly affects three classes of people first, business men and women whose occupations are largely sedentary and primarily mental, second, persons with poor eating habits (this includes overindulgence in beverages and foods at the evening meal, when the body requires the least energy-producing foods Such eating habits tend to produce an undesirable, and often a dangerous obesity, which may occur even in the very young), and, third, chain smokers and those getting very little, if any, exercise—active or passive

E J Fowler, Jr ,[2] states, "Blood sludge has been observed in many instances coincidentally with disorders of the ears and in this author's opinion it is frequently involved in the etiology of tinnitus, vertigo and deafness"



Arteriolar spasm and its sequelae

Fig 1 Hilger's mechanism of arteriolar spasm The transudation between endothelial cells results in extravascular fluid The latter compresses lymph capillaries and localized stasis causing sludged blood A—artery V—vein C—capillary N—nerve

I am indebted to Jerome Hilger[3] for a graphic illustration of sludged blood (see Fig 1)

Hallberg[4] points out, "The most common cause of sudden deafness is thought to be vascular accidents and that these lesions may consist of thrombosis, embolism, spasm, sludging or other conditions of/or in the vessels carrying blood to the labyrinth"

Kimura and Perlman[5] in their basic investigation of arterial obstruction of the labyrinth conclude, " one hour after

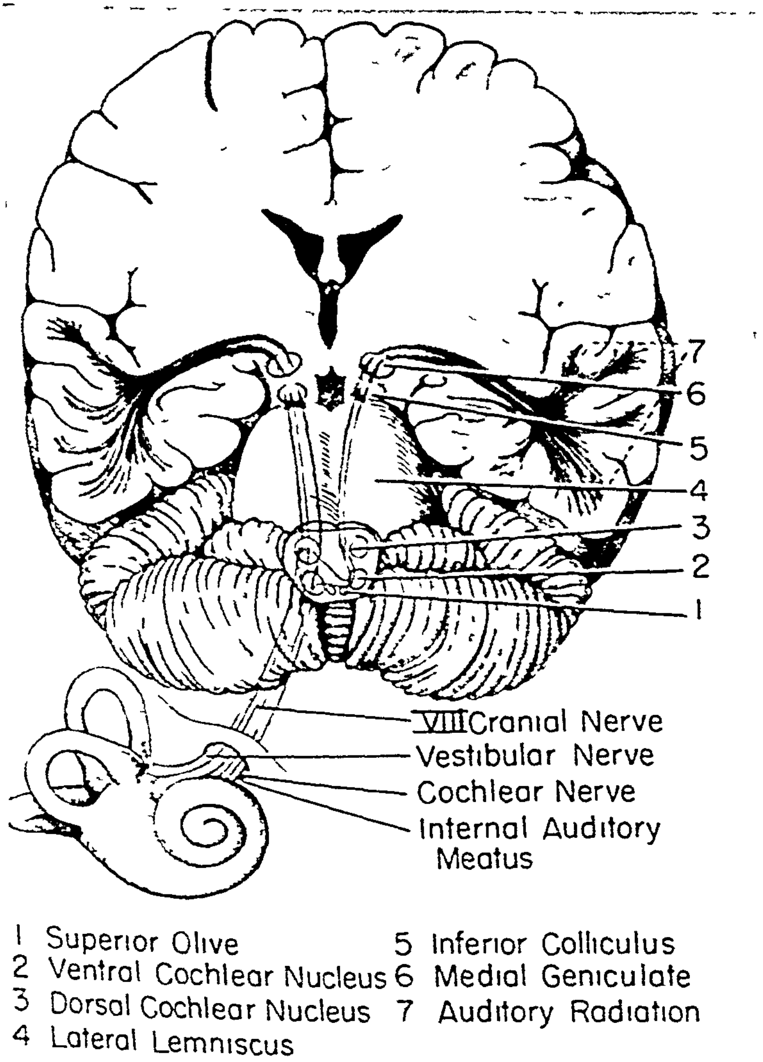


Fig 2 VIIIth nerve pathways where neurovascular dysfunctions are likely to occur

arterial obstruction the entire cochlear nerve stained poorly ", "Myelin droplets appeared in four days ", "This experimental arterial occlusion produced a severe localized brain stem lesion "

My impression, clinically derived, is that vascular dysfunctions and their sequelae, such as anoxia, are the most common cause of all chronic dysfunctions of both divisions of the VIIIth nerve These dysfunctions may occur in the labyrinth, in the central pathways, in the associated nuclei or in the control areas in the brain stem extending to the cerebral cortical terminations (see Fig 2) Another impression, also clinically derived, is that progressive VIIIth nerve degeneration could be nicely explained on the basis of slow deterioration of myelin sheath of the VIIIth nerve and, later, axon destruction

Dennis and Calkins[6] reported a case of total blindness with bilateral optic nerve demyelination (Devic's disease) It seems reasonable by comparison that the VIIIth, another cranial nerve, could be affected in the same manner (see Fig 3)

What affects the myelin sheath in one part of the body most likely affects it elsewhere

The preganglionic fibers of the autonomic nervous system are also myelinated This could be at least one cause of autonomic imbalances, which control the entire vascular system

HISTOANATOMY

Shambaugh[7] was among the first to demonstrate the distribution of blood vessels in the labyrinth (see Fig 4)

Williams[8] has shown a schema of the histoanatomy of the uncoiled cochlea (see Fig 5)

HISTOPATHOLOGY

Vascular histopathology is essentially the same in all parts of the body It matters not whether the pathology is in the

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*This affords an admirable opportunity for a joint comprehensive clinical research program The occulist can visualize the neurovascular changes in the fundus the otologist is able to measure the destructive changes in the acoustic nerve

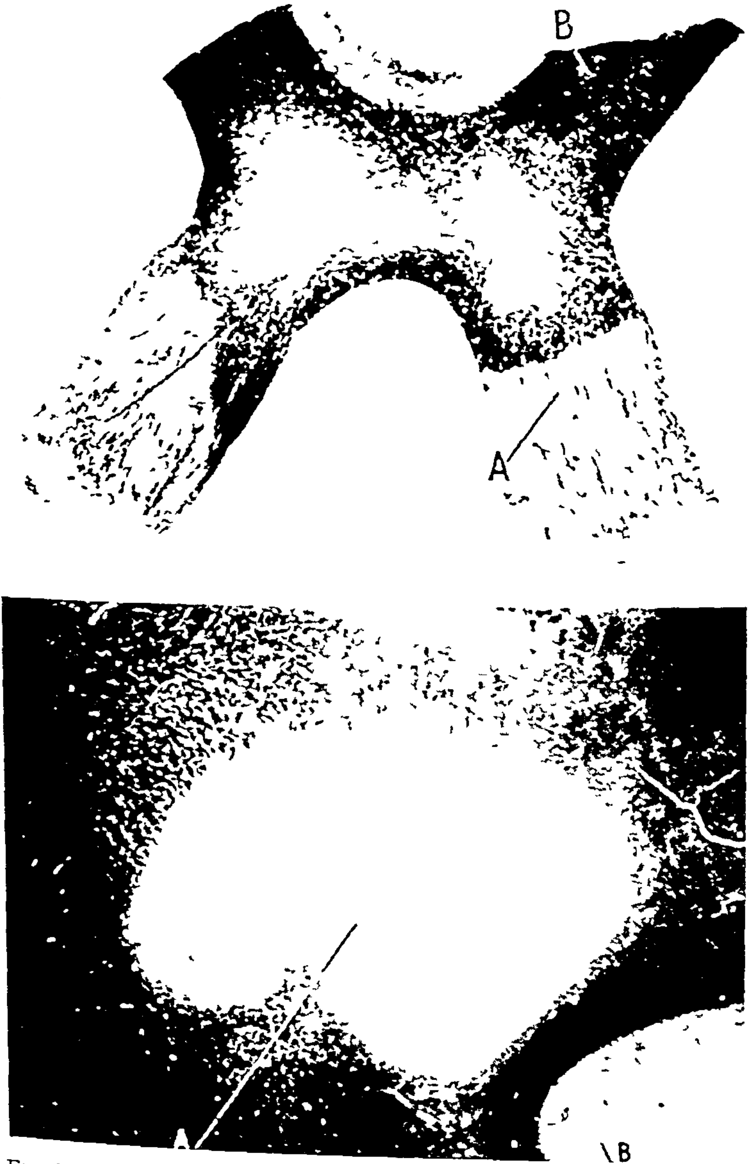


Fig 3 Optic chiasm showing demyelination of optic nerves (A) The optic tracts are not involved (B × 8) (Dennis and Calkins)

DECENNIAL PUBLICATIONS X PLATE X

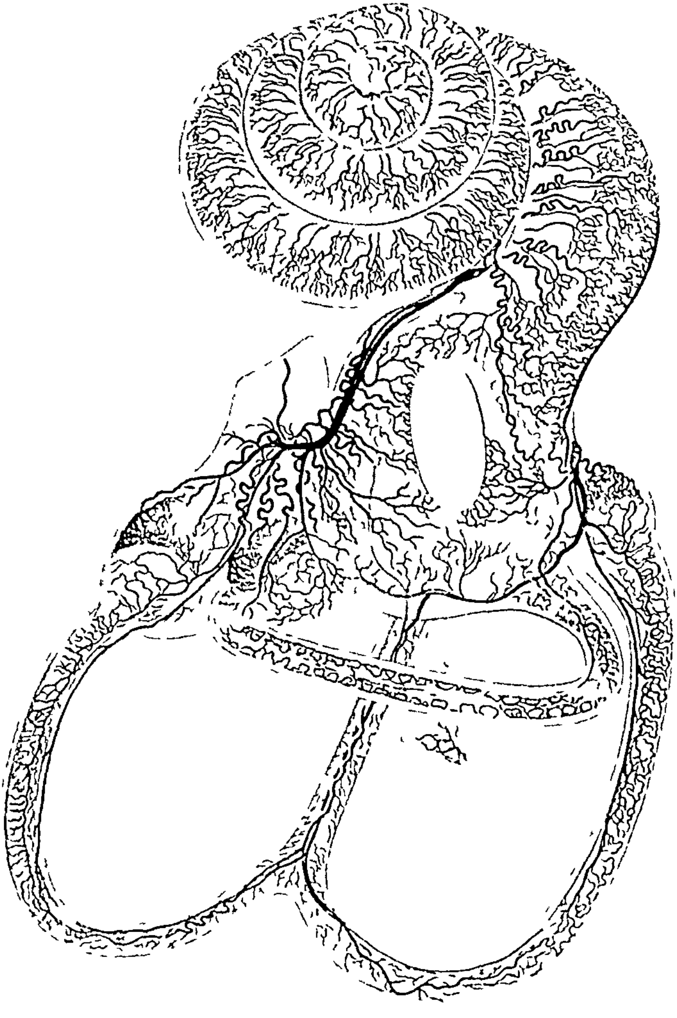
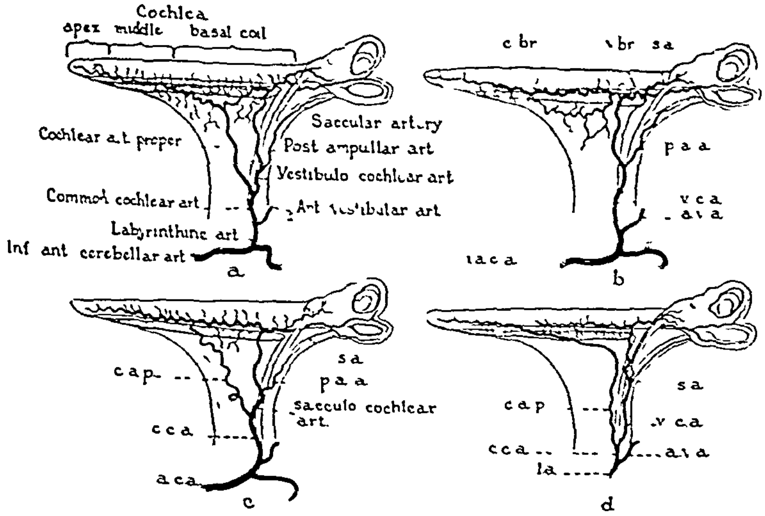


Fig. 1 Labyrinthine Circulation (Geo S Shambaugh Sr)

tiny vessels of the labyrinth, not as large as a human hair, or in the larger cerebral or coronary arteries

Since neuro-histopathology of the VIIIth nerve after it leaves the labyrinth is almost nonexistent, demyelination (diffuse or patchy seems a reasonable postulation *



Distribution of blood supply *a, b,* and *c,* according to Nabeya, *d,* Siebenman's schema The branches of the anterior vestibular artery are not shown

Fig 5 Blood supply of the labyrinth (From Henry Williams Meniere's Disease Chas C Thomas 1952)

Whenever neuro-histopathology of the general nervous system is reported there is much ambiguity in terminology The use of such terms as "slight increase in cellular element," "proliferation of cells" and "exudates" are common, but the term most frequently used is simply "swelling" or "edema "

INCIDENCE

Alvarez[9] points out that neurovascular accidents are the most common disease of man He is supported in this opinion by Dr. James Kernohan,[10] pathologist of the Mayo Clinic Since the neural elements of the labyrinth with its terminal circulation and the entire VIIIth nerve with its myelin sheaths are all extremely sensitive to, and expressive of, ISCHEMIC changes and edema, the incidence of neurovascular accidents in this region is, therefore, probably even greater than elsewhere in the body

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*This furnishes an excellent opportunity for basic research

SUSCEPTIBILITY AND ETIOLOGY

Susceptibility of neurovascular dysfunctions can no longer be regarded strictly as a disease of the aging or aged Millman[11] reports that "More than 75 per cent of a group of 500 American soldiers killed on the battlefields of Korea showed at autopsy unmistakable signs of coronary disease Their ages ranged from 19 to 23 Against this shocking total there was only a mere 11 per cent among the Koreans and Orientals who died under identical field conditions"

Susceptibility is divided into seven groups

1 Inhabitants of the Western nations, mainly in the U S A, who consume large amounts of animal fats along with other heavy foods and are much more prone to vascular dysfunctions than the Orientals who consume mostly the plant fats and live much of their lives on a near starvation diet

2 Those who are most susceptible, however, in all geographic areas are the obese of *all* ages† with hypercholesteremia and diastolic hypertension (see Figs 6 and 7)

Behnke[12] has illustrated graphically the various physical types

A nutritional paradox exists in this country In this land of plenty, even of the burdensome surpluses of good food, we are probably one of the poorest fed *overfed* nations on earth On the patient's chart we designate this as overfed and undernourished (O F.U N)

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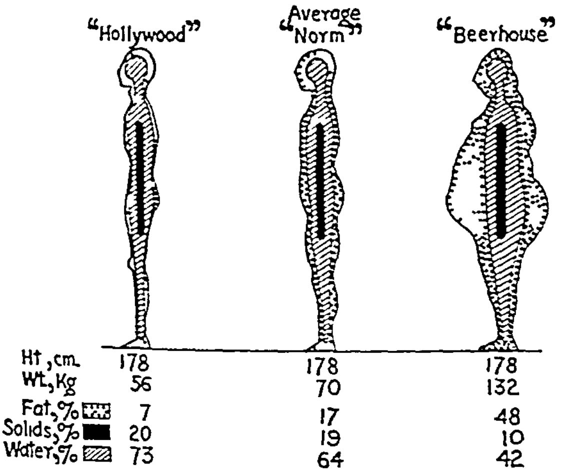
*Animal (saturated) fats come from all kinds of meat especially pork hamburgers weiners canned meats gravies commercial frying fats and canned soups Animal fats also come from butter whole milk cream cheese (except cottage cheese) and eggs

Plant (unsaturated) fats in high concentrations are present in corn oil (Mazola) soy oil wheat germ oil cotton seed pecans peanuts walnuts and other nuts Other common food sources of unsaturated fats are olives coconut avocado sweet potato winter squash and nearly all cereals

†I feel that juvenile hypometabolism is one of the specific causes of juvenile delinquency in both boys and girls These children are physically ill frequently social outcasts often downright hostile to parents and friends and at times brutally vindictive

It is not uncommon to find many young executives and employed persons, as well as teenagers, whose breakfast consists only of coffee and a sweet roll, or only coffee and numerous cigarettes These persons are naturally exhausted by



"Nature finally dictates slimming down--Beerhouse and his brethren"

Behnke, A R , Jr.,

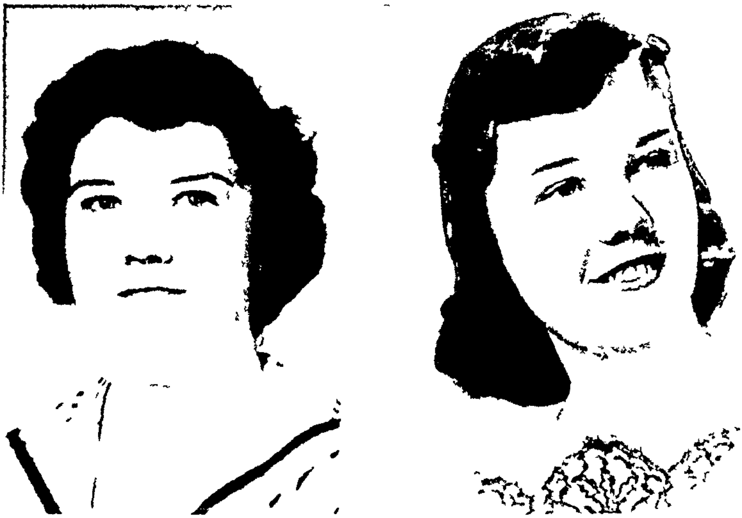
Fig 6 Beerhouse by Behnke Hypometabolism due to obesity in the older persons

mid-morning due to hypoglycemia so they start going to the coke machine and candy bar This gives them a slight temporary lift but also increases the hypoglycemia At the evening meal, as a consequence, they are starved and tend to overindulge in food, rich dessert, and beverages producing a dangerous obesity

3 Individuals (chiefly males) who have peptic ulcer and have been forced to ingest large quantities of whole milk or half milk and half cream, which, of course, predisposes strongly to hypercholesteremia

4 Males until middle life, thereafter, in postmenopausal or oophorectomized females, the susceptibility becomes more equally divided This strongly suggests a gonadotropic genesis

Masters and Ballew[13] have shown there is no significant reduction in the function of the pituitary, adrenal, thyroid or pancreas during aging—even including the eighth and ninth decades of life The gonads are alone in their obvious and well-substantiated retrogression during the fifth and sixth decades of life



Susan age 14 May 1957

Susan age 15 Feb 1958
less 40 pounds

Fig 7 Hypometabolism due solely to obesity in the very young

Page,[11] former president of the American Heart Association, humorously points out that while the sex factor does not seem to have been invented primarily with the intent of preventing women from developing vascular dysfunctions until after menopause it seems to be working out that way

5 Persons with autonomic imbalances, those with a domi-

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*This suggests strongly that gonadal substitution therapy may be indicated from the climacteric (male and female) to the grave nant sympathetic autonomic nervous system—the get-it-done type

6 Persons under stress, emotional reactions pre-disposing to vasospasm and its sequela

7 Individuals with hereditary background of neurovascular dysfunctions

SYMPTOMS AND DIAGNOSIS

Otologists are frequently consulted before serious neurovascular damage has occurred elsewhere This is often due to an annoying tinnitus or just an occasional ping in the ear A mild vertigo is frequently the reason for an ear consultation There is no other place in the human body where a slight vascular dysfunction such as a vasospasm, a tiny embolus or a thrombosis can so quickly cause minor symptoms or, at times, such a great calamity!

Again, careful and thoughtful history is of great importance in the early diagnosis of neurovascular dysfunctions of the VIIIth nerve Alvarez[2] has repeatedly pointed out that the physician often neglects to inquire and the "patient often fails to tell the physician of a bad dizzy spell at the start of his trouble" Tinnitus of a roaring, pounding or pulsating type and wedging as shown on many audiograms are at least presumptive evidence of a primary vascular narrowing or obstruction

I believe the violent seizures of Ménière's disease are just as diagnostic of gradual narrowings or occlusions of the blood vessels supplying the VIIIth nerve as the pain of angina pectoris is to the narrowing or occlusion of one or both of the coronary arteries

The *flashing of bright lights* in the outer canthus of the eye occurs less often but it is of significant diagnostic importance

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*There are now five mammalian genetic laboratories in the U S A devoted to the study of genes While many agents are involved in this investigation the endocrine glands seem to be under constant scrutiny Someone has pointed out that misplaced egotism will probably be the biggest obstacle to applying eugenics

Only a few routine, inexpensive laboratory procedures are required Roentgenograms are useless

THERAPY

The causes of any dysfunction or any pathologic state are the sum total of all conditions required to produce it, therapy, therefore, must be directed at the causes

Regarding treatment, Fowler, Sr ,[1] stresses, "It is important not to be dogmatic in these matters, to say it can't be done Keep trying with newer and newer ideas, old definitions and old concepts are not enough " Therapy is divided into three categories nutritional, the use of pharmaceutical agents and the correction of faulty habits The dietary treatment is extremely simple, only a question of specific patient instructions

Nutritional

1 Weight reduction when indicated which, of itself, lowers serum cholesterol,

2 Dietary manipulation reduction of all fats, especially animal (saturated) fats and substitution of plant (unsaturated) fats (see Fig 8) ,

3 Substitution of seafoods for the main meat course several times a week because they are low in fat, have a high iodine number and abound in minerals Otherwise, lean meats, cottage cheese and other protein as desired,

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* All treatment must be individualized and subject to change from time to time except the nutritional phase which remains constant until newer scientific knowledge is available

A completely fat-free diet is not only impractical but dangerous Both fats are used physiologically mainly as a source of immediate and remote energy Patients who without medical advice placed themselves on a fat-free diet continued to gain weight and lose strength The addition of fats to their diet especially in the form of plant fats entirely reversed the process

Moderation and balance in everything should be the motto Too many factors and regulators of metabolism are still unknown to warrant tampering with a well balanced diet Vegetable fat should be emphasized in most American diets but not to the entire exclusion of animal fats

Further information concerning the development of this regimen stems mainly from the teaching of Henry A Schroeder of Washington University Mechanisms of Hypertension published by Chas C Thomas Springfield Ill 1957 the various writings and personal communications of William H Masters of Washington University Seymour H Rinzler of Cornell University and author of The Clinical Aspects of Arteriosclerosis published by Chas C Thomas Springfield Ill 1957 (p 29) and my own experiences, Ear Nose and Throat Dysfunctions Due to Deficiencies and Imbalances published by Chas C Thomas Springfield Ill 1957

4 Elimination of refined sugar and substitution of artificial sweetening (Jelsweet) Wilder and Keys[15] have shown "Sugar only supplies calories, it has no other nutritional value, and the vitamins provided by other foods are sapped by sugar in liberating these calories"

Woolley[16] points out " sugar can be shown to be antagonistic to the action of thiamine, since the thiamine requirement of an animal may be increased by raising the carbohydrate content of the diet The reason carbohydrates, which are not antimetabolic, can be adequately supplied by baked potatoes, whole wheat bread, cereals, fresh fruits etc Their carbohydrate content is more slowly digested than refined sugar—thereby keeping up a constant trickle of sugar into the blood stream and preventing hypoglycemia

5 Elimination of all sweet desserts and beverages The reason persons who regularly consume sweetened desserts fail to include in their diet fresh fruits (three large servings each day are desirable) with a high content of vitamin C and minerals* And,

6 One or two raw vegetable salads daily, luncheon and supper Cooked vegetables as desired (see Figs 8 and 9)

Pharmaceutical †

Massive doses initially of B complex, especially B_6 and B_{12}, parenterally, Betalin Complex F C, (Lilly), and orally Optilets-M (Abbott), and others depending later on adequate nutrition to supply necessary vitamins and minerals

Estrogen-Androgen Balance These potent anabolic agents, estrogen and androgen, in a balanced combination, so well presented by Masters[18] are employed in all patients, male or female, middle life or past Masters points out that the probable synergism of these combined steroids permits smaller doses of each to achieve the same physiologic effect This is probably due to protein anabolism, maintenance of nitrogen and phosphorus balance, as well as body electrolytes Neither masculinization nor feminization with increased hirsutism was observed in this study

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*Vitamin C deficiency produces a disturbance in the formation and maintenance of the connective tissue of the body which supports all the blood vessels and vital organs Obviously therefore a defect in this tissue could be responsible for certain allergic hemorrhagic and other types of blood vessel and nerve diseases throughout the entire body which so far remain unsolved

Neither man monkey nor guinea pigs can synthesize vitamin C and most likely cannot store it

†Details of the uses of most of these pharmaceutical agents have been previously reported by me[1]

Gevrine (Lederle) and Mediatric (Ayerst) seem to be the

RELATIVELY LOW SATURATED FAT AND LOW CALORIC DIET

Food	Permitted	Avoid
BEVERAGE	Coffee tea skimmed milk buttermilk tomato juice and fruit juices	Whole milk cream limit or avoid alcoholic beverage and beverages prepared with egg yolk or fats
BREAD	Whole wheat Staff Pepperidge not more than 2 slices daily—Krinko is excellent	White bread or biscuits unless fortified flour is used Breads prepared with egg yolk such as corn bread muffins butter crackers
CEREALS	Wheat germ Kellogg's Special K Quaker Oats Add 1 or 2 teaspoons of Lecithin (Leciphos) to all of these cereals	Highly milled (not whole grain) noodles
CHEESE	Low fat cottage	All others
EGGS	2 or 3 weekly poached or boiled (Fry or scramble in Mazola Oil or soy bean oil)	Fried or prepared in any other form
FAT	Vegetable such as Mazola safflower or soy in salads and cooking Nuts	All others especially animal fats
MEAT	Lean 4 oz ¼ pound once or twice daily or poultry (Remove all visible fat)	Pork hamburger wieners etc gravy canned meats
FISH	Broiled roasted (remove all visible fat) Fish all kinds Shell fish are superior	
POTATO	Baked 3 each week (jacket contains valuable minerals)	Fried or potato chips
SOUP	Vegetable oyster prepared fresh with skimmed milk Meat soups only if fat is skimmed off at icebox temperature	Soups and sauces made from animal fat and all canned soup except Campbell s bouillon and beef
BUTTER	Spreads made from plant fat not more than 2 or 3 pats each day unless otherwise instructed	Ordinary butter and hardened margarine
DESSERT	Fresh or unsweetened frozen fruit or juices berries or melons Use Jelsweet for all sweetening	All sweet dessert (See Hidden Sugars)
VEGETABLE	All green leafy red and yellow Add Brewer s yeast	None

NOTE Wheat germ Lecithin vegetable oils and Brewer s yeast (Thompson s) may be purchased at all Natural Food stores and at many food markets

MEAL PLAN

Breakfast	Dinner	Supper
Citrus fruit	Fruit or juice	Low fat meat—fish or cottage cheese mixed with whole lemon and/or onion
Whole grapefruit or 8 oz. of fresh or frozen orange juice or 2 oranges All kinds of berries and melons or other fruit in season	Lean meat—poultry or fish Remove all visible fat (Small serving)	Raw vegetable salad French dressing
Cereal	Raw vegetable salad	Fresh or frozen fruit or berries
Fish all kinds	Beverage	Skimmed milk
Coffee or skimmed milk	Fruit dessert	
Toast—1 slice		

Sea foods or fish of some kind should be served as the meat course three times a week

Midmorning or midafternoon tomato juice fruit juice buttermilk or skimmed milk substituted for the usual coffe or coke break

All obese persons should know that they may with safety remove undesirable fat by serving only HALF portions of a variety of well-balanced high vitamin-mineral foods

Suggestion To curb that ravenous appetite A bowl of fresh fruits such as bananas oranges apricots etc with cereal skimmed milk sweetened with Jelsweet one hour before luncheon or supper You should be able to skimp or skip the regular meal

Fig 8 Instructional Card No 1 for dietary manipulation given to each patient

agents of choice Gevrine is used chiefly in the female and Mediatric in the male

Other hormones, such as thyroid extract, have been discontinued as therapeutic agents, except in the thyroidectomized patient—then only in very small dosages for a short period of time When the endocrine glands are supplied with an adequate and balanced chemical media (through nutrition), I believe strongly they will secrete their own hormones

In autonomic imbalances Bellergal Spacetabs (Sandoz) are effective stabilizers in establishing a better integration between the autonomic, glandular and vascular systems One-half tablet should be taken morning and evening for a short period of time until the other factors of hypometabolism have been corrected

Enzymes

The knowledge of enzymes is undergoing such extensive basic investigations at the present time that clinical enzymology must await more fundamental research These investigations may be a prelude, however, to an entirely new phase of medicine which will concern hypometabolism

Anticoagulants

Heparin is used in small dosages concurrently with and after complete vasodilation Laboratory control of prothrombin activity is essential

HIDDEN SUGARS IN FOOD

Eliminate all of these foods and beverages from your diet Substitute fresh or unsweetened frozen fruits and fresh fruit juices

Compiled by
Michael J Walsh, M Sc, F R I C, A I Ch E
Consulting Nutritionist, Beverly Hills, Calif

Food Item	In Ordinary Servings
Beverages—	Colas, soda pop, root beer and most carbonated beverages contain from 3½ to 5 teaspoonfuls of sugar
Cakes and Cookies—	Angel food, chocolate cake, donuts, etc, contain from 3 to 7 teaspoonfuls of sugar
Candies —	Chocolate cream, Hershey bar (small), fudge (one ounce square) contain from 2 to 4½ teaspoonfuls of sugar
Canned Fruits and Juices—	Canned apricots, peaches and stewed fruits contain from 2 to 3½ teaspoonfuls of sugar
Dairy Products—	Ice cream, ice cream cone, ice cream sundaes contain from 3½ to 7 teaspoonfuls of sugar
Jams and Jellies—	*Apple butter, strawberry jam and various jellies (one tablespoonful)* contain from one to four teaspoonfuls of sugar
Desserts (Miscellaneous)—	Apple pie, apricot pie, cherry pie, Jello and various puddings contain from 4 to 10 teaspoonfuls of sugar

Fig 9 Instructional Card No 2—*Foods and beverages to be eliminated from diet*

Vasodilators

Histamine and nicotinic acid When the situation is urgent, as in Ménière's disease and sudden deafness, histamine, usually with heparin, is definitely the agent of choice

Horton[19] points out that " histamine given by the intravenous route causes a greater increase in blood flow to the central nervous system than any other known drug" He also believes "Histamine is a drug of choice in the management of neurovascular disturbances of the VIIIth cranial nerve and is quite an effective agent in *edema* occurring anywhere in the body

Mufson[20] lists the following reasons for choosing histamine *a* It causes the most intense and extensive erythema, *b* It is safest because it is a natural body constituent, and *c* It may be used in coronary—but not in asthmatic patients

Intravenous infusions of histamine and heparin are office procedures, hospitalization is neither necessary nor desirable The infusion, however, must be administered by nurses trained in the technique and skilled in venipuncture Such skilled nurses are seldom available in general hospitals The infusions require 90 minutes each, followed by 30-minute rest periods (see Fig 10) With proper timing of appointments, a room 7½′x9′ as shown in this photograph (see Fig 10), can easily care for six patients each work day

MANAGEMENT OF FAULTY HABITS CONTRIBUTING TO METABOLIC DYSFUNCTIONS

Sleep

Adequate and restful sleep is necessary to preserve the normalcy of the neurovascular system Sleep is one of our most useful measures in the correction of even the most serious neurovascular dysfunctions

Phenergan (promethazine hydrochloride), an antihistamine, 12 5 mg, at bedtime is usually effectual for all types of insomniacs Barbiturates, of which I am a bit fearful because of their cumulative effects or addiction, should be prescribed in minimal and receding dosages Alcohol, as hard liquor in moderation, especially in the aging patient, has a mild sedative effect by its depressive action on the cortical control centers It is a quick-acting hypnotic of short duration with vasodilator activity There is no direct harm to the before supper or bedtime "toddy" to assure nights of sound sleep

Lack of Physical Exercise

Only in the most serious neurovascular dysfunctions should bed rest be prescribed and then only for a short time Muscles of action (exercise) give the blood vessels the helpful squeeze to speed up circulation

Massage and heat have a distinct therapeutic value in nearly all vascular dysfunctions A brisk early morning walk and/or a vigorous massage, by a trained masseur or masseuse with strong hands, are excellent circulatory stimulants (no gadgets or shaking tables) ! A routine morning cold sponge is also an effective circulatory stimulant

Fig 10 Intravenous histamine room

Tobacco

Circulatory disturbances caused by smoking may vary from vasospasticity of the peripheral vessels by diminishing the flow of blood to complete thromboangiitis obliterans (Buerger's disease) Roth,[21] by using a special thermometer, has shown that the temperature may fall even more than 5 3° F in the extremities

RESULTS

Nearly all patients had subjective improvement—especially in general health Many had arrestability of further functional loss Some had subjective and objective functional improvement of the VIIIth nerve, apparently not spontaneous or coincidental None was cured

SUMMARY

The important unit of medical consideration should always be the patient as a whole—the present state of his metabolism and his biologic past—not just his diagnosed disease[1] I believe the broad subject of hypometabolism is the most neglected phase of medicine, chiefly because there is no definitive laboratory proof of an organic lesion By implication it could be responsible for many other metabolic and degenerative diseases which so far remain unsolved

I concur in the conclusions of Schroeder[22] when he states, "The final answer to this prevalent disorder of our civilization cannot be predicted with certainty There is more to be learned than has been learned in spite of the advent of *successful* therapy"

If the outlined therapy is applied when indicated, it is my conviction that we do have a treatment now that is surprisingly helpful

The fundamental knowledge of the diagnosis and management is easily acquired and easily applied Certainly we may all look forward with high anticipation to substantial progress in otology during the next decade—when the medical aspects of otolaryngology will be more clearly understood We will then no longer have to give a dour prognosis to patients with VIIIth nerve lesions The excitement and joy of growing with a problem are the great rewards of research, both clinical and basic

With sufficient motivation by all those engaged in research and treatment—and such motivation has been demonstrated here today—the problem of VIIIth nerve dysfunction will be solved

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COGAN'S SYNDROME*†

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In 1945 Cogan[1] reported four cases and found a fifth[2] in the literature, of a syndrome consisting of nonsyphilitic interstitial keratitis associated with vestibulo-auditory symptoms In 1949 he added four[3] more cases to the literature The interstitial keratitis is usually bilateral, characterized by rapid remissions and exacerbations and may or may not lead eventually to diminution of visual acuity The vestibulo-auditory complaints consist of severe vertigo, tinnitus, nystagmus, ataxia and progressive bilateral sensory-neural deafness, although here, too, there may be remissions and exacerbations Complaints referring to either the eyes or the ears may herald the onset of the disease

In discussing Cogan's syndrome only those cases that meet the afore-mentioned criteria will be considered true representatives of this syndrome, therefore, cases such as that reported by Lindsay,[4] in which the patient had iritis and vestibulo-auditory symptoms, cannot be included in the group of true examples

Since Cogan's original papers were published, 14[4-12] additional cases of the syndrome of nonsyphilitic interstitial keratitis and vestibulo-auditory symptoms have been reported

Our purpose in presenting this paper is *1* to report four additional cases of the rare syndrome of nonsyphilitic interstitial keratitis and vestibulo-auditory symptoms, *2* to dem-

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onstrate, through a review of our cases and those reported in the literature, that in our opinion Cogan's syndrome is only one manifestation of a systemic disease, although most authors have considered it to be a nosologic entity, *3* to report two atypical cases of Cogan's syndrome and to review the literature on other such examples, because through a discussion of these cases we feel that we support our argument that Cogan's syndrome is not a nosologic entity, *4* to strengthen the hypothesis, through our cases and a review of the typical and atypical cases of Cogan's syndrome in the literature, that Cogan's syndrome, as previously proposed, is only one manifestation of periarteritis nodosa[8], *5* to discuss a few of the syndromes once considered nosologic entities, as in the case of Cogan's syndrome, but which were later found to belong to the group of collagen diseases, and *6* to show a trend which could indicate that all the collagen diseases are only variants of the same basic pathologic process All cases reported herein are from Mayo Clinic in the section of otolaryngology and rhinology

REPORT OF CASES

Case 1 A 20 year old university student was first seen in the Section of Otolaryngology and Rhinology of the clinic on October 24, 1950

On October 15, 1949, while she was attending Northwestern University, an upper respiratory infection had developed with headache and gradually developing photophobia Two days later it was noted that the nasopharyngitis was not clearing, also, her eyes were becoming bloodshot, and she had an episode of vertigo all of which led to her hospitalization on October 19 Within three days after her admission to the hospital, severe vertigo developed associated with loss of equilibrium, rotatory nystagmus, nausea, vomiting and bilateral tinnitus which, when loudest, was associated with temporal pain The onset of bilateral deafness was noted and was rapidly progressive At the same time, ocular symptoms became exceedingly severe, and a diagnosis of bilateral interstitial keratitis was made The severity of interstitial keratitis fluctuated greatly, and improvement or exacerbation in one or the other or both eyes often occurred within 24 hours A cough usually accompanied a flare up in her eyes

She was dismissed from the hospital after approximately one month with complete deafness of the right ear and partial deafness of the left ear She was able to manage with a hearing aid until January, 1950, when deafness became complete in her left ear also On one occasion during the winter of 1950 however although the hearing was totally absent at 7 00 P M, it had returned sufficiently by 11 00 P M so that the patient could hear the telephone ring upstairs, a dog scratching on the door and an airplane overhead She continued to have remissions and exacerbations of interstitial keratitis every few days

From October 19, 1949, to October 24 1950, extensive laboratory investigations had been performed Leukocyte counts varied from 7000 per cubic millimeter with a normal differential to 14,700 with 77 per cent neutrophils, and at no time was there any eosinophilia. The rate of sedimentation of erythrocytes (Wintrobe method) ranged from 20 to 38 mm. per hour. Urinalysis gave normal results, and values for the following factors were normal: serum proteins and albumin-globulin ratios, serum cholesterol, serum nonprotein nitrogen, carbon dioxide combining power, serum chlorides, cephalin-cholesterol flocculation test, thymol turbidity, bleeding time, clotting time, platelet count and tests on the spinal fluid including chemistry studies. Results of the following tests were negative: serologic tests for syphilis, the treponema immobilization test, agglutination tests for Brucella and Proteus OX-19, and tests of urine for porphyrins. Repeated blood cultures with the use of both the aerobic and anaerobic technique did not show growth. Cultures from the vaginal secretions, throat and lacrimal fluid were negative for pathogenic organisms. Virus studies were done on scrapings from the conjunctivas, on washings from the eyes and nasopharynx, on serum and on stools, but nothing of significance was demonstrated. Roentgenograms of the thorax, teeth, hands, feet, skull and mastoid processes did not show evidence of abnormality.

During this same year she received a variety of treatments including a course of aureomycin, a one-week course of terramycin, two courses of chloromycetin, one lasting 11 days and the other 30 days; aureomycin (duomycin) was applied to her eyes, she had one intravenous injection of typhoid vaccine, antihistaminic drugs had been used, as well as a one-week course of Cortisone totaling 1100 mg., and a two month desensitizing course of histamine. All these therapeutic measures were of no benefit, and the course of the disease remained unchanged. During the summer of 1950 the patient began studying lip reading.

On general examination at the clinic, it was noted that she had had an appendectomy in 1948. The past and family histories were otherwise noncontributory. The blood pressure was 104 mm. of mercury systolic and 68 mm. diastolic, pulse 96 beats per minute and temperature 98.4° F. She had slight ataxia, and the deep reflexes appeared hypoactive, but she would not remain for a neurologic examination.

Her vision was as follows: right eye 6/60, left eye 6/60. The ophthalmologists found edema of the corneas chiefly limited to the substantia propria in the central portions. The periphery of the corneas was clear except for large vessels deep in the substantia propria passing from the limbus to the edematous regions. Photosensitivity was mild in degree.

The audiogram showed essentially no hearing. The Kobrak cold caloric and Barany chair tests demonstrated no response from either labyrinth.

Urinalysis showed red blood cells, grade 1 (on the basis of 1 to 5 with 1 as the least and 5 as the greatest number) and white blood cells, grade 3 (on the basis of 1 to 4); the values for hemoglobin, erythrocyte count, leukocyte count and differential count and the erythrocyte sedimentation rate (Westergren method) were normal. One examination of the stool for parasites, agglutination tests for Brucella and a roentgenogram of the thorax did not show any abnormality. The roentgenogram of the skull showed evidence of slight calcification in the parasagittal meninges. The patient was given Cortisone ophthalmic ointment and dismissed on October 27, 1950, unimproved.

There is no doubt that this case is a typical example of Cogan's syndrome. The patient not only had remissions and exacerbations of the ophthalmic symptoms and signs, but she onstrate, through a review of our cases and those reported in the literature, that in our opinion Cogan's syndrome is only one manifestation of a systemic disease, although most authors have considered it to be a nosologic entity, *3* to report two atypical cases of Cogan's syndrome and to review the literature on other such examples, because through a discussion of these cases we feel that we support our argument that Cogan's syndrome is not a nosologic entity, *4* to strengthen the hypothesis, through our cases and a review of the typical and atypical cases of Cogan's syndrome in the literature, that Cogan's syndrome, as previously proposed, is only one manifestation of periarteritis nodosa[8], *5* to discuss a few of the syndromes once considered nosologic entities, as in the case of Cogan's syndrome, but which were later found to belong to the group of collagen diseases, and *6* to show a trend which could indicate that all the collagen diseases are only variants of the same basic pathologic process All cases reported herein are from Mayo Clinic in the section of otolaryngology and rhinology

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From October 19, 1949, to October 24, 1950, extensive laboratory investigations had been performed Leukocyte counts varied from 7000 per several deep infiltrations about 1 mm in diameter and in the upper portions of each cornea some distance from the upper limbus one infiltration measured about 7 mm in length and 1 mm in breadth The latter infiltrations were vascularized from above by vessels that lay in the posterior layers of the cornea The picture was typical of that described by Cogan

The patient was dismissed on February 15, 1955, with the diagnosis of Cogan's syndrome

This was a typical case of Cogan's syndrome encountered at the clinic for diagnostic confirmation The patient was not seen in a general diagnostic section, and a general history and examination were not obtained, therefore, whether or not she ever displayed any systemic manifestations throughout the course of the illness, we are unable to answer It is interesting that the interstitial keratitis had its onset during the second half of a pregnancy

Case 3 A 34-year-old married woman was first seen at the clinic on July 22 1940 In 1934 she had awakened one morning with bilateral deafness tinnitus severe vertigo, occipital headache and a generalized warm feeling After three months the deafness cleared spontaneously and the tinnitus and vertigo also ceased In 1937 the patient experienced a similar loss of hearing associated with the same complaints that had developed previously This attack lasted six months and again cleared spontaneously One year prior to being seen at the clinic she had a third episode of deafness and vertigo which had persisted up to the time of registration at the clinic The attacks of vertigo seemed to concern her more than the bilateral deafness During each episode of hearing impairment the patient had been troubled with a productive cough She had been treated with sulfanilamide prontosil and vitamin B_1 without benefit She had had a mastoidectomy on the left side in 1935 There was no history of syphilis

The patient was 5 feet 3½ inches tall and weighed 137 5 pounds her blood pressure was 130 mm of mercury systolic and 78 mm diastolic, the pulse was 106 beats per minute and the temperature 98 8° F A systolic murmur was heard at the apex of the heart She had severe bilateral sensory neural deafness as shown by the tuning fork tests She could not hear the 128-cycle fork She heard the 2048 cycle tuning fork for 15 seconds less than did the examining physician Results of the Rinne test were positive bilaterally, and those of the Weber test did not indicate lateralization She could not hear a watch with either ear

Examination of the eyes showed her vision to be as follows right eye, 6/60 left eye 6/20 There was bilateral deep scarring of the corneas ghost vascularization of old interstitial keratitis and scattered small areas of healed chorioretinitis which was more marked in the right eye than in the left eye

The results of urinalysis and the values for hemoglobin and leukocytes were normal Roentgenograms of the thorax and sinuses were normal The basal metabolic rate was minus 3 per cent The Kline Kahn and Kolmer flocculation tests gave negative results The Hinton flocculation test was doubtfully reactive and was not considered significant The patient was dismissed on July 24 1940 also had a single remission of the hearing loss. There are several findings that suggest that the disease process was of widespread distribution such as the nasopharyngitis, the headache and temporal pain, the neutrophilic leukocytosis, the elevated erythrocyte sedimentation rate and the number of red and white blood cells in the urine. It is interesting to speculate on the reason for abdominal pain leading to appendectomy in 1948. Abdominal pain with subsequent appendectomy is not uncommonly seen in periarteritis nodosa, but in this case, unfortunately, we have no detailed information concerning the findings at operation.

Case 2. A 25 year old married woman was seen in the Section of Otolaryngology and Rhinology of the clinic on February 14, 1955.

In August, 1951, bilateral interstitial keratitis had developed during the latter part of a pregnancy, and the patient was treated with Cortisone ophthalmic drops. In November, 1951, she delivered spontaneously. Approximately three months after delivery, the ocular condition had cleared spontaneously. The interstitial keratitis recurred, however, and for two months she received pyromen intravenously without any apparent benefit. She had experienced remissions and exacerbations of the ophthalmologic condition up to the time she was seen at the clinic.

In May, 1953, severe rotatory vertigo associated with nausea and vomiting and tinnitus developed. The attack lasted two weeks and subsequently the patient noticed loss of hearing in both ears. This loss had progressed gradually although on occasions she had noticed some fluctuation in her auditory acuity.

In November, 1955, the patient had been hospitalized for investigation and a letter from her home physician stated that values obtained from study of blood chemistry, blood counts, and agglutination tests for various bacterial and viral diseases were normal. Biopsy of a lymph node revealed nonspecific hyperplasia and biopsy of a muscle did not show any abnormality. A diagnosis of nonsyphilitic interstitial keratitis with bilateral deafness was made, and she was given a course of systemic Cortisone but without benefit. The dosage was not specified in the letter.

She began to wear a hearing aid in November, 1955, which was of great help at first but which gradually became of less value, so she began to study lip reading.

Her history revealed that she had had difficulty maintaining her equilibrium in the dark and when walking, and she had difficulty focusing her eyes while driving a car. The audiogram showed almost complete loss of hearing in the right ear, some hearing being apparent at 95 decibels at the 2000 frequency. In the left ear the loss of hearing followed almost exactly the same line for total loss for speech except at the 2000 frequency where it was found to be 75 decibels. There was no recognition of phonetically balanced words in the right ear, but the percentage for phonetically balanced words in the left ear rose to about 35 per cent at 15 decibels above the threshold. On the Kobrak's modification of the cold caloric test there was no response to 30 cc. of ice water in either ear. There was no spontaneous or positional nystagmus.

Ophthalmologic examination showed that the vision without correction was 6/7+ in both eyes. In the lower portions of the corneas there were protein of 64 3 mg per 100 cc found elsewhere was significant so a lumbar puncture was not done

Her vision was 20/20 for both eyes, the results of ophthalmoscopic examination were negative The audiogram showed

Frequency	125	250	500	1000	2000	4000	8000	10 000
AC—Left	10	15	20	20	25	55	0	0
AC—Right	30	30	35	30	30	65	0	0

The bone-conduction curve closely paralleled the air conduction curve in each ear Evidence of distorted hearing of the type seen in disease of the cochlear end-organ was manifested by slight recruitment of loudness in the right ear over that in the left ear, threshold of pain at 90 decibels in the right ear and 95 decibels in the left ear and 10 per cent reduction of discrimination for speech as the signal intensity was increased 15 decibels above the level of most comfortable loudness

Caloric tests showed essentially complete lack of response of both labyrinths Urinalysis showed 8 white blood cells per high power field the leukocyte count was 13 500 per cubic millimeter of blood with 84 per cent neutrophils The concentration of hemoglobin erythrocyte count and roentgenograms of the thorax and skull including Stenver s view, were within normal limits Skin sensitization tests and cytologic examination of a cervical smear gave negative results

The patient was dismissed on September 12 1958, with the diagnosis of toxic labyrinthitis

One week after her dismissal the patient discontinued the use of meticorten and subsequently noticed an increase in the loss of hearing She was hospitalized in her home town for several days, and her hearing deteriorated to complete bilateral deafness

On September 18 rotatory vertigo, nystagmus, lacrimation on the left side photophobia, ptosis and rhinorrhea developed Although she had no vertigo from that time on complaints involving the left eye and the nose persisted She was again hospitalized and treated with kenacort eye drops four times a day and with four intravenous injections of histamine without any benefit

On October 11 she woke up with a sharp "catching" pain over the lower left costal margin both anteriorly and posteriorly This pain was aggravated by deep breathing changes in position lying on her left side and walking The same day a dull ache developed in her left shoulder and extended into her neck, with nausea and vomiting which were not associated with vertigo At this time the leukocyte count was 15,000 per cubic millimeter of blood the erythrocyte sedimentation rate was 49 mm in one hour and her temperature was 101° F Thereafter she had episodes of the same pains associated with nausea On October 16 1958, she returned to the clinic and was admitted directly to the hospital On her admission the blood pressure was 120 mm of mercury systolic and 90 mm diastolic her pulse was 104 beats per minute and the temperature, 99 5° F There was tenderness over the left border of the cervical portion of the trapezius muscle the lower left ribs posteriorly and the left costovertebral angle She was still moderately ataxic

The audiogram showed the patient to be almost completely deaf She reported that she felt speech at 90 to 95 decibels but she was unable to understand speech at any intensity On delayed feedback tests there was no change in the rhythm or quality of her speech

This patient displayed dramatic fluctuations in loss of hearing. There were not many indications of a systemic disease process, those recorded being only productive cough, occipital headache and generalized warm feeling during the episodes of hearing impairment. It is impossible to say whether or not the systolic heart murmur had been present prior to the onset of the disease. As in Case 2, the history and examination, unfortunately, were not complete.

Case 4. A 25 year old married woman was first seen at the clinic on September 9, 1958. On December 3, 1957, when she was one month pregnant, a buzzing sound developed in both ears. She was given nicotinic acid and after 24 hours the tinnitus stopped. It occurred again one month later but again stopped after 24 to 48 hours. Each month she had a similar episode of tinnitus until March, 1958, when she had an attack of rotatory vertigo associated with nausea while she was in a grocery store, the attack lasted half an hour. In April she had a second similar attack of vertigo while attending a movie. The vertiginous attack persisted for three days. From this time until her delivery on July 31, 1958, she had more frequent attacks of vertigo associated with nausea and tinnitus. Her delivery was spontaneous and uneventful. On August 11 a severe attack of vertigo associated with vomiting developed. She was hospitalized and sedated with promazine hydrochloride (sparine) for 36 hours. At this time she began to notice bilateral loss of hearing and aching over both cheeks. After one week she was dismissed from the hospital. During her pregnancy she had been on a low sodium diet and from the initial appearance of tinnitus she had been taking 100 mg of nicotinic acid four times a day.

The patient's loss of hearing was progressive, and on August 28, 1958, she was rehospitalized for further investigation. At this time the results of general examination were within normal limits and neurologic examination showed that she had a mild type of locomotor ataxia. An audiometer test showed a sensory neural type of hearing loss of 55 decibels on the left and 45 decibels on the right. Response to the caloric tests bilaterally was completely absent.

The erythrocyte sedimentation rate (Westergren method) was 35 mm in one hour and the value for protein in the spinal fluid was 64.3 mg per 100 cc. Urinalysis showed 6 to 10 red blood cells and 7 to 10 white blood cells per high power field. Laboratory investigation including values for hemoglobin, hematocrit reading, leukocyte count and differential count, blood serology, fasting blood glucose, serum urea, total serum protein and albumin globulin ratio, serum sodium, serum potassium, carbon dioxide combining power, serum chlorides, a complete study of the spinal fluid, an electrocardiogram and roentgenograms of the thorax and skull, gave results or evidence that was within normal limits. She was placed on a regimen of 80 mg of prednisone (meticorten) a day for one week. After two days of therapy she noticed some improvement in her hearing. On September 5, an audiogram revealed 20 to 25 per cent improvement in each ear. She was dismissed from the hospital on September 6 and began decreasing the dose of meticorten by 5 mg each day.

When first seen at the clinic on September 9, 1958, it was noted that she had had severe urticaria during her first pregnancy. The results of neurologic examination were negative except for hearing impairment and ataxia. The neurologists did not feel that the value for spinal fluid impression that we were dealing with periarteritis nodosa, and we treated her for this condition The patient's hearing improved early in the course of the disease when she was given meticorten, her subsequent complete loss of hearing when the medication was discontinued seemed to indicate that the gluco-corticoids can be beneficial if given early in the course of the disease As in Case 2, this patient's trouble began during pregnancy

In this case the disease impressed us as one which was confined not only to the ear and eye but rather as a disease process affecting many systems, the manifestations of which were subject to remission and exacerbation and produced an unrelated picture of systemic disease not unlike that seen in periarteritis nodosa or disseminated lupus erythematosus In order to substantiate our opinion, we reviewed the cases encountered at the clinic in which the disease has been considered to be Cogan's syndrome

COMPARISON OF CASES

Of the four cases we have reported, we feel that Cases 1 and 4 tend to indicate that Cogan's syndrome is only a local manifestation of a more generalized disease process Unfortunately, Cases 2 and 3 were not thoroughly investigated and it is impossible to say whether or not more generalized symptoms and signs such as were seen in Cases 1 and 4 were ever exhibited

In a further attempt to correlate the systemic findings in our cases with any that have occurred in previously reported examples, Table I was made It shows manifestations that tend to indicate that Cogan's syndrome is only one of the manifestations of a systemic disease Table I shows the 23 reported cases and our additional four cases with symptoms, signs and laboratory findings The findings characteristic of Cogan's syndrome such as vertigo, nausea and vomiting associated with vertigo, nystagmus, loss of equilibrium, abnormal responses to caloric tests, sensory neural deafness and interstitial keratitis were omitted from Table I

Symptoms and signs that seemed to be related to the disease

Examination of the eyes showed narrowing of the left palpebral fissure The left conjunctiva was slightly hyperemic Keratitis involved a wedge shaped portion in the inferior nasal quadrant of the left cornea The cloudiness in this portion was mild and mostly stromal

The laboratory investigation included several determinations for erythrocyte sedimentation rate (Westergren method) which varied from 114 to 77 mm per hour and a leukocyte count of 11,000 per cubic millimeter of blood with a normal differential count There was no eosinophilia at any time An electroencephalogram showed a general disturbance of function without evidence of lateralization The concentration of hemoglobin, erythrocyte count, urinalysis, urinary determinations for porphyrins, determinations for calcium, phosphate, total protein, albumin globulin ratio and urea in the serum, L E clot test, serologic tests for syphilis, blood tests for Toxoplasma antibodies, an electrocardiogram, studies of spinal fluid and roentgenograms of the thorax and lumbosacral portion of the spinal column were all within normal limits

On October 19 the patient had aching pain and slight tenderness in the muscles of her right calf, but no nodules for biopsy were noted On October 21, under the assumption that this was periarteritis nodosa with Cogan's syndrome as a local manifestation, the patient was placed on a daily dose of 200 mg of Cortisone orally and 2 5 per cent hydrocortisone ophthalmic drops every two hours On October 24 the patient's muscle pains were gone, and she was feeling better She received 5 cc of milk intramuscularly on October 26, 5,000,000 typhoid bacilli intravenously on October 28 and 15,000,000 on October 30 On October 28 it was noted that the stromal infiltration of the left cornea was almost completely gone The patient's temperature had varied from 96° to 100 5° F since admission and always spiked during the afternoon

During the night of November 1, 1958, chills and cramping, burning pain developed in both of the patient's legs There was some diffuse tenderness over the leg muscles On November 2 her temperature spiked to 103 4° F and excruciating pains developed in both legs Large doses of narcotics and packing her legs in ice were required to relieve the pain After two days, when the severe pain had ceased, an electromyogram was made in an effort to find a suitable region for biopsy There was no abnormality of nerve conduction or of response to needle electrode examination and, in view of this, blind biopsy was not attempted

On November 6 it was noted that the patient's left eye was almost normal and that she had only slight discomfort in her legs Improvement continued, and she was dismissed on November 8, 1958, on 200 mg of Cortisone daily A follow up letter on November 25, 1958, revealed that she had had one flare up of the interstitial keratitis which lasted two days but that she was showing some evidence of general improvement

This case of Cogan's syndrome is unusual in that only unilateral interstitial keratitis had developed The patient displayed involvement of multiple systems with diffuse muscle aches, pains and tenderness, a pleuritic type of pain, bilateral peripheral neuritis, nasal complaints, a spiking temperature which, on one occasion, reached 103 4° F, electroencephalographic abnormalities, and several other abnormal laboratory findings

From the time of her second visit to the clinic, it was our

7	Cogan[1] (Case 6)	Appendectomy 5 mo after onset					Leukocytosis	
8	Cogan[1] (Case 7)						Leukocytosis	
9	Cogan[1] (Case 8)			Stiff neck 1 mo prior to onset			Leukocytosis	
10	Rosen[5]	Nausea, vomiting		Dull nuchal ache and tightness around neck	Cold and cough 3 mo prior to onset		Leukocytosis	Fatigue and loss of weight during 3 mo prior to onset
11	Robson[6]							
12	Lindsay and Zuidema[4] (Case 1)							
13	Lindsay and Zuidema[4] (Case 2)				Hemoptysis	Palpitation pain in chest enlarged heart		
14	Lindsay and Zuidema[4] (Case 3)							
15	Donald and Gardner[7]	Nausea vomiting	Drowsiness, 2 convulsions, EEG showed diffuse organic changes	Pain and stiffness of neck	Severe respiratory infection 2 mo prior to onset			Generalized fatigue

TABLE I

Findings in Cases of Cogan's Syndrome Other Than Those Characteristic of the Disease

Case No and Author Reporting	Symptoms Signs and Laboratory Findings									
	Gastro-intestinal Tract	Nervous system including eye and ear	Cutaneous	Lymphoid tissue spleen liver	Musculo-skeletal system	Respiratory tract	Genito-urinary tract and urinalysis	Cardio-vascular system	Blood tests	Miscellaneous data
1 Mogan and Baumgartner[2]			Profuse perspiration left side of body							
2 Cogan[1] (Case 1)				Hepatomegaly					Leukocytosis, 6% eosinophils	
3 Cogan[1] (Case 2)		Earache in left ear					Albumin (3+) and white blood cells in urine		Leukocytosis	
4 Cogan[1] (Case 3)	Ptomaine poisoning just prior to onset								Leukocytosis 5% eosinophils	
5 Cogan[1] (Case 4)						Bronchopneumonia coldsore in nose			Leukocytosis 6% eosinophils	
6 Cogan[1] (Case 5)				Lymphadenopathy 1 mo prior to onset	Stiff neck 1 mo prior to onset	Sore throat 1 mo prior to onset		Rheumatic fever 2 yrs prior to onset grade-3 systolic murmur and cardiomegaly	Leukocytosis elevated sed rate of erythrocytes 28% eosinophils	Lost 30 lbs in 8 mo prior to onset biopsy deltoid muscle negative

20 Crawford[11] (Case 1)	Nausea, vomiting, diarrhea, melena, abdominal tenderness, paralytic ileus	Frontal headaches, toxic psychosis, bilateral Babinski sign, positive Kernig's sign, cerebrospinal fluid—53 cells per cc	Erythematous rash over palms of hands	Cervical and axillary lymphadenopathy	Nuchal rigidity, generalized muscle aches and pains	Cold 1 wk prior to onset, acute pharyngitis and rhinitis	Testicular tenderness, urine—8-10 red and 3 white blood cells per high-power field, granular casts, and albumin		Leukocytosis, elevated blood sugar	Fever, necropsy—periarteritis nodosa
21 Crawford[11] (Case 2)		Headaches, neuralgia of Vth cranial nerve			Muscle and joint pains, nodules in muscles of extremities	Pharyngitis	Albuminuria (slight), casts in urine	Elevated blood pressure	Leukocytosis	Fever, biopsy—periarteritis nodosa, loss of weight
22 Crawford[11] (Case 3)	Massive hemorrhage	Occlusion of central vein with secondary optic atrophy			Joint pains	Cold(?), ulcerating lesions of mouth and throat	Albuminuria	Elevated blood pressure, systolic heart murmur	Leukocytosis, anemia, 15% prothrombin time, reversed A/G ratio	Fever
23 Quinn and Falls[12]		Severe frontal and temporal headaches			Migratory and generalized pains	Severe upper respiratory infection	Albumin (2+), myriads of red blood cells in urine	Grade-1 systolic apical murmur	Leukocytosis, anemia, elevated sed rate of erythrocytes, C-reactive protein 5 to 6+, 2 blood Kahns 1+, abnormal electrophoretic curve	Fever, biopsy skin and muscle negative, persistent lassitude and fatigue

TABLE I (Continued)

Case No and Author Reporting	Symptoms Signs and Laboratory Findings									
	Gastro-intestinal Tract	Nervous system including eye and ear	Cutaneous	Lymphoid tissue spleen liver	Musculo-skeletal system	Respiratory tract	Genito-urinary tract and urinalysis	Cardio-vascular system	Blood tests	Miscel-laneous data
16 Oliner and Co-authors[8]	Nausea vomiting, diarrhea melena abdominal pain and tenderness		Purpuric lesions, maculo-papular rash sub-cutaneous nodules	Lymphadenopathy spleno-megaly		Pleural friction rub		Heart murmur enlarged heart	Leuko-cytosis 6% eosino-phils, anemia reversed A/G ratio, blood culture—hemolytic strepto-coccus	Fever, biopsy—periarte-ritis nodosa
17 Stevens[9]		Absence of patellar reflex		General-ized lymphad-enopathy 3 mo prior to onset		Sore throat			Leuko-cytosis, 8% eosino-phils	Fever
18 Boyd[10] (Case 1)	Diarrhea	Drowsi-ness		Diffuse lymphad-enopthy, spleno-megaly hepa-tomegaly	Stiffness of neck	Cough				Fever
19 Boyd[10] (Case 2)	Diarrhea abdominal cramps	Drowsi-ness		Cervical lymphad-enopathy spleno-megaly and hepa-tomegaly 3 mo prior to onset	Pain in legs backache			Tender-ness along carotid and femoral vessels	Leuko-cytosis 6% eosino-phils	Fever biopsy 2 muscles negative

and preceded the onset of complaints involving the eyes and ears were included in Table I These included unexplained attacks of abdominal pain, lymphadenopathy and upper respiratory infections which either preceded the onset of the syndrome, with a complete remission before the eyes and ears were affected, or were the initial complaints but were still present at the onset of the characteristic features of the syndrome Whether these forerunners of the syndrome were part of the same disease, were the factors responsible for precipitating the disease, or were just coincidental occurrences, we cannot say with absolute assurance, but the evidence suggests that these early symptoms and signs are only a few of the manifestations of a systemic disease subject to fluctuation

In only five of the 27 cases (Cases 1, 11, 12 14, 25) there were no significant findings other than interstitial keratitis and damage to the VIIIth cranial nerve (see Table I) On the other hand, in 13 cases there were multiple additional symptoms, signs and laboratory findings Other findings included leukocytosis in 18 cases, involvement of the respiratory tract in 15, of the nervous system in 12, of the musculoskeletal system in 11 and of the gastrointestinal tract in ten, fever in nine, involvement of the cardiovascular system in eight and of the genitourinary system in seven, and lymphadenopathy in six

One patient died[11] and at necropsy, periarteritis nodosa was found, in two other cases biopsy yielded evidence to substantiate the diagnosis of periarteritis nodosa[8,11] In our fourth case and in three other instances[10,11] the diagnosis of periarteritis nodosa was made on clinical grounds It was Oliner and associates[8] in 1953 who first suggested that Cogan's syndrome might be only a localized manifestation of periarteritis nodosa

Of the five cases with no findings other than those characteristic of Cogan's syndrome, apparently only Cases 1, 11 and 12 were thoroughly investigated (see Table I) The report of Case 14 is short with no mention of a general history and examination, and it is impossible, as it is in Case 25, to say

TABLE I (Continued)

	Symptoms, Signs and Laboratory Findings									
Case No and Author Reporting	Gastro-intestinal Tract	Nervous system including eye and ear	Cutaneous	Lymphoid tissue spleen liver	Musculo-skeletal system	Respiratory tract	Genito-urinary tract and urinalysis	Cardio-vascular system	Blood tests	Miscel-laneous data
24 Cody and Williams* (Case 1)		Headache temporal pain				Nasopharyngitis cough associated with flare-ups in eye	Urine—red blood cells grade 1, white blood cells grade 3		Leukocytosis elevated sed rate of erythrocytes	
25 Cody and Williams* (Case 2)										Biopsy—negative
26 Cody and Williams* (Case 3)		Occipital headache				Productive cough		Systolic apical murmur		Generalized warm feeling
27 Cody and Williams* (Case 4)	Nausea vomiting	Peripheral neuritis abnormal EEG			Nuchal rigidity pain and tenderness in shoulder left costal margin leg muscle	Pleuritic pain rhinorrhea	Urine—6-10 red blood cells per high-power field 7-10 white blood cells per HPF		Leukocytosis elevated sed rate of erythrocytes	Fever

*Present paper

REPORT OF CASES OF ATYPICAL COGAN'S SYNDROME

Case 5 A 33 year old man was first seen at the clinic on January 1 1956 During the late summer of 1952 a lesion had developed on the patient's right ankle followed by the development of eczematoid dermatitis of his legs and arms He received X ray therapy for this condition during September and October In early October an upper respiratory infection developed, accompanied by severe pain low in the back and by headache Following the upper respiratory infection a suspected industrial accident occurred in which cutting oil was supposed to have accidentally irritated both of his eyes A diagnosis of conjunctivitis was made at this time The eye condition rapidly grew worse, and the patient was hospitalized with severe uveitis and corneal ulceration Near the end of October dizziness and impairment of hearing developed, the latter progressing rapidly in one week to total bilateral deafness He was treated with an unknown quantity of Cortisone and ACTH

In December 1952 he was again admitted to the hospital with uveitis and meningismus An acute hemorrhage developed in the gastrointestinal tract while he was in the hospital Eleven transfusions were required and subsequent roentgenograms revealed a small ulcer on the lesser curvature of the stomach He gradually improved in the hospital and was dismissed in March 1953 At that time his vision, uncorrected, was as follows right eye, 20/50 left eye 20/30 He was totally deaf and showed no response to the caloric test The edema redness and photophobia gradually cleared, and in 1954 he was able to go back to work in a machine shop

In March 1955, redness, edema and photophobia of both eyes again developed and from that time until he was seen at the clinic, he had frequent remissions and exacerbations of his ocular condition The use of neosone ophthalmic ointment seemed to be of benefit during the exacerbations

The general history and examination revealed that the patient had not had symptoms of ulcer since the gastric hemorrhage in 1952 For the previous 15 years he had been troubled with allergic rhinitis consisting of lacrimation rhinorrhea and sneezing during the months of September and October He weighed 155 pounds and was 70 inches tall his blood pressure was 100 mm of mercury systolic and 58 mm diastolic his pulse was 80 beats per minute and temperature, 98° F An axillary furuncle and slight benign prostatic hypertrophy were noted The dermatologist stated that there were multiple nevi over the upper part of the chest, the back and the upper part of the arms, but poliosis or vitiligo was not evident The audiogram showed complete bilateral sensory-neural deafness Neither spontaneous nor positional nystagmus was present Kobrak's modification of the cold caloric test resulted in a minimal response bilaterally, the response being slightly more active on the left side

The ophthalmologist stated that the patient had sclerosing keratitis and although the disease in the patient's eyes was rather severe, he still had remarkably good vision Vision in the right eye measured 20/60, in the left eye 20/25

The results of urinalysis were normal and values for hemoglobin erythrocyte sedimentation rate and erythrocyte count were normal The leukocyte count was 11 600 per cubic millimeter of blood and the Kline flocculation test was nonreactive A roentgenogram revealed no abnormality in the esophagus stomach or duodenum The patient was dismissed on January 20, 1956

In this case vestibulo-auditory findings characteristic of whether or not any manifestations of systemic disease ever were present

In the nine cases in Table I in which minimal additional findings such as leukocytosis, eosinophilia, and abnormal findings in the urine or an upper respiratory infection were reported, there is not enough evidence to say that manifestations of a generalized disease actually were exhibited At least such manifestations were suggested Thorough investigations were reported in six of the nine cases (Cases 2, 3, 4, 5, 8, 9), although we do not know just what the ptomaine poisoning immediately prior to the onset of Cogan's syndrome in Case 4 entailed (see Table I) In Case 13 examination seemed to have been complete, but the presentation is short and laboratory findings are not mentioned There is no indication that thorough investigation was made in either Case 7 or Case 26, and we do not know the circumstances in Case 7 leading to appendectomy five months after the onset of the illness From the reports available in these two cases (Cases 7 and 26) we have no way of knowing whether any further features indicative of a more generalized disease process were ever manifested

In the remaining 13 cases in Table I (Cases 6, 10, 15-24, 27) it is our opinion that we have sufficient findings, other than those characteristic of Cogan's syndrome, to consider them as examples of a generalized disease process In Table I, therefore, the evidence seems adequate for us to state that in 13 or 48 per cent of the cases of Cogan's syndrome thus far reported, the disease process is one of generalized distribution

In nine cases there are findings that seem to indicate that the disease is not confined to the cornea and VIIIth cranial nerve, while in the remaining five cases there were no manifestations other than those characteristic of Cogan's syndrome

In reviewing the cases of Cogan's syndrome and related conditions encountered at the clinic, we found two cases which seemed to be examples of Cogan's syndrome except for ophthalmologic findings which were not typical of Cogan's syndrome the speech reception test in the right ear at 70 decibels 15 decibels above threshold the patient had 55 per cent of the words correct In the left ear at 70 decibels 20 decibels above threshold he had 55 per cent of the words correct On the Kobrak equilibrium test there was no response to 30 cc of ice water

Examination of the eyes showed perilimbal injection on the left and a minimal and isolated patch of perilimbal injection on the temporal side of the right limbus A small disruption of epithelium of the inferior nasal quadrant of the cornea and two disruptions of corneal endothelium were noted immediately below the center of the cornea of the right eye Palpebral conjunctivitis was bilateral The ophthalmologist's opinion was that the patient did not have the typical interstitial keratitis seen in Cogan's syndrome but that he had a condition more like episcleritis

Results of urinalysis and values for hemoglobin erythrocyte count, leukocyte count and erythrocyte sedimentation rate were normal Results of the serologic tests for syphilis also were negative Roentgenograms of the head and thorax were without evidence of abnormality, a roentgenogram of the stomach showed evidence of a duodenal ulcer The patient was placed on an ulcer diet and systemic treatment with Cortisone under the supervision of his home physician was advised He was dismissed on July 18 1956

All features of Cogan's syndrome except those for typical interstitial keratitis were present in this case In our opinion, however, this case had to be classified as an atypical example of Cogan's syndrome in the same manner as Lindsay and Zuidema's[4] case with bilateral iritis (see Table II) The presence of many manifestations such as cervical lymphadenopathy, sores on the right ear, hoarseness associated with loss of hearing, loss of weight and duodenal ulcer suggested that the disease process was of widespread distribution

These three examples of what we considered to be atypical Cogan's syndrome led us to wonder whether or not other such cases had been reported in the literature and, if so, what diagnosis, either clinical or histologic, had been given by their authors

REVIEW OF THE LITERATURE

We were able to find seven cases in the literature, in addition to our Cases 5 and 6, that showed characteristics of Cogan's syndrome or could be considered as atypical examples of the syndrome In five of the nine cases in Table II, a diagnosis was made of periarteritis nodosa[13-17], in one case the diagnosis was disseminated lupus erythematosus,[18] in one case, Cogan's syndrome,[4] and in our two cases atypical Cogan's syndrome Cases 1, 2, 3, 5, 7, 8, and 9 (see Table II) are close-

Cogan's syndrome were displayed, but the conjunctivitis, superficial keratitis, uveitis, and sclerosing keratitis were not typical of the syndrome Involvement of multiple systems in the disease process was displayed during the early stages These findings consisted of dermatitis, upper respiratory infection, pain in the back, headache, meningismus and leukocytosis We do not know how much Cortisone and ACTH the patient had received prior to the appearance of the massive gastric hemorrhage and, therefore, cannot be certain whether or not this treatment was responsible for the gastric ulcer

In this paper the term "atypical Cogan's syndrome" is defined as a condition with vestibulo-auditory manifestations and ocular findings that include keratitis, uveitis, episcleritis and others but not interstitial keratitis

Case 6 A 22 year old farmer was first seen at the clinic on July 11, 1956 Between 1950 and 1952 the patient had episodes of an appearance of a lump below the lobe of the right ear associated with small sores over the pinna These episodes lasted about one week and recurred two or three times a year

In the spring of 1952 redness, soreness, burning and lacrimation of both eyes developed These symptoms cleared spontaneously but recurred again in 1953, and from that time on the patient had experienced remissions and exacerbations of the ocular complaints The soreness and burning in his eyes during exacerbations were frequently sufficiently severe to keep him awake at night Cortisone drops brought relief while they were used

Biopsy of a cervical lymph node was done in 1952, but the reasons for this examination and the findings were not available In 1953 the patient was treated for a duodenal ulcer at which time he had epigastric pain relieved by the ingestion of food He had no further trouble during the two years prior to being seen at the clinic

Early in 1954 the patient was working on a tractor when severe rotatory vertigo associated with nausea and vomiting developed The episode lasted three days Approximately two weeks later he had a similar attack of vertigo, but this time bilateral tinnitus also developed, louder in the right ear than in the left Within the next month he began to notice impairment of hearing in his right ear The attacks of vertigo continued but gradually became less severe In June, 1954, he began to notice impairment of hearing in his left ear For the next year he noticed fluctuation in his ability to hear From March 1955 until the patient was seen at the clinic, he had been free of vertiginous attacks but tinnitus had persisted The patient noticed that his voice was hoarse during the onset of impairment of hearing

On examination the patient weighed 119 5 pounds, he said that he had lost 15 pounds in the previous three years The blood pressure was 130 mm of mercury systolic and 68 mm diastolic, pulse 64 beats per minute and temperature 97 8° F

An audiogram showed a 40 to 65 decibel loss in both ears The loudness balance test showed essentially symmetric increase of loudness On ly related to Cogan's syndrome and probably can be considered atypical examples It is interesting to note that the diagnosis in four of these cases was periarteritis nodosa, and in our cases findings were suggestive of a systemic condition Case 4 is representative of the eight cases reported by Cogan[19] of periarteritis nodosa and corneoscleral lesions, however, Parker and Kernohan[20] pointed out that when periarteritis nodosa involves the nervous system, any clinical syndrome may be produced, and this is especially applicable to the eye [21] Harris and associates,[22] in an analysis of 101 reported cases of periarteritis nodosa, found that 23 per cent of the patients had visual disturbances and that changes such as papilledema, exudates and hemorrhages in the fundus are not necessarily a result of hypertension but may be due to the arteritis itself as exemplified by two of the reported cases Case 6 is included to draw attention to the role of disseminated lupus erythematosus in lesions of the eye Baehr and co-authors[23] found that 12 of 23 patients having disseminated lupus erythematosus had abnormalities of the fundus, he stressed that these changes were the result of essential vascular pathologic conditions in the retina It has been pointed out that, just as in periarteritis nodosa, but less commonly, all the layers of the eye may be involved in disseminated lupus erythematosus [24]

In the literature, reports of cases of collagen diseases with sensory-neural deafness resulting from arteritis are rare, the cases of periarteritis nodosa with perception deafness are included in Tables I and II

In Table I a significant number of patients having Cogan's syndrome showed manifestations that are highly suggestive of systemic disease In our cases and in those reported in the literature these manifestations are frequently seen to undergo remissions and exacerbations which produce a rather unrelated clinical picture In seven of the 27 cases in Table I a diagnosis of periarteritis nodosa was made on histologic or clinical grounds Seven cases in Table II can be considered as atypical examples of Cogan's syndrome In four of these seven cases a diagnosis was made of periarteritis nodosa, and in two, manifestations suggested that the condition was of a systemic nature It was pointed out that although few cases

TABLE II

Cases Showing Some of the Characteristics of Cogan's Syndrome

Case No and Author Reporting	Involvement			Diagnosis
	Eye	Auditory	Vestibular	
1 Middleton and McCarter[13]	Failing vision right eye left eye poor from injury 9 years before pupils did not react to light	Bilateral deafness, bilateral tinnitus	Dizziness nystagmus	Periarteritis nodosa
2 Bernstein[14]	Three to four attacks of amblyopia in left eye, occlusion of left central retinal artery	Bilateral deafness		Periarteritis nodosa
3 McNeil and associates[15]	Drooping right eyelid	Bilateral deafness, bilateral tinnitus	Vertigo	Periarteritis nodosa
4 Ingalls[16]	Bilateral uveitis and keratitis			Periarteritis nodosa
5 Spiegel[17]	Episcleritis iritis periorbital swelling right eye	Bilateral deafness		Periarteritis nodosa
6 Macrae and O Reilly[18]	Loss of visual acuity mild papilledema of fundus exudates and hemorrhages	Bilateral tinnitus	Vertigo	Systemic disseminated lupus erythematosus
7 Lindsay and Zuidema[4]	Bilateral iritis and secondary glaucoma of left eye	Bilateral deafness bilateral tinnitus	Vertigo bilateral hypoactive caloric response	Cogan s syndrome
8 Cody and Williams* (Case 5)	Bilateral conjunctivitis superficial keratitis uveitis sclerosing keratitis	Bilateral deafness	Dizziness bilateral minimal caloric response	Atypical Cogan s syndrome
9 Cody and Williams* (Case 6)	Bilateral episcleritis	Bilateral deafness bilateral tinnitus	Vertigo bilateral absence of caloric response	Atypical Cogan s syndrome

*Present paper

selection definitely includes a large number of patients with fatal disease.

In Cogan's syndrome, although the disease is systemic, lesions of vital organs are usually of minor importance, and from the available evidence it would appear that because Cogan's syndrome has a relatively favorable prognosis, an association with periarteritis nodosa is certainly not negated.

The uncertainty of random biopsy of muscle in yielding a diagnosis in periarteritis nodosa and the necessity of repeated biopsies is well documented in the literature,[26, 28, 29] and it is generally accepted that negative findings on biopsy do not rule out the possibility of periarteritis nodosa. Rose and associates[28] described four cases which contained necropsy reports of periarteritis nodosa. Even though multiple biopsies had been performed in two cases and a single biopsy in two cases, yet a diagnosis of periarteritis nodosa had been confirmed on microscopic examination in only one instance. It is certainly evident, therefore, that negative results on biopsy in Cogan's syndrome do not rule out the possibility of periarteritis nodosa.

Klinge[30, 31] was the first to introduce the concept that a group of systemic diseases affects connective tissue. Klemperer and associates[32] introduced the term "collagen diseases" to indicate the site of the characteristic lesion in these diseases, and under this term he[32-34] included rheumatic fever, rheumatoid arthritis, polyarteritis nodosa, acute disseminated lupus erythematosus, generalized scleroderma and dermatomyositis. Since the origination of the term "collagen diseases," many syndromes once considered separate entities seem to fit either partially or entirely into this group of diseases. Such is the case in Libman-Sacks syndrome,[35, 36] idiopathic midline granuloma,[19, 37-42] temporal arteritis,[39, 43, 44] Loeffler's syndrome[28, 39, 45-47] and thrombotic thrombocytopenic purpura.[21, 35] It is not surprising, therefore, to see Cogan's syndrome identified with the collagen diseases.

Just as these syndromes, previously considered as separate entities, are being grouped together under the broad term "collagen disease" and present a somewhat clearer picture to of sensory-neural deafness in collagen diseases are reported in the literature, it is not unusual to find lesions involving practically all structures of the eye in periarteritis nodosa, disseminated lupus erythematosus, or even in rheumatoid arthritis [12, 21]

In conclusion, therefore, we feel it is justifiable to say that Cogan's syndrome is not confined to the cornea and VIIIth cranial nerve but rather that it is only one manifestation of a systemic disease—a systemic disease that on occasions involves so many seemingly unrelated structures and produces such a bizarre clinical picture that one cannot help but link it with the collagen diseases, also, from the available evidence, it would seem that the syndrome could be included in the group with periarteritis nodosa In the discussion of the paper by Quinn and Falls,[12] Norton expressed the opinion that the relatively good prognosis and the frequent failure of biopsy of muscle to yield a diagnosis were two important objections to the hypothesis that Cogan's syndrome is only a local manifestation of periarteritis nodosa

There are many cases of periarteritis nodosa reported in the literature, such as the case of Cury and associates,[25] in which the patients have lived six or more years with the disease Grant,[26] in a review of the literature up to 1941, drew attention to patients who had survived from four to 32 years and felt that in many of the other cases in which the duration was reported to be much shorter, the onset of disease might have been considerably antedated by taking into account previous similar illnesses reasonably regarded as expressions of periarteritis nodosa Weiss[27] stressed that the disease is not invariably fatal and that the prognosis would improve with earlier clinical recognition of the disease Rose and associates,[28] in 1950, estimated that at least 5 to 10 per cent of patients recovered and they, along with Spiegel[17] and Ragan,[9] stressed that the prognosis depended upon the extent of involvement of the vital organs If the arterial lesions heal, and in the process do not infarct a vital area, the patient will recover, and no clue to the basis of the illness will be available, therefore, when angiitis is discussed, it probably includes only a small portion of the total group with the disease, and the

Pagel and associates[56] emphasized the general involvement of the vascular system as the underlying cause of the muscular and dermal changes in dermatomyositis although they said that these changes were not specific for this disease as they were also seen in scleroderma and rheumatoid arthritis. Later, Pagel[59] stated that the basic vascular changes in periarteritis nodosa, disseminated lupus erythematosus, dermatomyositis, scleroderma, "arteritis granulomatosa allergica," and temporal arteritis are similar, if not identical, although there are differences in the frequency and distribution of the lesions in the different diseases.

Ellman and Ball,[24] Pirani and Granville,[illegible] and Macrae and O'Reilly[45] drew attention to the morphologic similarities between the lesions of rheumatoid arthritis, rheumatic fever, periarteritis nodosa, disseminated lupus erythematosus, scleroderma and dermatomyositis, and Pirani and his co-workers[illegible] stated that though fibrinoid degeneration is less frequently seen in rheumatoid arthritis, scleroderma and dermatomyositis, they run a more chronic course and pathologic findings in early stages are scarce. These are only a few of the many references in the literature which seem to show that all collagen diseases have the same basic vascular alterations, the differences being only in degree and distribution.

The literature contains numerous reports of cases which displayed either clinically, histologically, or both, the characteristics of two or more of the collagen diseases. Ophuls[illegible] in 1923 stated that one did not have to study many cases of periarteritis nodosa to discover its close relation to the rheumatic diseases, and he drew attention to the similarity between granulomas in periarteritis nodosa and rheumatic nodules. Since then others[13, illegible] have re-emphasized this impression. There are many examples[illegible] in the literature of an association of rheumatic fever and periarteritis nodosa in an individual patient, and in 1956, Rose[64] estimated that 12.5 per cent of cases of polyarteritis nodosa showed evidence of past or active rheumatic fever. Periarteritis nodosa also has been seen in association with rheumatoid arthritis,[illegible] with findings characteristic of both rheumatic fever and rheumatoid arthritis,[illegible] with disseminated lupus erythematosus,[illegible] the clinician, so the individual members of this group seem to be more closely related As the volume of literature on the respective collagen diseases grows, it is increasingly evident that we are not finding a clearer differentiation between one and the other, but that we are discovering more overlapping on the histologic, clinical and experimental levels

The term "collagen diseases" refers to the fact that alterations of the extracellular portions of the connective tissue are prominent and systemic in these diseases,[33] and the changes may be degenerative or proliferative Both the degenerative and proliferative alterations terminate frequently in sclerosis with or without hyalinization [48] It was Banks'[36] impression that the vascular lesions in many of the collagen diseases were similar and that some cases begin as one and terminate as another He wondered whether all of them had the same denominator

Mallory[49] pointed out that the collagen diseases merged with one another so frequently that it was extremely difficult to separate them He stated that at the Massachusetts General Hospital and elsewhere, there had been a great many cases of clinically typical disseminated lupus erythematosus that showed widespread periarteritis nodosa at necropsy, and this also was true of rheumatic fever and dermatomyositis [50] He felt that there were no histologic findings pathognomonic of disseminated lupus erythematosus In support of this, Baggenstoss[51] cited a case in which there were typical skin lesions of disseminated lupus erythematosus and the L E cell phenomenon in the bone marrow but which showed widespread arterial lesions indistinguishable from those observed in typical cases of periarteritis nodosa

Ball[52] reported cases showing that the arteritic changes found in the rheumatoid patient all fall within the histologic variants of polyarteritis nodosa, and he felt that the arteritis of rheumatic fever was only another histologic variant Dowling, with Griffiths[53] and alone,[54] and Brock stated that the early cutaneous, muscular and vascular changes in scleroderma and dermatomyositis show precisely the same alterations though they vary in degree in individual cases a trend indicating that all of the collagen diseases have a common basic pathologic relation in the vascular system that is molded, through constitutional or other unknown factors into the patterns of syndromes and diseases known today as individual members of the collagen group of diseases

SUMMARY

There have been 23 cases of Cogan's syndrome reported in the literature, and we have reported an additional four cases Of these four cases, a clinical diagnosis of periarteritis nodosa was made in one instance, and in two cases there were findings which seemed to indicate that Cogan's syndrome is only a local manifestation of a more generalized disease

Of the entire 27 cases of Cogan's syndrome 13, or 48 per cent, had sufficient findings other than those characteristic of Cogan's syndrome to be considered examples of a generalized disease process In nine cases features were present that at least suggested that Cogan's syndrome is a generalized disease Five cases showed no findings other than those characteristic of the syndrome In three cases a diagnosis of periarteritis nodosa was made histologically and in four cases clinically

We reported two cases of atypical Cogan's syndrome with features indicating that each was a generalized disease process Five additional cases of what seemed to be atypical Cogan's syndrome were found in the literature, and in four of these cases a diagnosis of periarteritis nodosa had been made

We concluded that Cogan's syndrome is not confined to the cornea and VIIIth cranial nerve, but that it is only one manifestation of a systemic disease From the available evidence it would seem that this systemic disease is periarteritis nodosa

The relatively good prognosis and the frequent failure of muscle biopsy to yield a diagnosis are not serious objections to the hypothesis that Cogan's syndrome is only a local manifestation of periarteritis nodosa

It is now generally considered that Libman-Sacks syndrome idiopathic midline granuloma, temporal arteritis, Loeffler's and with findings characteristic of both disseminated lupus erythematosus and rheumatoid arthritis.[49] Disseminated lupus erythematosus has occurred in patients having rheumatic fever,[18,69] rheumatoid arthritis,[29,35,70] scleroderma[36,71] and dermatomyositis[36,72] and in patients with findings characteristic of both rheumatic fever and dermatomyositis.[73] The similarities between dermatomyositis and scleroderma have been mentioned. Dermatomyositis has been seen in patients with rheumatic fever[74] and scleroderma in patients having rheumatoid arthritis.[24]

With an overdosage of desoxycorticosterone acetate to rats, Selye and associates[75-76] were able to produce polyarteritis nodosa, nephrosclerosis, myocardial and joint lesions similar to those associated with acute rheumatic fever, and in some instances lesions were reminiscent of the proliferative stage of scleroderma.

Two laboratory procedures should be mentioned briefly. The L.E. cell phenomenon is a useful test in the diagnosis of disseminated lupus erythematosus; however, the recent literature has shown that it is not a completely specific one. As previously mentioned, Baggenstoss[51] cited a case in which the L.E. cell phenomenon had developed in the bone marrow, but histologically it was typical of periarteritis nodosa. A sizable proportion of patients with rheumatoid arthritis have positive L.E. cell tests[29,66] and there are examples in the literature of false-positive L.E. cell phenomena occurring in diseases such as miliary tuberculosis.[77]

The second group of procedures to be considered is the serology tests for syphilis which occasionally are found to give falsely positive results in both disseminated lupus erythematosus[78-79] and periarteritis nodosa.[28,80] We are not in a position to state the significance of these falsely positive reactions to serology tests, but it is of interest that they occur in both of these conditions.

We made no attempt to review all of the voluminous literature on the collagen diseases, and from the available information it is impossible to make any dogmatic statements. It would seem, however, that we have been able to demonstrate

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syndrome and thrombotic thrombocytopenic purpura also may be identified with collagen diseases We have shown a trend which could indicate that all collagen diseases have a common pathologic basis in the vascular system which is moulded through constitutional or other unknown factors into the patterns of syndromes and diseases that are known today as individual members of the collagen group of diseases

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ACOUSTIC TRAUMA OF THE COCHLEA FROM EAR SURGERY*†‡

HAROLD F. SCHUKNECHT, M.D.,
Detroit, Mich.,
and
JUERGEN TONNDORF, M.D.,
Iowa City, Ia.

It is now well established that high energy acoustic signals delivered to the cochlea can cause tissue damage which is usually most severe in the region of the upper basal turn, and is manifested functionally by deafness most severe for high frequencies. A classical clinical example is found in patients exposed to a single, or repeated, air borne blast of high intensity (as from musketry, fire crackers, explosions).

It has been shown recently that a blow to the head results in a similar mechanical insult to the cochlea, having its greatest impact likewise on the upper basal turn.[1,2]

Case 1. Mr. X. A 29 year old male salesman was involved in an automobile accident following which he was unconscious for an hour and had discharge of blood and cerebrospinal fluid from the right ear. On regaining consciousness he complained of hearing loss in the right ear. He reported having experienced good hearing prior to the accident. Roentgenograms revealed a longitudinal fracture of the right temporal bone. The next day there was a moderately severe combined conductive and sensory neural hearing loss and drainage of serohemorrhagic fluid from the right ear. An audiogram taken four months later showed persistence of the sensory-neural deafness. There was a slight hearing loss in the midfrequency range and a profound loss for frequencies above 2000 cps (see Fig. 1a).

The evidence from an animal experiment should have finally dispelled the common misconception that the deafness follow-

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*Read at the meeting of the Middle Section, American Laryngological, Rhinological and Otological Society, Inc., Chicago, Ill., Jan. 18-19, 1960.

†This work was supported in part by a Grant from the Central Bureau of Research of the American Otological Society and in part by Grant B-1900 of the Public Health Services.

‡The high-speed motion pictures referred to in the text have been made possible through an equipment loan with the Office of Naval Research Nonr 1893(00).

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OTOLOGICAL FUNCTIONAL MICRO-SURGERY POST GRADUATE COURSE

A practical course of surgical techniques in otological surgery will be conducted by Dr Michel Portmann at the University of Bordeaux, France, Ear, Nose and Throat Department, June 26 to July 5, 1960 This course will be given in English

The number of assistants will be limited to twenty, divided into two groups The fee of $100 00 is to be paid at the Faculty of Medicine of Bordeaux, at the beginning of the course Applications should be sent to Dr Michel Portmann, 45 Cours Foch, Bordeaux, France

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The evidence from an animal experiment should have finally dispelled the common misconception that the deafness following a head blow could be the result of VIIIth nerve or brain injury[13] The hearing deficits, as well as the spatial distribution and histopathological appearance of the cochlear lesions of animals subjected to head blows, were identical to those resulting from the discharge of a 32 caliber cartridge near the ear[8]

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Cat 1 Ches The right ear was destroyed and baseline behavioral pure tone thresholds were taken The animal was

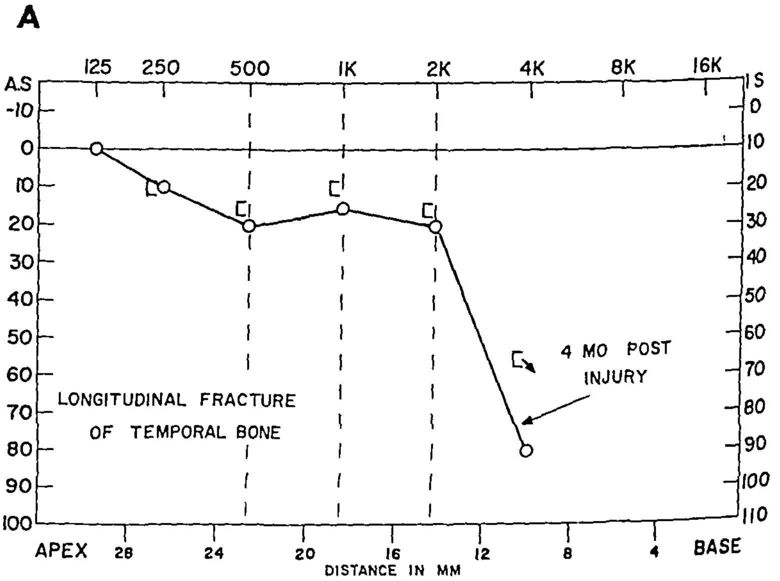

Fig 1-a Case 1 Audiogram showing hearing loss existing four months following head injury The cochlear injury is the result of so-called 'inner ear concussion

anesthetized, the skull exposed by a midline scalp incision, and a blow delivered to the left temporoparietal area of the skull The scalp incision was closed with silk sutures Auditory testing was resumed two days after administering the head blows, and the animal was tested about every four days until sacrificed on the 22nd day

Postoperative behavioral audiograms revealed a moderate hearing loss for frequencies up to 2,000 cycles, a profound loss for 4,000 and 8,000 cycles, and near normal hearing for

16,000 cps The auditory thresholds improved over a two-week period, after which they remained stationary, with the final audiogram at the end of three weeks showing a persisting 4,000 and 8,000 cps dip

On histological examination there was a loss of outer hair cells from 7 75 to 10 5 mm region The degree of injury varied considerably in the ten cats in this series, but in all cases the maximum injury occurred in the region of the upper basal turn, which is the region of maximum importance for the frequencies 4,000 to 8,000 cps (see Fig 1-b)

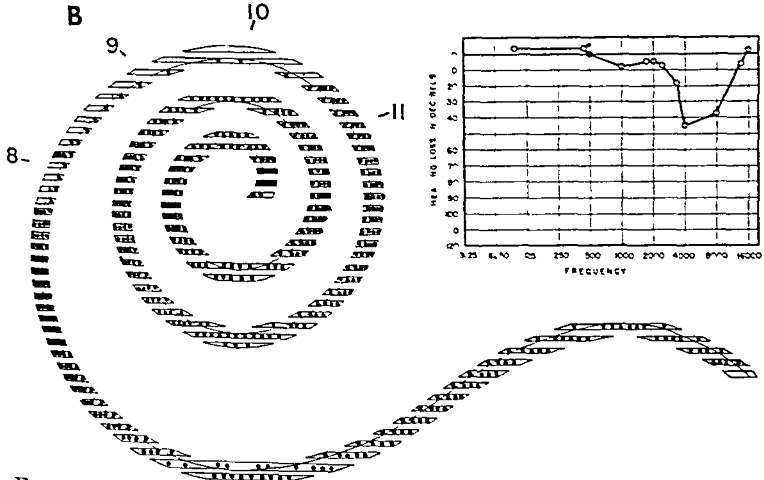

Fig 1-b Cat 1 Audiogram and cochlear graphic reconstruction of cat following blow to the head There is a loss of hair cells in the 7 5 mm to 10 5 area of the upper basal turn which has resulted in a 4000 and 8000 cps hearing loss

It is obvious that a blow to the head can result in a bone conducted acoustic signal of sufficient magnitude to injure the organ of Corti

The concept that surgical procedures can create acoustic energy of sufficient magnitude to cause cochlear injury is not new The improper use of the mallet and gouge in exenteration of sclerotic mastoid bone can create such an injury

Case 2 B B This nine year-old boy had a chronic draining left ear since the age of one There was a perforation in Shrapnell's membrane with a foul smelling discharge The pars tensa was intact

At operation (1949) the mastoid cortex was removed with mallet and gouge Many firm blows were used to accomplish proper exenteration The cell partitions were removed with an electrically driven burr The epitympanic space was widely exteriorized because of cholesteatoma

The postoperative course was satisfactory except that there was a sensory neural type hearing loss for frequencies above 2,000 cps (see Fig 2 a)

Ruedi and Furrer[11] have produced similar high frequency hearing losses (temporary) in human volunteers by reflecting the scalp over the mastoid bone and delivering mallet blows directly to the bone

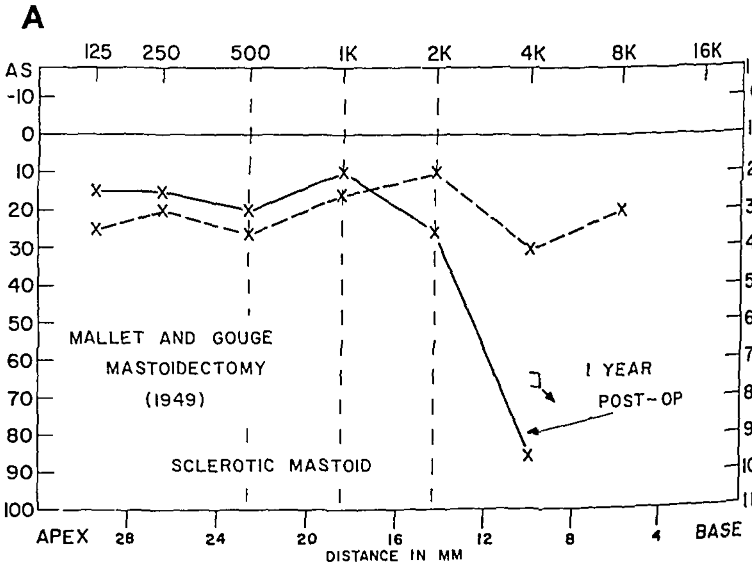

Fig 2-a Case 2 Pre- and postoperative audiograms showing high frequency hearing loss resulting from mastoidectomy

In many human surgical procedures and in a series of 60 cats in which electrically driven burrs (cutting, polishing, and diamond) were used, there have never been hearing deficits attributable to acoustic stimulation Apparently, the acoustic intensities generated by the burr are at safe levels This might not be true if the burrs were applied directly to any part of the intact ossicular chain

It is becoming increasingly apparent with the development of new surgical procedures, that there is more danger of acoustic injury to the cochlea. In tympanoplasty procedures, for example, a number of potentially injurious maneuvers may be performed: e.g., palpation of various parts of the ossicular chain, incision of the incudostapedial joint, removal of granulations, connective tissue bands, and hyalinized collagen (tympanosclerosis) from the stapes or round window areas, repositioning the ossicles on the stapes, placing the pars tensa or a skin graft on the stapes; inadvertently

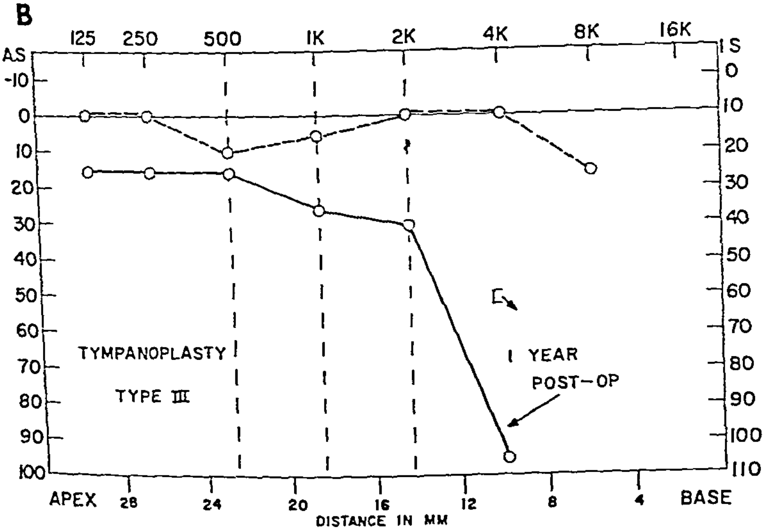

Fig. 2-b. Case 3. High frequency hearing loss secondary to stapes manipulations occurring in the course of middle ear surgery.

striking the stapes with an instrument, etc. This kind of surgical complication is demonstrated by the following case.

Case 3. V.B. No. 933792. This 25 year old school teacher had intermittent suppuration from the right ear for five years. There was a large perforation of Shrapnell's membrane.

At operation squamous epithelium was found to line the epitympanum, envelope the head of the malleus and body of the incus and extend into the anterior mesotympanum. The pars tensa was intact. To accomplish complete removal of the squamous epithelium, it was necessary to remove the incus and the head of the malleus. The pars tensa was mobilized partially and depressed to make contact with the head of the stapes. After appropriate surgical preparation, the epitympanum and mastoid

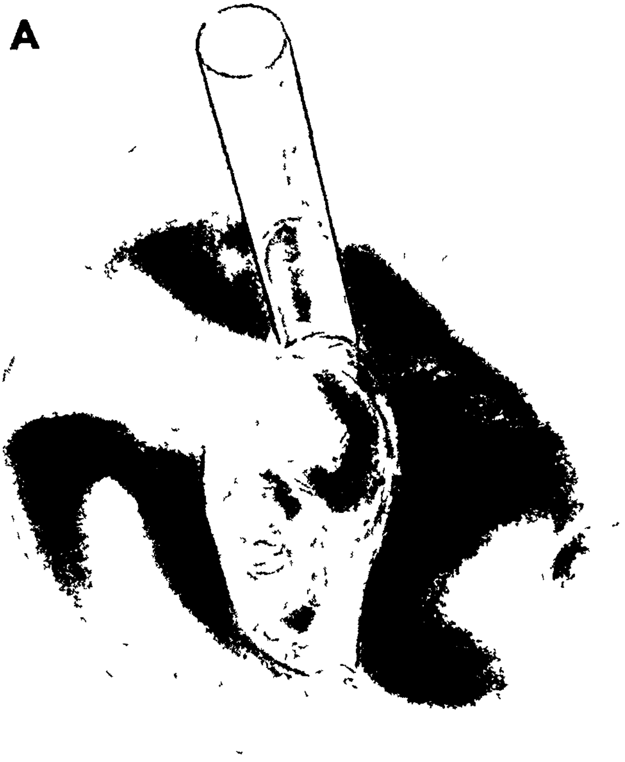

Fig 3-a Drawing showing chisel located at the anterior margin of the footplate as formerly commonly used for stapes mobilization

antrum were covered with a free split thickness graft from the inner surface of the arm

The postoperative course was satisfactory Although the bone air gap in the speech range was about 15 db the ear sustained a severe sensory neural hearing loss for frequencies above 2,000 cps The injury apparently was secondary to stapes movements coincident with removal of the incus and repositioning of the pars tensa (see Fig 2 b)

Stapes operations for otosclerosis are even more apt to create cochlear damage The stapes is often manipulated drastically with a variety of picks and chisels designed to accomplish mobilization (see Fig 3-a) After mobilization, the intact stapes is often rocked back and forth, or pushed in and out of the oval window, creating amplitudes of movement which are enormous (in comparison to physiological movements) The sudden mobilization of an ankylosed footplate or the maneuvers to remove an intact footplate probably create similar intense stimulation Following is an example of such a case

Case 4 E P, No 054776 The history and findings were typical for bilateral otosclerosis At operation the anterior one-third of the foot plate was found to be involved in the otosclerotic lesion The head neck and crura were removed by disarticulating the incudostapedial joint and pushing the stapes toward the promontory so as to break the crura at their bases The posterior two thirds of the footplate was then manipulated with small needles and chisels until separated and free at all margins A tantalum wire prosthesis was then interposed between the incus and mobilized portion of the footplate

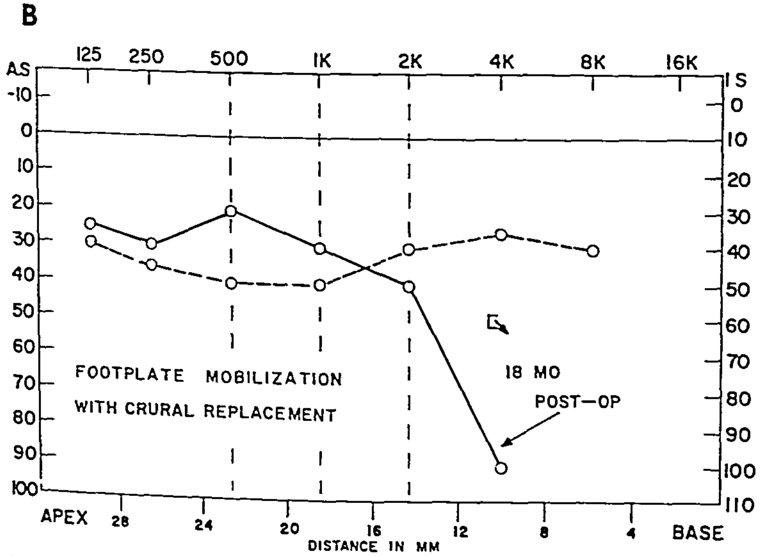

Fig 3-b Case 4 High frequency hearing loss resulting from extensive manipulation of the stapes footplate

The hearing gain for speech frequencies was unsatisfacory but in addition there was a severe sensory neural loss for frequencies above 2 000 cps Apparently the operative manipulations of the footplate were of such magnitude as to damage the cochlea (see Fig 3 b)

The nature of the cochlear injury resulting from stapes manipulation has been demonstrated in an experiment on stapes fracture in cats[14] Of 12 ears with experimental fractures, there was operative displacement of the large part of the footplate in four In one of these, the footplate was subluxated out of the oval window and in this ear the saccule and cochlea remained normal In the other three the displacement of the footplate fragment was inward but probably not so far as to come in contact with the saccule In these the saccular wall was ruptured and the organ of Corti damaged in the upper basal level and/or lower middle turns Protocols for these animals follow

Cat 2 Jo, left Using a postauricular approach and aseptic technique, the crura were fractured from the footplate and a small needle was used to perforate the center of the footplate Postoperatively, no nystagmus or ataxia was apparent Sacrificed in three months

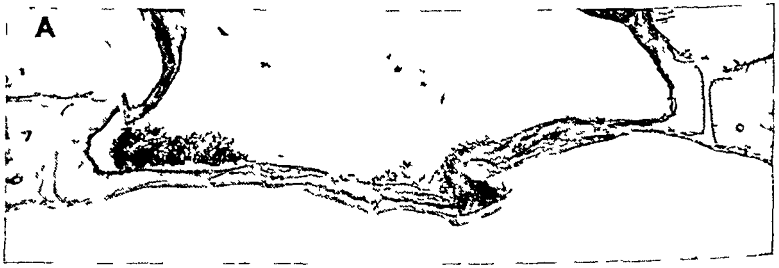

Fig 4-a Cat 2 Experimental fracture through the central portion of the footplate Sacrificed three months later a—The stapes footplate showing complete bony healing, following comminuted fracture of its midportion

Histological examination showed a comminuted fracture of the footplate healed by profuse bony regeneration There was a rupture of the saccule with collapse of its wall onto the underlying neuro-sensory epithelium which appeared normal The other vestibular sense organs appeared normal There was injury and partial loss of outer hair cells of the organ of Corti in two anatomically separated areas, one in the upper basal turn from 9 5 mm to 10 2 mm and the other in the lower middle turn from 11 5 mm to 12 mm (see Figs 4-a, 4-b, and 4-c)

Cat 3 Til, right By an aseptic operation through a postauricular approach, the oval window area was exposed and the crura were fractured from the footplate and removed A

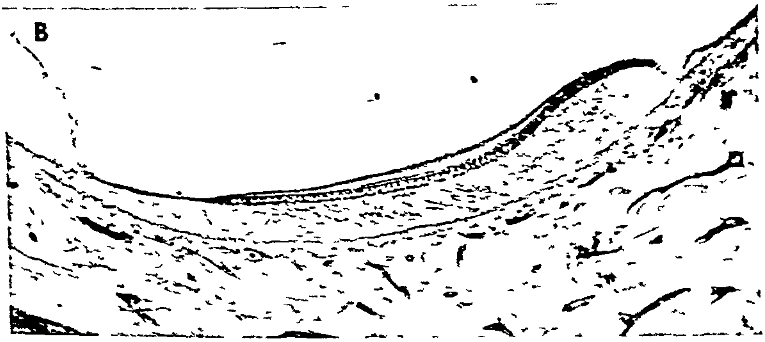

Fig 4-b Cat 2 Rupture and collapse of the saccular wall

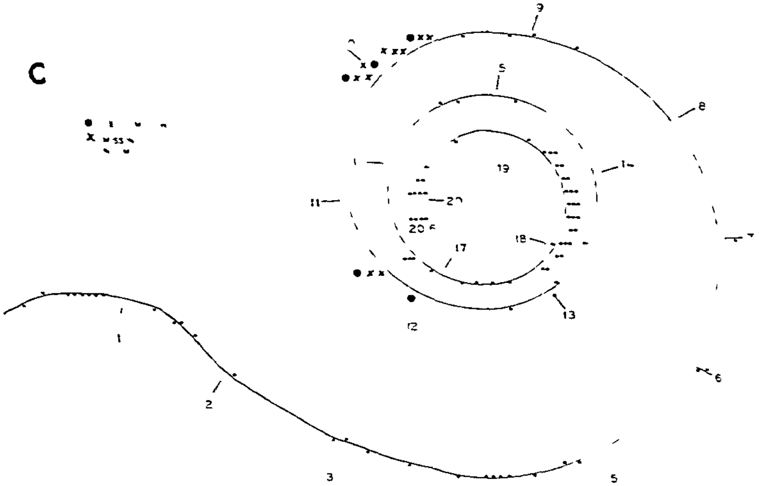

Fig 4-c Graphic reconstruction of the cochlea showing hair cell loss in the upper basal and lower middle turn

fine needle was used to dislocate the anterior margin of the footplate and depress it into the vestibule In this maneuver, the anterior three-fourths of the footplate fractured from the posterior one-fourth, (which remained in its normal position) Subsequently, the mobilized section returned near its normal location in the oval window Sacrificed in three months

Histological study revealed a linear vertical fracture extending through the posterior part of the footplate There

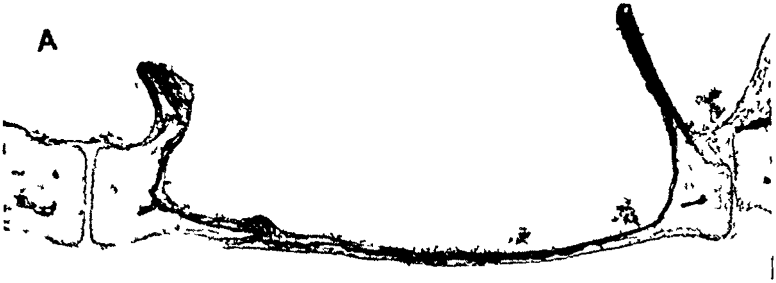

Fig 5-a Cat 3 Fracture dislocation of the anterior two-thirds of the stapes footplate Sacrificed three months later a—View of the footplate showing slight inward subluxation of the anterior two-thirds There is bony healing of the fracture located at the junction of the posterior and middle thirds of the footplate

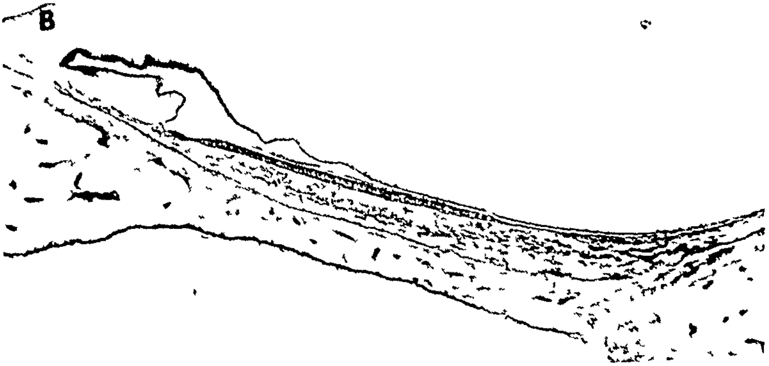

Fig 5-b Cat 3 Rupture and collapse of the saccular wall

was a tear in the saccular wall and a decrease in the hair cell population of the saccular macula The other vestibular sense organs appeared normal There was moderate injury to the organ of Corti involving the outer hair cells in two distinct regions of the upper basal turn, between 7 mm and 9 mm and between 10 5 mm and 11 mm (see Figs 5-a, 5-b, 5-c, and Fig 6-b)

Cat 4 Jr, left By an aseptic operation through the post-auricular approach, the oval window area was exposed, and the crura were fractured from the footplate and removed A fine needle was used to depress the anterior margin of the footplate A linear fracture occurred at the junction of the middle and posterior thirds of the footplate, with the anterior two-thirds being depressed into the vestibule This anterior segment remained in this depressed position There was some escape of perilymph which was controlled with the application of a small piece of gelfoam Sacrificed in three months

Histological examination showed that the anterior two-thirds of the footplate was slightly depressed into the vestibule The gap at the stapediovestibular joint anteriorly was healed by a thin two-cell layer thick membrane, and the fracture line was firmly healed by new bone formation There was a tear in the saccular wall and a decrease in the hair cell population of the saccular macula The other vestibular sense organs appeared normal There was moderate injury to the organ of Corti involving the outer hair cells in the region of the upper basal and lower middle turns between 10 mm and 12 mm (see Figs 7-a and 7-b)

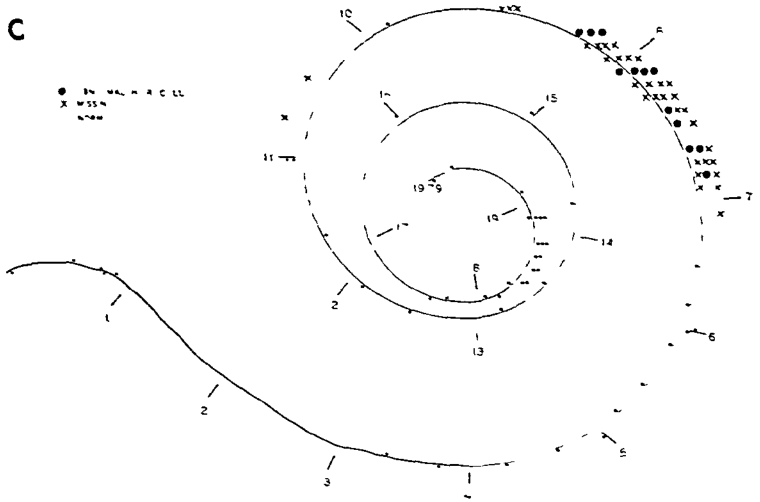

Fig 5-c Cochlear reconstruction showing severe loss of hair cells in the upper basal turn (see Fig 6-b)

McCabe and Lawrence[9] found tears and collapse of the saccular walls, as well as cochlear damage, in a high percentage of guinea pig ears subjected to high intensity noise

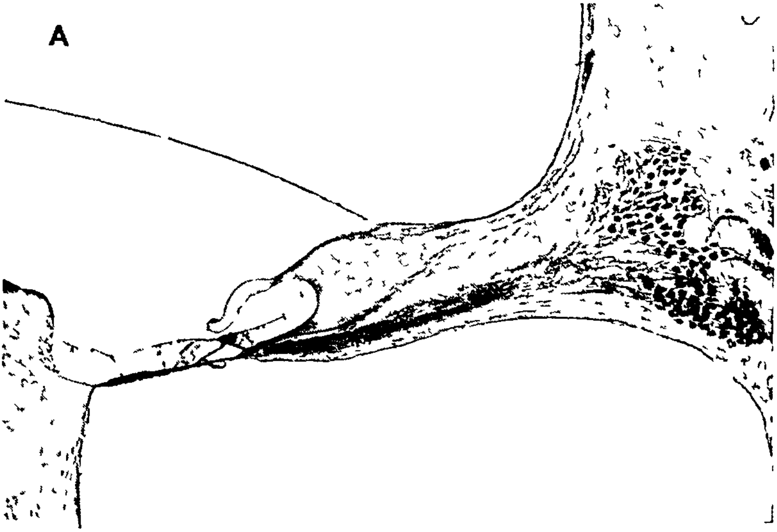

Fig 6-a View showing loss of outer hair cells of the organ of Corti in the upper basal turn, following an experimental blow to the head

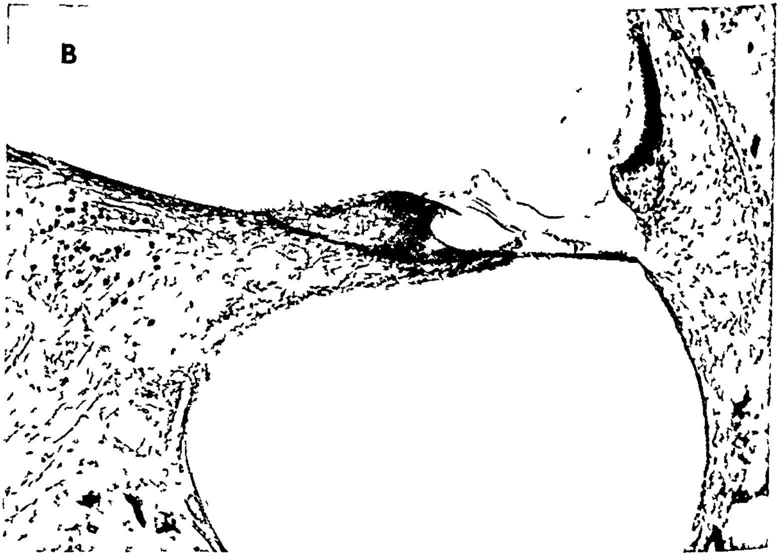

Fig 6-b Injury to the upper basal turn of the cochlea of Cat 3 showing rupture and collapse of Reissner's membrane extensive damage to the organ of Corti and secondary degeneration of spiral ganglion (See graphic reconstruction Fig 5-c)

In their animals, as in ours, there was no injury to the utricle and semicircular canals They suggested that this finding supported the concept of a functional dichotomy between the pars superior (utricle and canals) and pars inferior (saccule and cochlea)

There are two factors common to the injuries described in the foregoing *a* the lesions are confined to the region approximately 8 to 12 mm from the basal end of the cochlea, and *b* they have been caused by single blast-like acoustic events (or by their bone-conducted equivalents) Such events are commonly referred to as *transients* This statement necessitates a brief description of acoustic transients and of the cochlear response thereto After describing it first in 1933, Bekesy had termed the latter phenomenon the *traveling bulge*

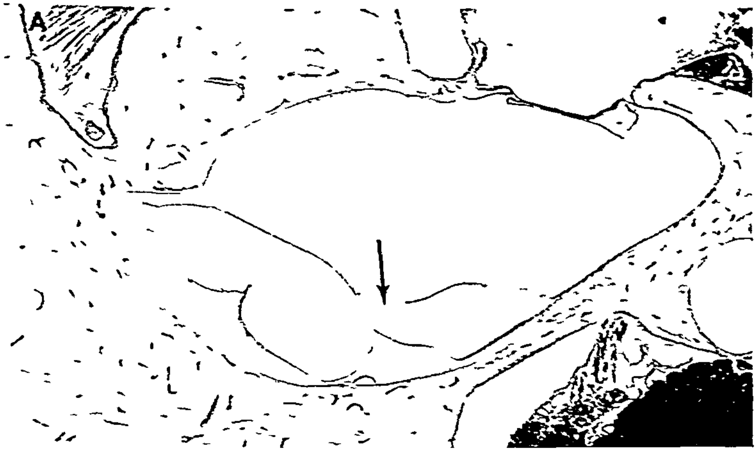

Fig 7-a Cat 4 Inward displacement of the footplate of the stapes Sacrificed in three months a—Low power view showing inward subluxation of the anterior two-thirds of the footplate and rupture of the saccular wall (Arrow)

Much of the following is derived from experiments on cochlear models built according to Bekesy's specifications Such models provide an excellent opportunity for studying mechanical events as they are most likely to occur in actual cochleae Very often interpretation of purely mechanical cochlear functions are made easier by the fact that these models, for instance, do not have a cochlear nerve attached to them. Their enlarged size permits visualization of the equivalent of events which themselves cannot be demonstrated in the ear by means of other presently known methods. Although ample evidence has been accumulated to the effect that qualitatively speaking such models are good replicas of the ear, quantitative interpretation of their results is somewhat more difficult.

Fig. 8 gives an example of the simplest case of an acoustic transient: a (gradual) step function. Shown are *a*—the time course of the applied signal, *b*— its spectrum, and *c*— the response of the model, *i.e.*, the traveling bulge and its envelope. It is noted that the envelope does not duplicate the signal spectrum. Spectrumwise, amplitude is inversely related to frequency, whereas the bulge forms a definite maximum along the partition. The position of this maximum along the partition varies with the inverse time constant of the signal. For brief time constants it is somewhat nearer the windows and vice versa.

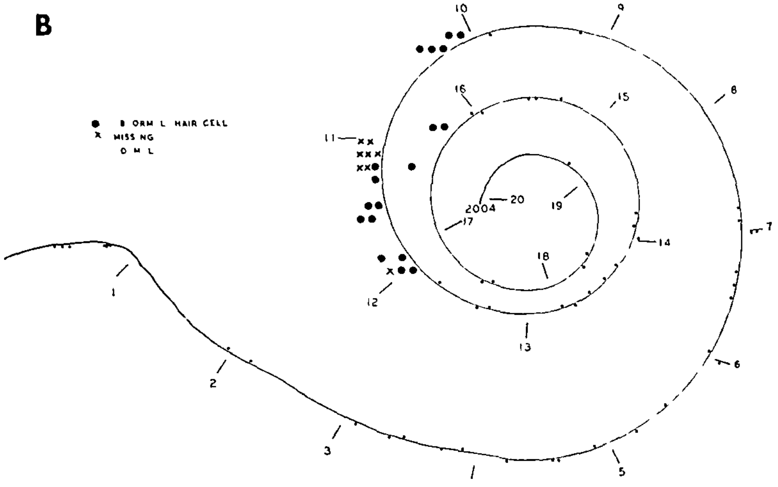

Fig. 7-b. Graphic reconstruction of the cochlea showing lesion of the organ of Corti in the 10 mm. to 12 mm. region.

This brief description may suffice to indicate the difficulty one faces when trying to explain the observations of cochlear injuries caused by stapedial manipulations (Figs 3 - 7) Although accurate data are not available, the motions of the footplate caused by such manipulations are likely to have relatively long time constants Even if one assumes that such injuries occur when the stapes footplate is broken loose (which appears quite logical), whereby the "snap" releases a large amount of energy quite suddenly, the involved time constant can hardly be expected to be sufficiently short to produce a bulge with significant displacement amplitudes along the basal portion of the partition The only point in which such stimuli stand out is their large amplitude At such occasions, stapedial amplitude may approach 1 mm Compared to that at the threshold of hearing (stapedial amplitude approximately 3 × 10^{-10} cm for a 1,000 cps signal) that is indeed an extremely

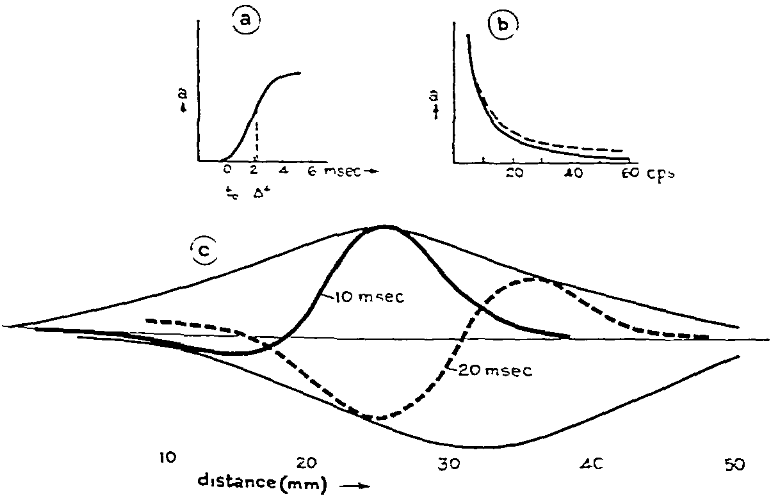

Fig 8 Transient response of cochlear model. a—Time course of signal gradual step function At time t_0 amplitude is gradually stepped up The time constant (Δt) the time at which 63 per cent of ultimate amplitude is reached is commonly used for definition of time courses b—Spectrum of above transient (solid line) and of an instantaneous step function (dotted line) The product of time constant and band-width approximates unity Therefore band-widths of signals with longer time constants are more restricted than those of signals with short time constants These spectra are of the continuous broad-band type c—Envelope over traveling bulge containing two instances of the bulge at an earlier (10 msec) and at a later time (20 msec) The envelope described only the maximum amplitudes of the first crests positive and negative

large value The notion had been expressed in an earlier communication[10] which was based upon some preliminary data that there might be a trading relationship between the inverse time constant and amplitude so that amplitude might make up for long time constants A number of parameters concerning cochlear fluid motion were examined in an effort to find such a trading relationship, yet in the end none was detected, therefore, as an alternate solution, the possibility of *nonlinear* distortion was considered

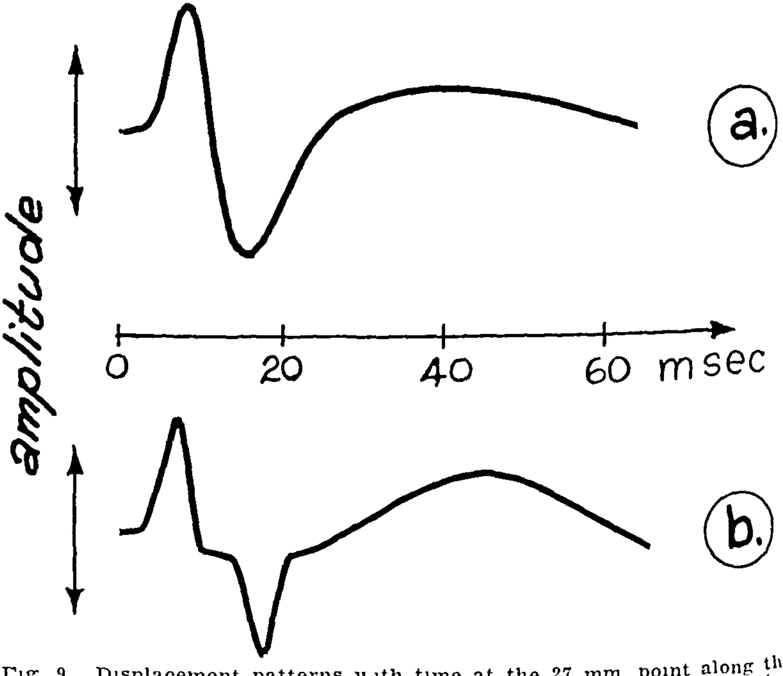

Fig 9 Displacement patterns with time at the 27 mm point along the partition a—without amplitude limitation, b—with amplitude limitation of the applied signal In high-speed motion picture films the spatial separation of the positive and negative peaks became quite noticeable

It is doubtful that the inner ear can follow very large amplitudes in a linear manner The elastic limitations of the annular ligament, if still present, and/or those of the round-window membrane are likely to be exceeded An amplitude limitation of the original window displacement is not simply followed by a like occurrence of the displacement pattern along the partition Since the displacement of the cochlear partition is 90° ahead in phase of the original window motion,[15] the ensuing phase alteration results in a *center-clipping* of the traveling bulge Fig 9 shows the displacement pattern of a point about halfway along the partition before and after amplitude limitation of the applied signal (It is noted in this drawing that displacement is plotted *vs* time in contrast to that of Fig 8 in which it had been plotted *vs* distance) The center clipping effect is quite obvious As a matter of fact when high speed motion picture films were taken of traveling bulges at the beginning of the present work on transients, a few such center clipped bulges had been produced inadvertently They were not recognized as such until the present phase of the work was reached These filmed bulges looked very peculiar with their spatial and temporal separation of successive peaks of the bulges

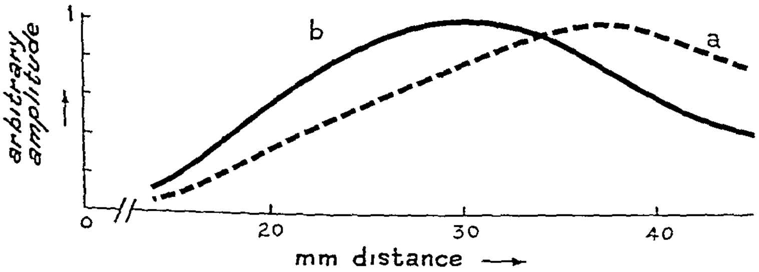

Fig 10 Envelopes over the second (negative) peaks of the displacement patterns of which Fig 12 gave two examples a—without amplitude limitation b—with amplitude limitation of the applied signal

Fig 10 shows that after amplitude limitation of the applied signal the envelope over the traveling bulge alters in that the amplitude maximum occurs more proximally along the partition The latter finding is easily explained The steeper slopes of the center clipped bulge have shorter time constants In this manner transient signals of long time constants but of extremely large amplitudes may produce traveling bulges of a shape which is normally associated with signals of shorter time constants; thus the assumption of nonlinear distortion would be able to explain the observation of cochlear injury after stapes mobilization. This explanation is admittedly not based upon direct evidence; it is offered as a hypothesis which appears to be consistent with other known facts of cochlear mechanics.

After this detail had been brought to a satisfactory solution, the present authors became interested in the more general problem of *acoustic trauma*. The cases presented in the foregoing are all very characteristic of acoustic trauma and, although from that standpoint not presenting anything new, confirm and document once more a familiar pattern: a hearing loss centered around 4,000 cps (the well-known 4,096 cps notch) corresponding to a localized cochlear lesion in the 8-12 mm region. Such lesions are characteristically produced by acoustic transients (or by their bone-conducted equivalents) of rather high peak intensities. This is in contrast to a so-called noise trauma which results from prolonged and repeated exposure to noise, usually of lesser intensities. The salient point is that whereas a noise trauma is most likely the outcome of a slowly accumulating exhaustion on the cyto chemical or enzymatic level without gross tissue destruction, acoustic trauma is the product of a *physical* injury involving local tissue lesions. Admittedly, much of what is finally seen histologically as the result of acoustic trauma is due to tissue degeneration *after* the injury; however, this is irrelevant from the present viewpoint and serves only to illustrate the apparent poor ability toward self-repair of the organ of Corti. In the following an attempt will be made to define the physical causes of acoustic trauma based upon purely mechanical considerations of cochlear stimulation and response.

The present inquiry to be sure is not the first into the problem of acoustic trauma. Two schools of thought appear to exist, specifically with regard to the origin of the 4,096 cps notch considered to be highly characteristic of acoustic trauma: *1.* the *mechanistic* view which holds that for one reason or another strong destructive forces develop in this particular region (*e.g.*, the "Dual-Eddy" Theory of Ruedi and Furrer[11] or the "Jet" Theory of Hilding[7]); *2.* the *locus-minoris-resistentiae* view which maintains that this region is especially vulnerable (*e.g.*, due to insufficient blood supply at the juncture of the cochlear and cochlea-vestibular arteries as suggested by Guild[5]). While not having anything new to offer, neither pro nor con, concerning the second viewpoint, the present authors feel that they have a new concept to present which concerns the possibility of a localized mechanical effect.

A brief comment may be in order concerning the cochlear action of headblows and of surgical manipulations, such as the use of mastoid gouges in the immediate vicinity of the inner-ear capsule. The stimulus is via bone conduction. In cochlear models, in which "bone" conduction can be demonstrated, the hydrodynamic events resulting from "air" and from "bone" conduction are exactly alike.[18] It is recalled that Bekesy,[2] and later Barany,[1] succeeded in mutual cancellation between air- and bone-conducted signals. Their results also serve to demonstrate the identity of these two modes of stimulation as regards their cochlear mechanism.

The stumbling block with respect to the localization of cochlear lesions caused by acoustic trauma lies again (as before in the problem of stapedial manipulations) in the fact that the amplitude in the basal portion is not the maximal amplitude. Fig. 8 presented a typical case with maximal amplitude at a point which is at 40 per cent of the total length of the partition; furthermore, the envelope over the displacement amplitude is rather broad (see Fig. 8) covering practically the entire length of the partition. Third, it is hard to realize why the most basal portion is so conspicuously spared. The question, of course, is whether with regard to the lesions observed the magnitude of the displacement amplitude is the significant factor. It appears not, according to the following simple consideration. If one tries to tear a piece of paper while holding it between both hands and suddenly moving them apart, it is not only important *how far* the hands move (*i. e.*, the amplitude) but also *how fast* such a motion is executed. Expressed more formally, the breaking point of the paper is determined by the *stress* produced during the action described. *Stress* is *force* per *unit area* and *force* is *mass* times *acceleration* (it is in the acceleration that the time factor comes in).

In applying the stress concept to the cochlea it appears that the mass (per unit area) of the tissues involved, *ie*, of the organ of Corti, does not vary greatly with distance It is, therefore, safe to state that the stress upon the cochlear partition encountered during its own displacement is in first approximation proportional to its acceleration From the data available on the transient response of cochlear models it is possible to derive the acceleration pattern, *i e*, the stress

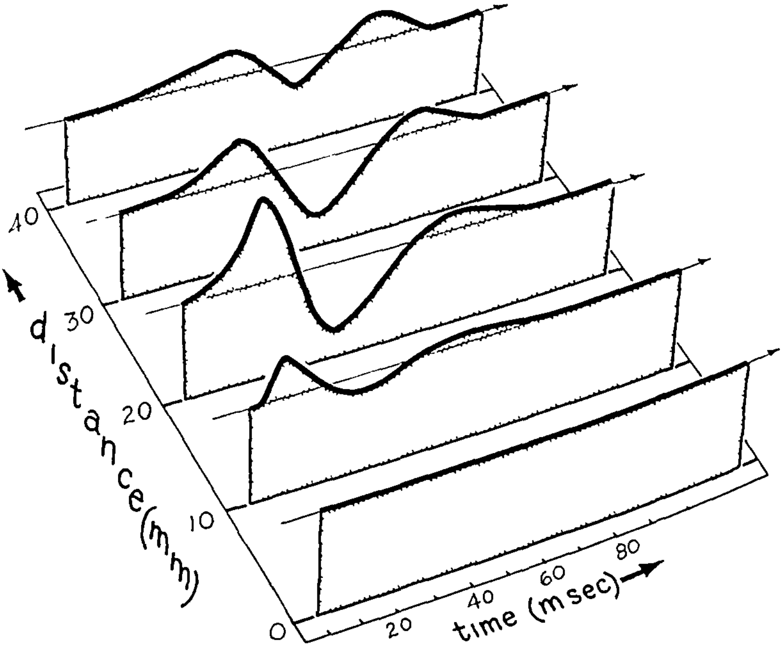

Fig 11 Displacement pattern with time at various points along the partition 3-dimensional presentation X-axis time Y-axis amplitude, Z-axis distance

Fig 11 shows once more the traveling bulge, this time presented in a 3-dimensional manner distance - time - amplitude Of the series of positive and negative displacements taking place at each point only the first positive peak is of interest here since it involves least time and consequently maximal acceleration The pertinent time factor, the *rise time* toward the first peak, is plotted *vs* distance in Fig 12-a, in a semilogarithmic manner The graph indicated that the rise time

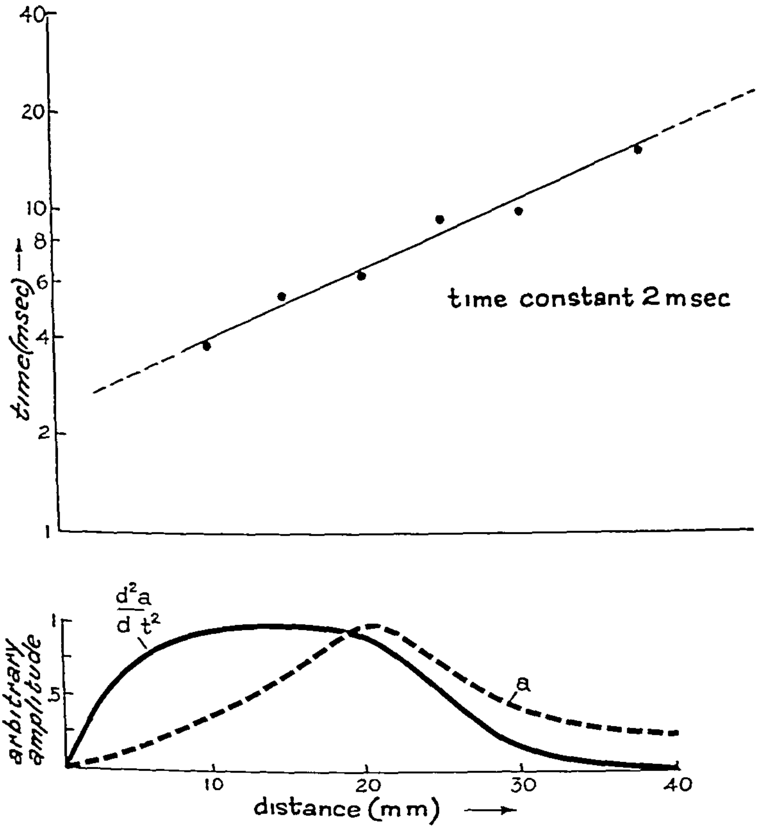

Fig 12 (above) Rise times of the first positive peaks of Fig 9 vs distance Time is given in a logarithmic manner (below) Envelope over the displacement amplitude (a) of the first peaks of Fig 9 and that over the corresponding acceleration pattern (d^2a/dt^2) Note—amplitudes in this and Figs 11-13 are arbitrarily set to unity

is shortest at the basal end of the partition, showing an exponential increment thereafter The ratio between the amplitude at each point (shown once more in Fig 12-b) and the square of the rise time gives the acceleration pattern along the partition (A more accurate way of doing this would have consisted in forming the second derivative of the time-displacement pattern of Fig 11 The difference between both methods is bound to be small) Fig 12 indicates that with reference to the envelope over the displacement amplitude the new envelope is shifted toward the direction of the cochlear base Although the maximum itself is still broad, the decrement of amplitude of acceleration with distance after the maximum is much steeper than before Since acceleration had been taken to indicate stress, Fig 12 shows that *most of the stress develops along the basal portion of the partition* Notably the extreme basal end remains free

Simple reconsideration of the operational steps involved in graphs 12-a and 12-b will show that the location at which maximal acceleration develops depends upon two factors *a* the place of maximal amplitude (which in turn depends upon the inverse time constant of the signal) and *b* the change in rise time of the bulge with distance Although the rise time is always shortest at the basal end of the partition, its rate of change again depends upon the inverse time constant of the applied signal This is illustrated by the example of Fig 13 The longer time constant makes the change in rise time less steep and produces the amplitude maximum farther distally than in the case of Figs 11 and 12 Consequently, the shift of the envelope over the acceleration pattern in Fig 13 is slightly less pronounced than in Fig 12, although it is again shifted in a proximal direction with respect to the envelope over the displacement amplitude Unless there is amplitude limitation due to overloading as in Figs 9 - 10, maximal stress will not in all cases be concentrated exactly in the same region in the basal portion

One other point deserves comment It was noted in connection with Fig 12 that the extreme basal end of the cochlear partition remained free of stress One might ask Was that true only for the particular situation depicted or is that a general rule? It would be logical to assume that for very short time constants the location of maximal stress should approach the basal end of the partition Although this is correct, it turns out that the *sensitivity* of the model to signals with such brief time constants is so low that no sizable displacement amplitude, and thus no appreciable stress, can develop unless it comes to very large signal amplitudes The low sensitivity of the cochlear partition to signals of brief time constants is a built-in property Basically, the model represents a series of low-pass filters Their upper-frequency limits vary from high to low along the partition These filters

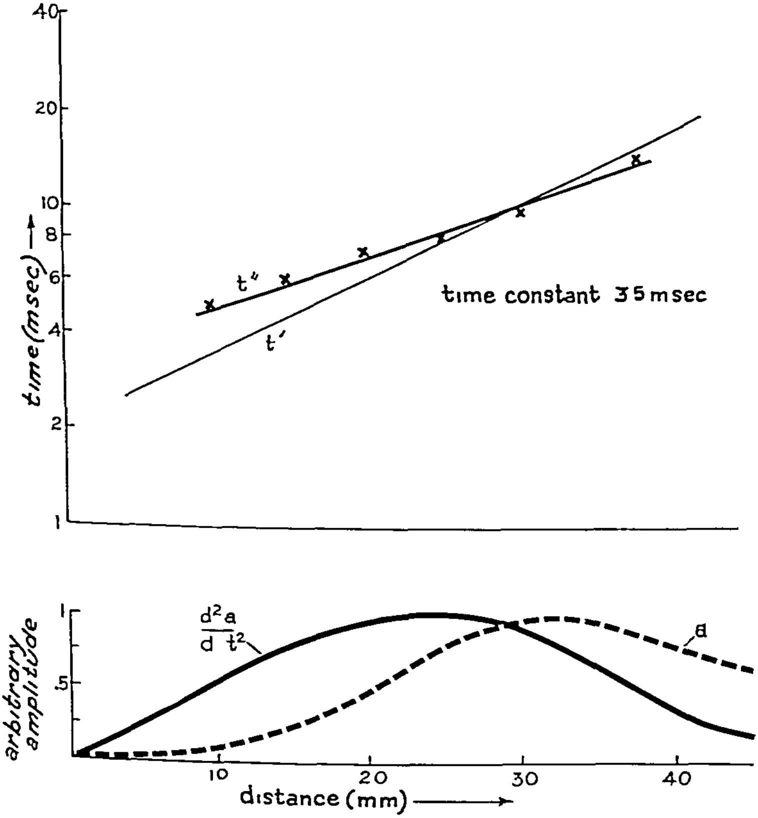

Fig 13 (above) Rise time (t) of the first positive peaks vs distance for a signal with slower time constant For the sake of comparison the function of Fig 12-a (t') is included (below) amplitude (a) and acceleration ($d\,a/dt^2$) pattern for the same signal

are not sharply tuned, nonetheless, there is a definite lessening of sensitivity as frequency exceeds a certain value, or what amounts to the same, as time constants become too brief, and optimal stress is a *compromise* between the *decreasing* sensitivity and the *increase* in rise time of the traveling bulge with inverse time constants of the applied signal Because of the spatial distribution of frequencies along the cochlear partition *optimal stress must develop some distance from the extreme basal end of the partition*

The question to be considered next is whether the stress associated with the displacement pattern along the cochlear ment of amplitude of acceleration with distance after the maximum is much steeper than before Since acceleration had been taken to indicate stress, Fig 12 shows that *most of the stress develops along the basal portion of the partition* Notably the extreme basal end remains free

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These results are the more significant as in cochlear models, when the response to noise bands is tested, the amplitude peak invariably occurs *below* the upper frequency limit of the pass band; therefore. Meyer z G's results when interpreted from the standpoint of the present concept seem to indicate that the most effective stress is always caused by the highest frequency in the band. *e g.* the frequency which (in terms of its traveling bulge) has the fastest rise time The upper limiting frequency of 4,000 cps then represents a *compromise* between the *decrease* in sensitivity toward higher frequencies and the still *increasing* rise time. The point of highest sensitivity of the human ear is known to be between 2,000 cps and 3,000 cps, *i.e.* definitely lower than 4,000 cps

SUMMARY.

It is well known that high energy acoustic signals such as those created by a blast in air or a blow to the head, can cause cochlear damage having its maximal effect in the upper basal turn. Such lesions can also be created by manipulation of the stapes during the course of surgical procedures The amplitudes of such maneuvers can be very large and the time constants are long. The resulting cochlear injuries are identical to those resulting from stimuli having short time constants.

Animals with experimentally produced footplate fractures frequently sustain rupture of the saccule, as well as injury to the upper basal and lower middle turns of the cochlea. Sac-partition is really representative of that leading to cochlear injury, *i. e.*, to the loss of hair cells in the organ of Corti. Since the latter results from overstimulation it may be well to state what constitutes the normal stimulation of the sensory cells in the organ of Corti. According to Bekesy's experimental evidence,[4] the ultimate stimulus to the hair cells consists of a bending of their sensory hair. This bending is caused by a *shearing* motion between the tectorial membrane and the organ of Corti proper. Bekesy[4] had distinguished two modes of shear motion which, in response to sinusoidal stimulation, occur in two distinctly different places: a. *radially directed* in the region proximal to the amplitude maximum of the traveling bulge and b. *longitudinally directed,* in the region distal to that point.

Shearing action can be duplicated in cochlear models.[15] An attempt was made to study its pattern in response to transient stimulation. Due to the minuteness of the amplitudes involved, no precise measurements were obtained; however, the following impressions were gained. The two different modes (radial and longitudinal motion) overlap each other widely, the radial motion forming its maximum more proximally than the longitudinal mode. With respect to time, the longitudinal motion precedes the radial motion. The envelopes over the two modes were found not to differ radically from that over the traveling bulge. Rise times at each point are identical to those of the traveling bulge; therefore, the conclusion seems justified that the *stress pattern in the domain of shearing motion* is *very similar to that of Figs. 12 and 13.*

The concept presented here, that acoustic trauma is determined by the distribution of *stress* along the cochlear partition, must also apply to pure tones. Stress was said to be proportional to acceleration. In sinusoidal events, acceleration varies with the square of frequency. This is borne out by experience. It is the consensus of opinion that (in the words of Davis, et al.[6]) "higher frequencies are more effective than low frequencies in producing hearing loss."

Of particular interest from the aspect of exposure to transients is a recent study by Meyer zum Gottesberge.[10] This

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AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY HOME STUDY COURSES

The 1960-1961 Home Study Courses in the basic sciences related to opthalmology and otolaryngology, which are offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on September 1 and continue for a period of 10 months Detailed information and application forms can be secured from Dr William L Benedict, the executive secretary-treasurer of the Academy, 15 Second street S W, Rochester, Minn Registrations should be completed before August 15 cular rupture also occurred in one of ten animals subjected to head blows The cause for the saccular injury is not clear

A new concept is developed concerning the mechanism of acoustic trauma The assumption is made that the ensuing tissue damage is a function of the physical stress resulting from acoustic stimulation Experiments on cochlear models using transient signals indicate that the closer the stress is concentrated to the basal end of the cochlea the shorter is the time constant of the applied signal Since the sensitivity of the model falls off for signals with very short time constants, a region of optimal stress develops close to but not directly at the extreme basal end of the cochlea

It is possible that signals of relatively long time constants, but of very large amplitudes, produce injurious stresses in the basal region of the cochlea due to nonlinear distortion

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scriptions of the 1890-1910 period and how little has been contributed since

To Valsalva in 1735, is attributed the first gross description of ossification of the annular ligament of the fenestra ovalis as a cause of deafness Other great anatomists like Morgagni in 1766, and Meckel in 1777, confirmed this observation Toynbee in 1844 dissected 41 temporal bones and found one case of bilateral ankylosis of the footplate of the stapes By 1857 Toynbee had studied 1,659 temporal bones and found 92 cases of osseous ankylosis of the stapes to the fenestra ovalis He called it one of the most common causes of deafness

In that era, many authors described grossly the pathological picture of otosclerosis, among them Voltolini, Politzer, Lucae, Schwartze and Troeltsch Moos reported an associated hyperostosis of the petrous bone and the narrowing of the porus acusticus internus

The first histological study listed in the American Otological Society Resume of Literature on Otosclerosis is by Haberman in 1891 He discussed the sharp characteristics differentiating pathological bone from normal bone, calling attention to the deeper staining with hematoxylin and carmine, the increased size of the marrow spaces, the very irregular construction of the bone, and the dilated vessels He remarked that the diseased bone looked like calcified cartilage This description of otosclerosis is still faultless today

Bezold in 1893 added the description of young osteoid tissue with many cellular marrow spaces and osteoclasts forming eroded lacunae The normal cartilaginous portion of the footplate was almost entirely replaced by osteoid tissue, and the anterior part was eight to ten times thicker than normal, due to new bone formation

In 1893 Politzer also concluded from his investigations that the disease originated from the labyrinthine capsule and extended to the stapes, finally leading to ankylosis, he also described round window and labyrinth involvement, and felt that the distinctive staining and the large marrow spaces were the important criteria for diagnosis of otosclerosis

HISTOPATHOLOGY OF THE OTOSCLEROTIC FOOTPLATE*

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and
COLETTE JEANTET, M D ,
Chicago, Ill

While a great deal of histopathological study of otosclerosis has been done at the level of macroscopy and light microscopy, for several reasons comparatively little has been contributed by the more advanced techniques of histopathology

First, the study of undecalcified bone is a difficult one and the technical problems, while not insurmountable have hampered progress in this direction Second, the only otosclerotic bone available in the past was obtained at autopsy or in recent years during fenestration, in the small percentage of cases where the cupula or an ossicle presented an otosclerotic involvement

With the advent of the Shea fenestration of the oval window, the footplate of the stapes is removed as entirely as surgically possible, and large amounts of biopsy material have become available in this way

The authors of this paper have collaborated and pooled their material in an attempt to contribute to the knowledge of histopathology of otosclerosis

In reviewing the literature on otosclerosis, it is interesting to note how detailed and accurate were the pathological de-

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*Read at the meeting of the Middle Section of the American Laryngological Rhinological and Otological Society Inc Chicago Ill Jan 18 1960

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In 1900 Manasse described typical Howship's lacunae with osteoclasts and coexistent bone apposition

Holinger in 1901, studied Siebenmann's specimens of stapes ankylosis with labyrinthine spongification He observed resorption and new bone formation occurring simultaneously, and replacement of interglobular spaces by newly formed bone, also the cartilage of the footplate was replaced by bone

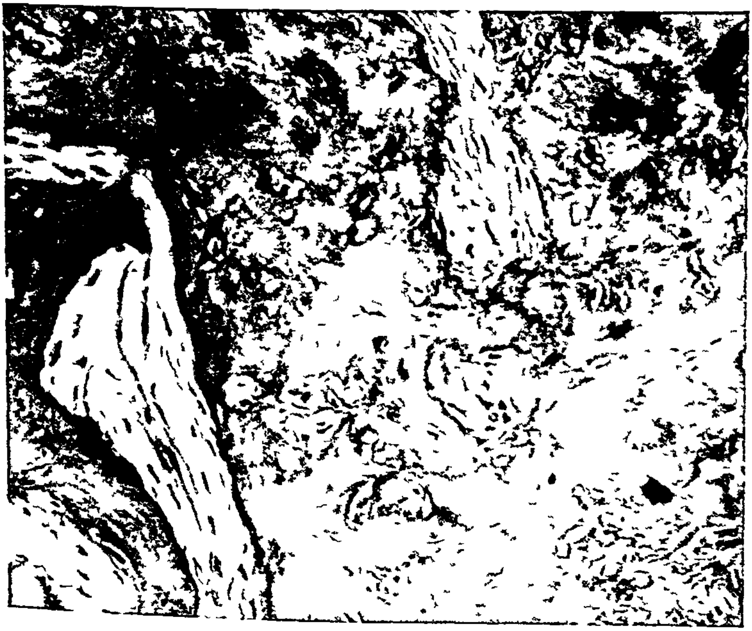

Fig 2 Otosclerosis Type III Fewer osteocytes Nuclei sometimes absent Absence of lamellation Marrow spaces less cellular

in connection with large blood vessels parting from the bone The spongy spaces were so large that the process could be called a true osteoporotic one, except that there was much new formation of bone He stated that sclerosis and similar words to indicate this disease are without meaning, pathologically or clinically "Spongification of the labyrinth is a well defined disease clinically or pathologically"

In 1905 Alexander reported a case of congenital deafness

Hartman in 1898 observed the abnormal shape, size and arrangement of the osteocytes The fact that they were more numerous in the vicinity of normal bone lead him to believe that otosclerosis consisted of newly developed connective tissue bone

Siebenmann in 1899 described absorption of bone by osteoclasts with dilatation of the Haversian canals and increased connective tissue spaces The newly formed osteoid zones showed irregularly arranged misshapen bone cells with abnormal extension processes In addition the interglobular cartilaginous spaces were replaced by newly formed bone

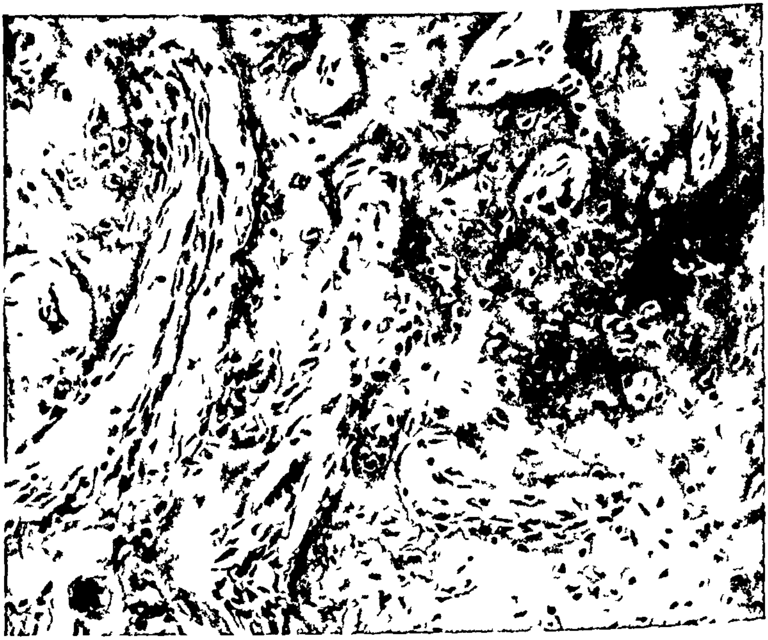

Fig 1 Otosclerosis Type IV Large number of osteocytes misshapen and not oriented Absence of lamellation Large cellular marrow spaces

He wrote that as the disease ages the spongy character disappears, replaced by a lamellar structure with concentric arrangement of bone cells with shrunken nuclei That which remains spongified proves to be old in its staining, the reaction is then strongly eosinophilic the composition of the marrow substance which shows more nuclei and connective tissue fibers than that of the ordinary spongiosa

In 1912, Alexander described otosclerotic foci as follows A very cellular bone rich in calcium, presenting remarkably large cavities which are traversed by numerous pathologically

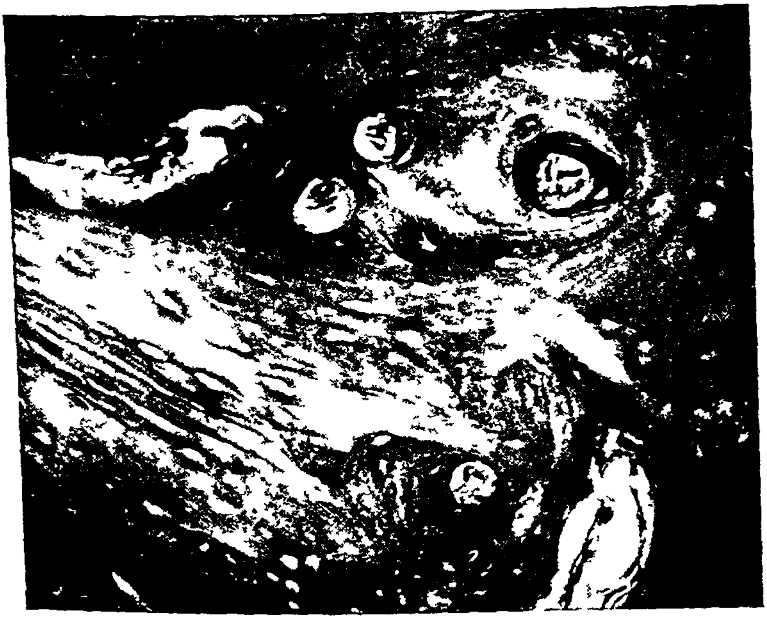

Fig 4 Otosclerosis Type I Well oriented osteocytes nuclei often missing Regular lamellation Marrow spaces narrow and inactive

enlarged blood vessels Between the bone and the vascular wall is a fairly dense network of connective tissue

Neuman in 1913 refuted a suggestion of Mayer that otosclerosis is identical with osteitis fibrosa of Recklinghausen His objections were founded on the absence of pronounced absorption in the sense of rarefaction in otosclerosis, absence of softening, granulating tissue and cysts and on the preservation of the compact character of the labyrinthine capsule in spite of the most radical transformation of its constituents with partial stapes ankylosis and typical otosclerotic foci in the labyrinthine capsule He used the term "ground substance" and stated that it was more deeply stained by hematoxylin in otosclerosis than the ground substance of normal bone

Bruhl in 1905 described the bluish coloration of the bone (blue spongiosa) replacing the annular ligament in a posterior

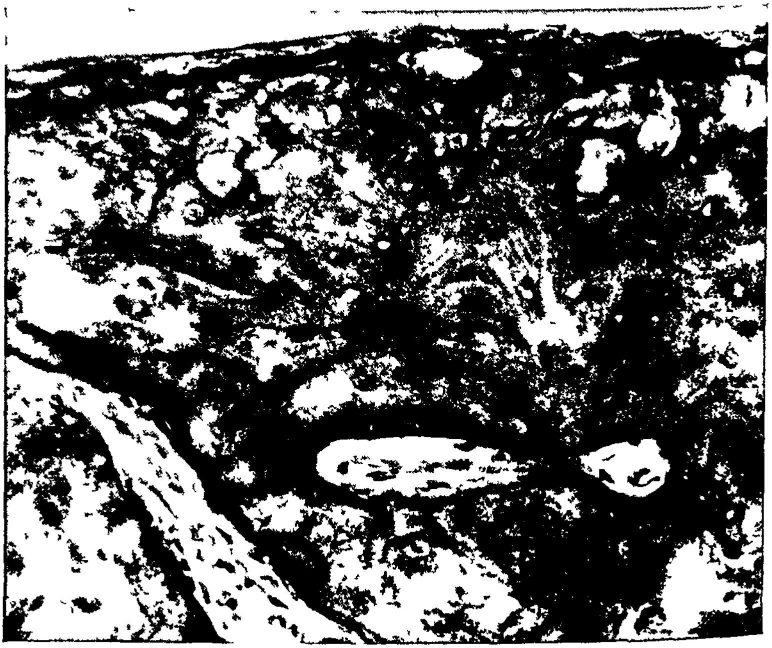

Fig 3 Otosclerosis Type II Nuclei often missing Presence of lacunar canaliculi Attempt at lamellation Marrow spaces small new bone apposition

process There the osteocytes were large, misshapen, and numerous, whereas in the more anterior focus staining pink, there was a concentric arrangement of small bone cells He later said that the combination of active formation of bone by osteoblasts and breaking down by osteoclasts produced an entirely new bone so that the pathological bone always appeared like a foreign body

In 1906 Schilling added to the usual list of characteristics, osteoblasts, new bone formation along the walls of the marrow spaces and distinctly red staining

In 1922 Rutin reported a case of otosclerosis associated with fragilitas osseum (osteopsathyrosis) and blue sclera Although the bone destruction was extensive, to a degree not common in otosclerosis, typical, dense otosclerotic bone was present in the usual site, at the anterior margin of the stapes

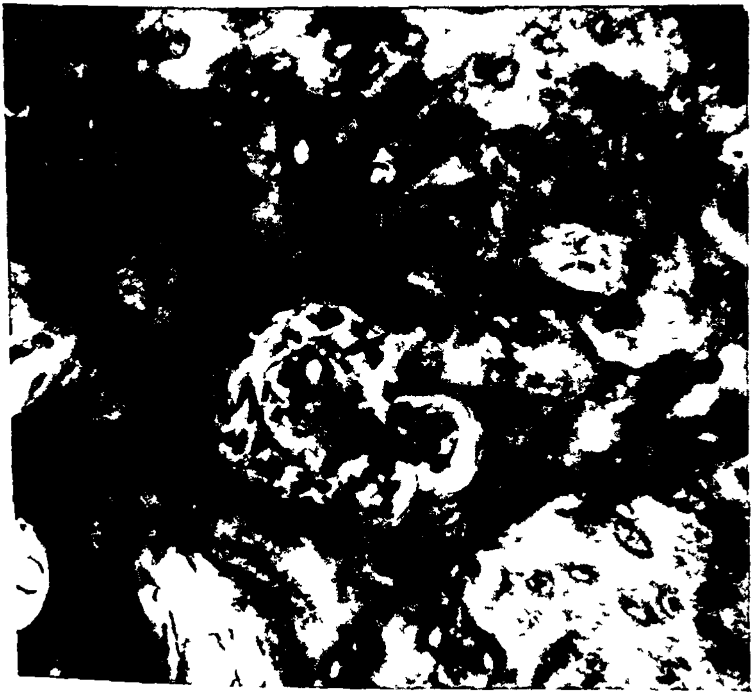

Fig 6 Type IV otosclerosis showing a marrow space with an osteoclast actively destroying bone together with new bone apposition on the opposite margin of the space

In 1923 Jenkins presented two cases of osteitis deformans with deafness and compared his findings with those of otosclerosis Both diseases showed osteoporosis, but the trabeculae in osteitis deformans were coarser and the bone lamellae more distinct The limits of osteoporosis were sharply seen in otosclerosis but found only by careful search in osteitis

In 1914 Komendentanow found many points of similarity between the bony changes characteristic of rachitis and those characteristic of otosclerosis

Between 1917 and 1921, Wittmack, Mayer, Manasse, Fisher, Lange, and others reported large series of cases with the now familiar description of the pathological changes of otosclerosis, the authors arguing only on the significance of some of these changes

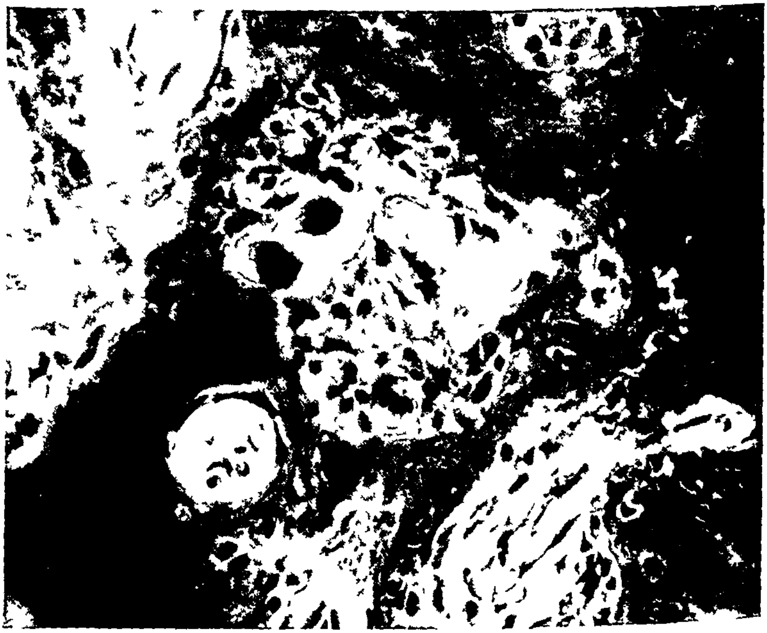

Fig 5 Two osteoclasts in a Type IV otosclerotic footplate

Escat in 1922 describes three phases in the pathology of otosclerosis The first phase consists of vascularization and return to the osteoid stage The second phase is that of osteospongiosis with diminution of the size of the capillaries, enlargement of the marrow spaces, appearance of osteoclasts, deep blue staining The third phase is the rebuilding of a compact osseous tissue, as otosclerosis, with appearance of osteoblasts, new bone formation along the walls of the marrow spaces and distinctly red staining

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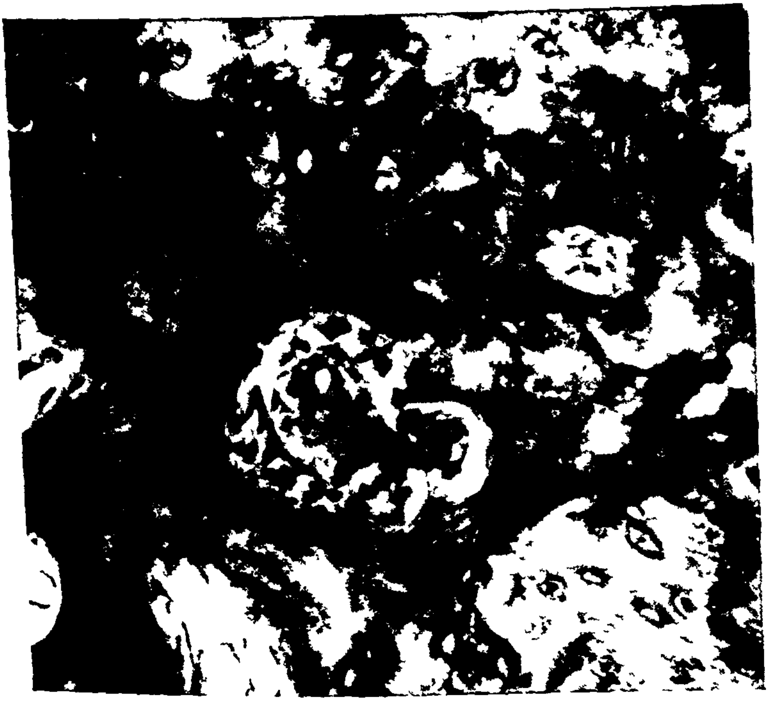

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Weber, in 1933, wrote an excellent discussion of the blue mantle in otosclerosis He defined the blue mantle as beginning as an area of resorption The cavitation thus formed represents a Howship's lacuna The blue mantle represents new bone formation after the previous resorption of the vessel space The new bone formation has a pathological matrix consisting of blue staining cementum and sparse criss-crossed fibrils forming a plexus He was the first to study otosclerosis under polarized light

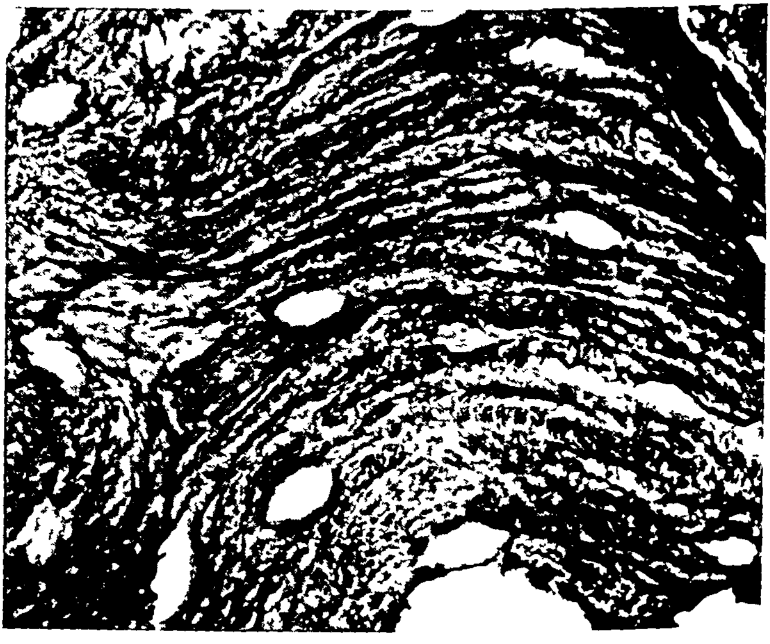

Fig 7 Appearance of the fibrillar substance in a well lamellated bone (Silver stain)

Greifenstein in 1935 showed that after traumatic injuries the marrow tissue of the young otosclerotic focus exhibits a lively regenerative activity in the sense of normal bone formation

In the following years one finds far less literature concerned with the descriptive pathology of otosclerosis The contributors include F R Nager in 1938 and 1939, S R Guild in 1944, Nylen in 1949 In 1950, S R Guild pointed out that nearly 30 per cent of the cases of otosclerosis he studied were unilateral and was concerned with the physiological fact that in any person the blood has supplied both otic capsules with the same nutritional factors, for identical lengths of time He apparently did not feel that constitutional factors and otosclerosis were in any way related

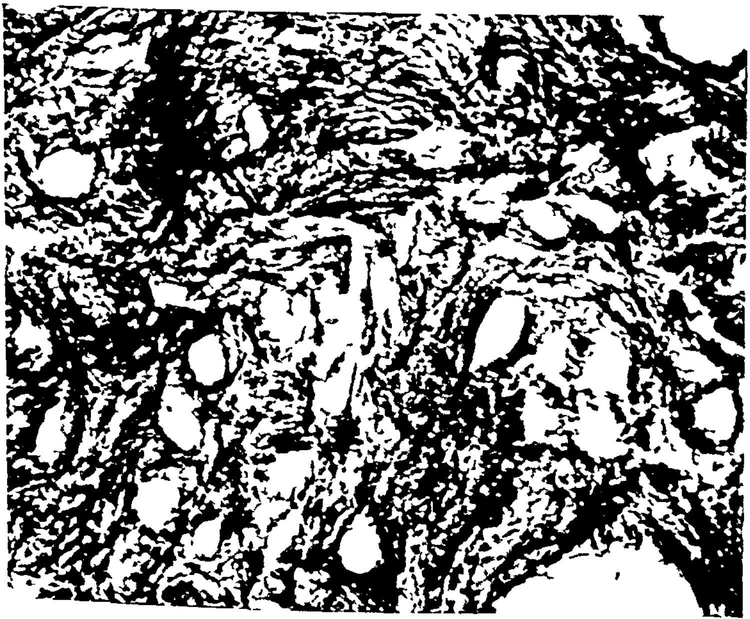

Fig 8 Disorientation of the fibrillar substance in otosclerosis (Silver stain)

Lindsay showed that an attempt to link otosclerosis with other general constitutional factors has so far demonstrated no definite relations either in clinical or in experimental investigations

Bast felt that otosclerosis could be an attempt to rebuild that part of the otic capsule which was left unstable by late fetal cartilage growth especially in the fissular area

In 1951 Nager, and Hale in 1952 and 1953 studied specimens taken at surgery from the site of the fenestra novovalis

In 1952 Brunner introduced the idea that there are preotosclerotic changes, consisting of degeneration of the cartilage at the frame of the window and of the appearance of the blue mantles previously described by Manasse These blue mantles are a broadening of the normal marginal sheaths of the blood vessels These are called preotosclerotic changes because they are found throughout the labyrinthine capsule in otosclerosis and in some temporal bones with no actual otosclerosis He felt that they are hereditary and do not produce clinical symptoms by themselves

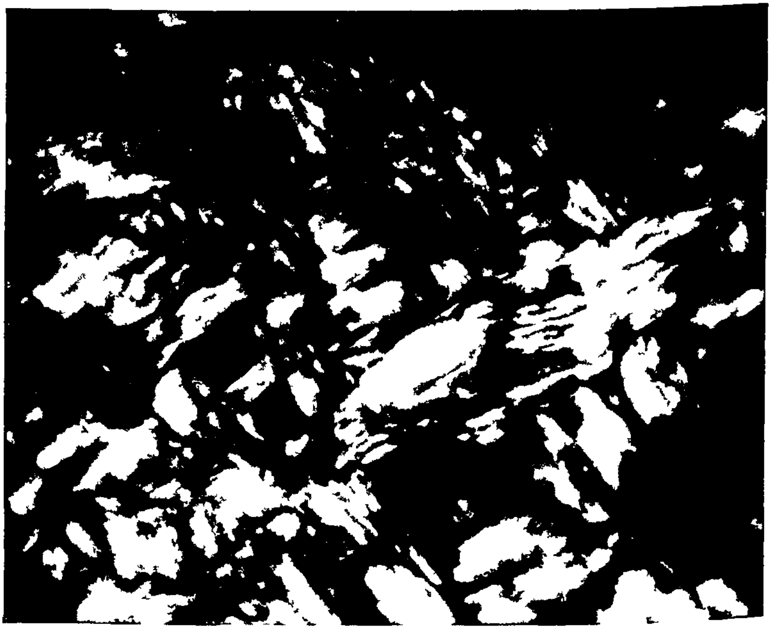

Fig 9 Braided appearance of otosclerotic bone on polarized light

In 1953, R F Ogilvie and I S Hall published a thorough description of otosclerosis of the fenestration site of 13 per cent of 125 otosclerotic subjects They remarked that the diffuse damages involve absorption and deposition of the bone through osteoclastic and osteoblastic activity respectively and thereby leads in sequence to an abnormally cancellous, mosaic, and relatively normal architecture of the bone The immature bone is basophilic, excessively cellular and woven, while the mature bone is acidophilic, normally cellular, and laminated As for the so called blue mantles they are regarded as a focal manifestation of the diffuse damage

Bellucci and Wolff in experimental animals created surgical traumas at the oval window, and reported the effects of cautery on the ossicles, and labyrinthine bone They were interested in the question of healing after stapes mobilization for otosclerosis

In order to provide a basis for discussion and mutual understanding of the types of otosclerosis involving the footplate, we have attempted a classification based on the degree of activity of the disease The experience with the first 120 footplates of otosclerotic stapes makes the basis for this report

The criteria determining this classification are primarily histopathological, but some consideration was given to the gross structural findings For instance, in a few cases, without the clinical and microscopic observation of annular involvement of the stapes and of the resulting ankylosis, some of the Grade I, stable, almost normal appearing footplates can be very difficult to diagnose as otosclerotic, also, the character of the focus may be such that most of it has to be removed by drill and escapes pathological study In some instances of diffuse anterior involvement the surgeon must be satisfied with the extraction of the posterior two-thirds of the footplate only These factors should be kept in mind as such specimens are not typical of the total disease, however, we were impressed with the findings of almost universal involvement of the stapedial footplates

The evaluation of the typical pathological features of otosclerosis should deal primarily with the cellularity of the process The osteocytes are more numerous than in normal bone, also, in normal bone, about 50 per cent of the osteocytes are vital as evidenced by the presence of nuclei The number of vital cells in active otosclerosis seems to be markedly increased The cells appear misshapen, disoriented with swollen, or distorted and pyknotic nuclei, and the cytoplasmic processes lying in the canaliculi are absent

A second feature of evaluation is the increase in marrow spaces In the normal stapes, the footplate shows few marrow spaces and the crura none, as a rule In otosclerotic bone, there is an increase in number and activity of the cells of the marrow spaces resulting in resorptive changes forming new spaces as indicated by the presence of osteoclasts At the same time and sometimes in the same spaces osteoblasts are seen with new bone apposition

A third most important feature of otosclerosis is the character of the ground or intercellular substance Intercellular substance consists of two major elements fibrillar substance and cementum In otosclerosis, the amount of the basophilic cementum is increased Otosclerosis is characterized by an immature type of bone, and the immature bone has been described as having a coarse fibrillar substance, but in our slides, thus far, we have not seen it Our findings indicate that the fibrils are fine, sparse and make patterns in the bone that appear braided or woven There is often a cementum line between these bundles which appears as a sharp break with silver stain or on polarized light The total unit volume of fibrillar substance is reduced, not only because of its fineness and sparsity but also because of the increased amount of marrow space and osteocytes

These histological features and their variations should be kept in mind when one undertakes to grade otosclerosis One should also note that if it were not for its remarkable and unique location in the temporal bone and for its, as yet, poorly understood relationship with genetics, otosclerosis is difficult to differentiate histopathologically from any other type of immature bone

We have classified otosclerosis in four main types

Grade IV is the most active otosclerotic focus of the more immature type It shows an increased cellularity to a very marked degree in the heavily thickened bone of the footplate, with irregular shape of the osteocytes, swelling of the nuclei, absence of demonstrable canaliculi and of lamellation

The marrow spaces are increased in size and number and show extensive vascularization They contain many osteoblasts and some osteoclasts Frequently in the same space, one may observe appositional surfaces of new bone formation contiguous to a very active process of bone resorption by osteoclasts The intercellular substance consists mainly of blue staining cementum with sparse braided bundles of fibrils

There usually is a complete absence of the cartilaginous elements in the footplate, a destruction of the annular ligament and an encroachment beyond the normal confines of the stapedial borders This process should be termed histopathologically, otospongiosa

A Grade III type of otosclerosis is often less extensive The cartilaginous elements of the footplate may have disappeared, and the ligament is totally involved The process cytologically appears definitely less active, there is some attempt at a more regular arrangement of the osteocytes which are fewer in number Their nuclei are sometimes shrunken, misshapen, and occasionally absent The marrow spaces are still large from the previous activity but contain less cells in a loose network, and more active bone apposition is taking place There is less blue staining cementum and more fibrillar substance in the otosclerotic bone.

Grade II otosclerosis shows patching invasion of the ligament and sparse involvement and calcific changes of the cartilaginous portion of the footplate The lacunae in a larger percentage than normal show the presence of osteocytic nuclei, but they are pyknotic In Grade II the marrow spaces are inactive, smaller and less cellular The woven appearance of the fibrillar substance is fuller and occasionally attempts to organize in a lamellar pattern The cementum is lessened The histological findings of invasion of the cartilage and annular ligament further substantiate the pathological diagnosis There is a tendency to the appearance of some cytoplasmic processes in the canaliculi

Grade I otosclerosis, the most inactive of the four types, shows a slight increase in the number of osteocytes The nuclei are shrunken The cytoplasmic processes appear with more regularity The lamellar arrangement on hematoxylin and eosin stain is quite regular There are few or no marrow spaces The cementum is not remarkably increased Grade I then would be difficult to diagnose at all except for three findings

1 Calcification of the cartilaginous portion of the footplate

2 Invasion of the annular ligament

3 Under polarized light or with silver stain the fibrillar substance appears braided rather than lamellated

SUMMARY

From the information obtained from our study of the literature, the dynamic approach of present day bone physiology, and the study of these surgical specimens, we present a description of our present theory of the development and extension of the otosclerotic focus *I* The vascular budding invades normal endochondral bone in the advancing or developing process *II* The marrow space is then created or enlarged by the action of osteolytic processes carried out primarily by the osteoclasts *III* This resorption leaves a Howship's lacuna with exposed fibrillar substance some of which is digested along with the resorbed ground substance *IV* The newly formed immature bone rich in cementum and poor in fibrillar substance creates a thickened bluish resting line at right angles to the previous linearity of the invaded bone The bone filling in the defect is highly osteoblastic at this phase and appositional but immature in type (Type IV) *V* As this phase becomes exhausted, the bone appositioning becomes more adult, leading to an osteosclerosis (shading down to Grade III, II, and I) *VI* This process occurs within the same focus, at different phases and at the same time, however certain "metabolic crises" may activate a whole area into advancing pathology The perivascular process of bone formation is rich in cementum *VII* Items I, II, III, and IV are what Manasse called the blue mantle

CONCLUSION

Otosclerosis can be considered as another genetic connective tissue disease wherein the advancing vascular buds lay down sparse abnormal fibrillar substance and excess cementum In conjunction with this disease of the intercellular substance is the excessive cellular activity of the abnormal bone in its most active phase Thus far, the questions as to why it is prominent in the temporal bone, why it can remain dormant for so long, and why it shows no variance from other immature bone remains unanswered

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PLASTIC AND PROSTHETIC REPAIR FOR PATIENTS WITH COMPLETE LOSS OF THE LOWER JAW*

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Complete loss of the mandible fortunately is an uncommon deformity. The patient with an epithelioma so large that the entire inferior maxilla must be sacrificed practically never is cured, and consequently reconstruction of the lower jaw rarely need be considered in such a case; however, there are certain pathologic conditions that involve total removal of the mandible and that do not affect the patient's span of life; in such instances, plastic repair of the defect is of the utmost importance. The conditions to which I refer are osteomyelitis, radionecrosis, and tumors such as adamantinomas involving the entire lower jaw. Then, too, trauma occasionally accounts for loss of the whole mandible.

Total reconstruction of the inferior maxilla by autogenous bone grafts is completely unsatisfactory from both the esthetic and the functional points of view, because there are no muscle attachments by which the graft can function as a mandible. Even though the ordinary kind of intra-oral prosthesis may be ineffective so far as mastication is concerned, it does restore facial contour in such cases and offers the only satisfactory means of reconstruction. In recent years I have found that an intra-oral prosthesis can be prepared with modifications that make it self-retaining (as much as any full lower denture) and allow for mastication of soft foods, while not neglecting cosmetic objectives (see Fig. 1).

As heretofore, the installation of a prosthesis to replace a lost mandible involves preparation of a skin-lined pocket in the mouth. In all cases in which the bone of the lower jaw

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is completely removed, the soft tissues of the lower half of the face become retracted and distorted by scars An intraoral incision made behind the lower lip, chin, and cheeks frees these structures so that they may be returned to a more normal position, however, this procedure leaves a raw cavity in the floor of the mouth The prosthesis for holding the lower lip and chin forward cannot be inserted into this pocket until the cavity has been lined with a split-thickness skin graft Construction of a stent (mold) to immobilize such a skin graft for a few days while it heals to the walls of the cavity poses a problem, because the mandible is absent, and no lower teeth are present to which the mold can be fixed To meet this situation, I have prepared a mold in two segments as shown in Fig 2

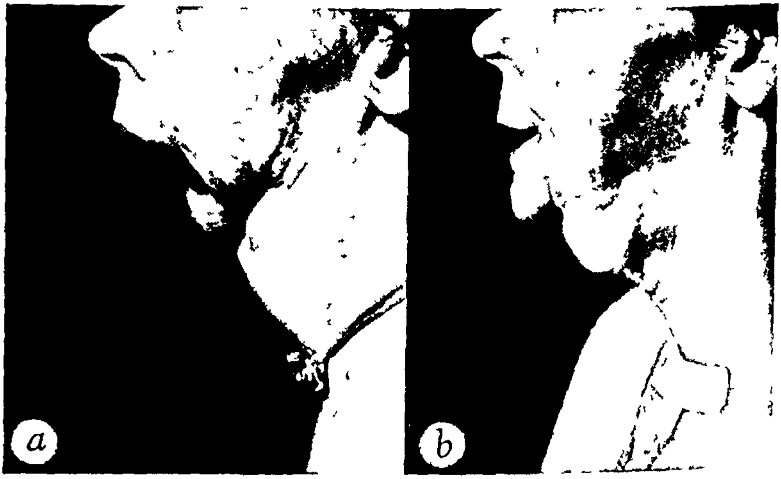

Fig 1 a—Complete loss of mandible from surgical removal of an adamantinoma b—Deformity corrected by combined use of intra-oral prosthetic appliance and clavicular tube flap of skin inserted in floor of the mouth and submental region

In the construction of the upper segment, softened dental compound contained in a metal tray is employed to obtain an impression of the upper dental arch and palate When the compound has cooled and hardened, it is removed from the mouth and tray, after which it is reinserted into the oral avity to form a sort of splint covering the upper teeth and alate (see Fig 2)

In preparing the lower segment, a large quantity of softened ental compound is introduced into the surgically prepared ocket back of the lower lip and cheeks With the upper egment of the mold in proper place over the upper jaw, the atient bites into the softened compound below and holds the maxilla motionless until the lower bulk of compound cools and hardens After removal of both segments from the mouth, the lower mass of hard compound is trimmed to the desired contour and a through-and-through hole is established anteriorly, into which a catheter or straw can be inserted for feeding the patient (see Fig 2).

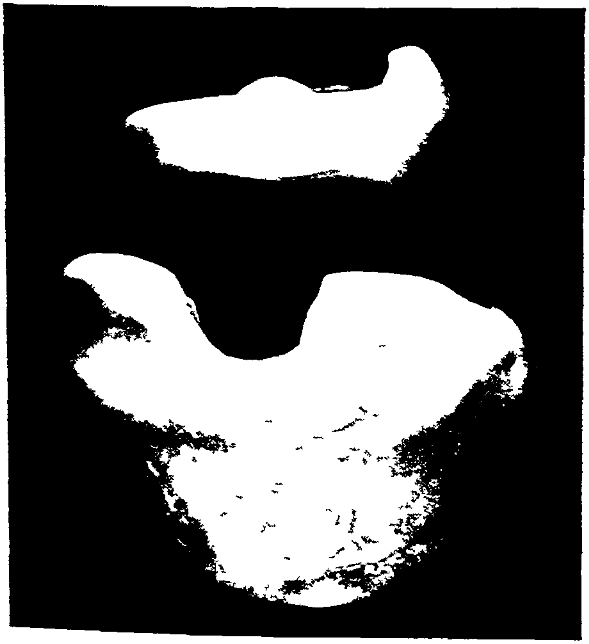

Fig 2 Mold (stent) made in two segments for immobilizing skin graft n mouth after total loss of mandible (Skin graft is used to line pocket urgically prepared behind lower lip and chin for reception of prosthetic device)

A thin split-thickness skin graft is wrapped around and stuck to the outer side of the lower compound segment with mastisol (a solution of mastic in benzene) The two segments of the mold are now introduced into the mouth of the patient The lower segment is immobilized against the upper segment and against the lower lip and cheeks by means of adhesive tape passed under the chin and up over the head

The two-piece stent is left undisturbed in the mouth for seven days, during which time the skin graft heals to the walls of the artificially created pocket Subsequently the mold is reproduced in an acrylic material, thus being made suitable for wearing until a prosthetic appliance can be prepared

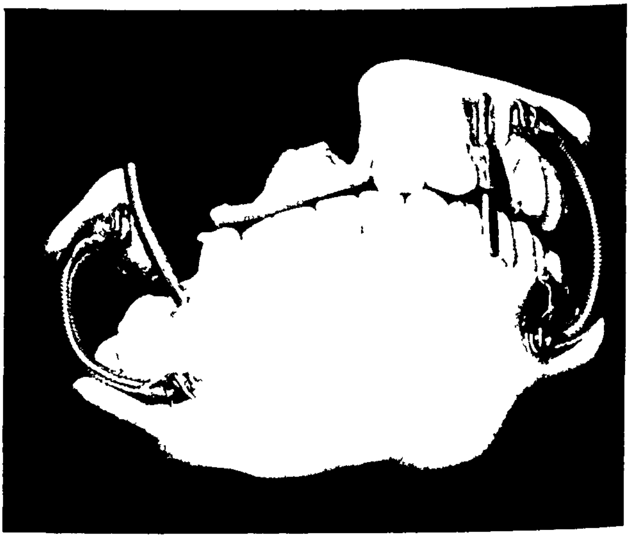

Fig 3 Intra-oral prosthetic appliance used in total loss of mandible when clavicular tube flap of skin is not used (see text)

In most cases of this kind I have had a dentist construct this prosthetic appliance as illustrated in Fig 3 Because there are no means of retaining the appliance in proper position, it would roll around in the mouth like a ball in a socket To give some stability to the apparatus in the mouth, metal springs from the prosthesis to a partial or full upper denture are necessary, these springs push the device down when the patient opens his mouth In addition, a vertical bar on each side of the prosthetic device maintains the lower appliance in correct alignment with the upper denture, these bars slide through metal rings in the upper denture

Often after such a prosthetic device has been introduced into the mouth, the lower lip is stretched and tense, sometimes to such a degree that the patient is unable to bring the lips into apposition If such is the case, elastic bands stretched between adhesive tape over the chin and lower lip below and

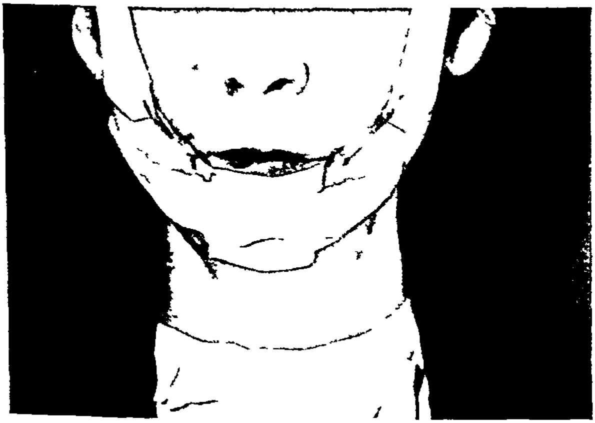

Fig 4 Elastic traction on adhesive tape for stretching lower lip and chin up over intra-oral prosthesis (see text)

adhesive tape over the head above will elevate the lower lip until it comes in contact with the upper lip (see Fig 4). Although this form of elastic traction to the lower lip need be used for only a few days, several months may be required for the muscles of the lower lip to develop sufficiently to impart to the lower lip a completely normal contour

Even though this type of prosthetic appliance is beneficial in its restoration of normal facial contour, it is unsatisfactory from a functional standpoint Usually patients find it necessary to remove such a device at mealtime because they are unable to manipulate it properly during the swallowing of liquid food

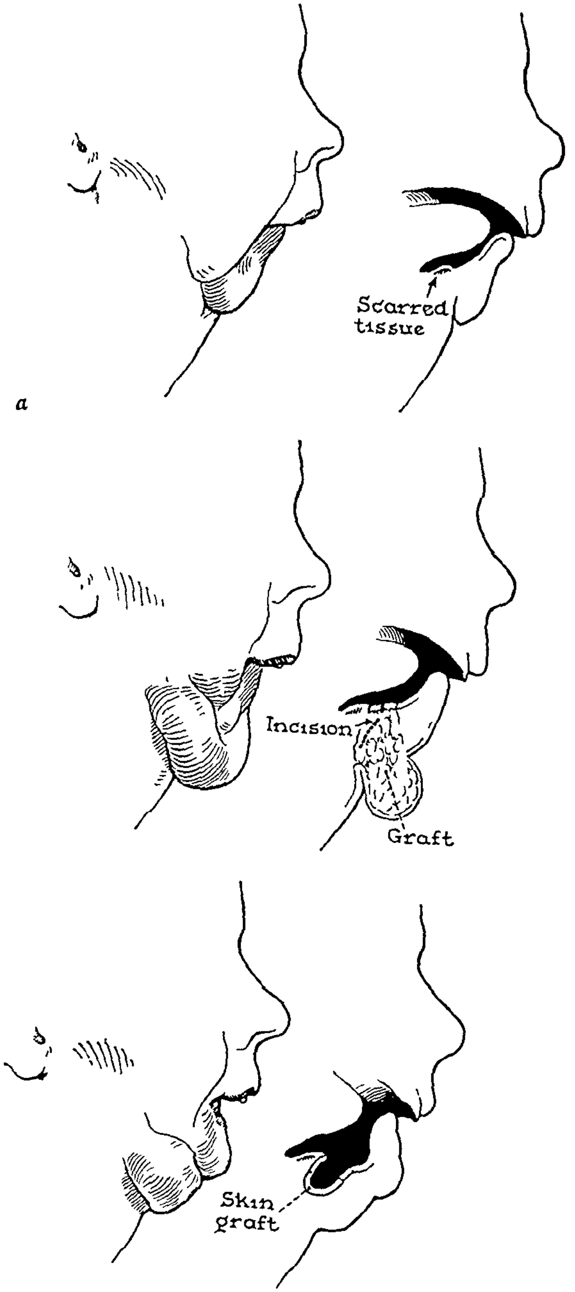

Fig 5 Use of clavicular tube flap in loss of mandible a—Retraction of lower lip and chin b—Clavicular tube flap has been wedged in between the lower lip and chin and the floor of mouth All fat in tube is left intact Dashed line indicates where intra-oral incision is made down into tube flap c—When edges of incision are separated resulting large pocket must be lined with split-thickness skin graft Pocket receives extension added to prosthesis to aid retention of appliance

In recent years I have learned that the prosthetic device is retained in the mouth much more effectively if a tube flap of skin elevated along one clavicle is inlaid in a horizontal direction between the chin and floor of the mouth (see Fig 5) The introduction of this mass of soft tissue lessens or eliminates the backward pull of scar tissue on the lower lip, otherwise, the lip often is so tight over an intra-oral prosthetic appliance that it tends to force the apparatus out of the mouth Moreover, the prosthesis can be extended into the inlaid tube flap, which provides considerable stabilization for the appliance (see Fig 5-c) As a matter of fact, in an oral receptacle constructed thus, the prosthetic device requires no attachments to the upper denture for retention and can be used to masticate soft foods

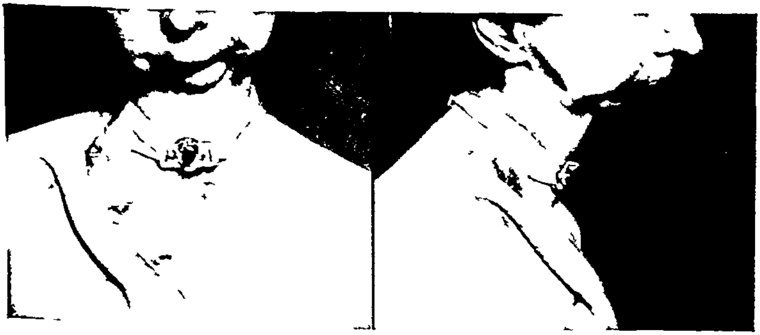

Fig 6 (Loss of mandible due to radiation necrosis) Front and side views of clavicular tube flap of skin elevated for use in providing soft tissue for scarred floor of mouth

In preparation of the tubed skin graft, a double pedicle flap of skin is elevated and tubed in an oblique direction across one clavicle (see Fig 6) After one month the supraclavicular end of the tube is detached and transferred to one or the other of the submaxillary regions Three weeks later the infraclavicular end of the tube is cut off and transplanted to the opposite submaxillary region (see Fig 7) After another three weeks, a through-and-through incision is made in the submental region into the floor of the mouth, thus completely freeing the chin and lower lip from the floor of the mouth

The tube flap then can be wedged into this incision to force the lower lip and chin forward

Horizontal incisions along the anterior and posterior surfaces of the tube divide it into an upper intra-oral portion and a lower external part The cut edges of the skin on the upper half of the tube are sutured to the oral mucous membrane, and the cut edges on the lower part of the tube are stitched to the adjacent skin margins By this arrangement the skin on the upper surface of the tube flap forms the epithelial

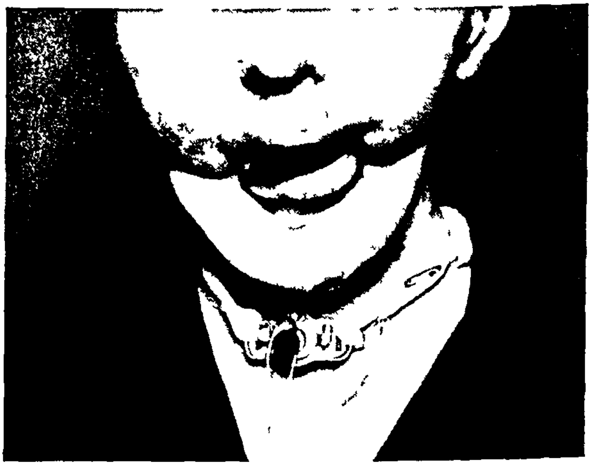

Fig 7 (Same patient as in Fig 6) Clavicular tube flap of skin attached to either side of chin

lining of the mouth between the lower lip and the floor of the mouth, and the skin on the undersurface of the tube becomes a part of the external skin covering the submental region No fat in the tube is disturbed or removed, so a bulk of soft tissue remains behind the chin to hold it forward and prevent subsequent contractures

After the tube flap is completely healed in its new position, an incision is made through the intra-oral part of the tube flap (see Fig 5-b) This incision is carried down into the underlying fat of the tube and backward under the tissues of the floor of the mouth When the incision is spread apart, a rather large pocket is produced for the reception of an ex-

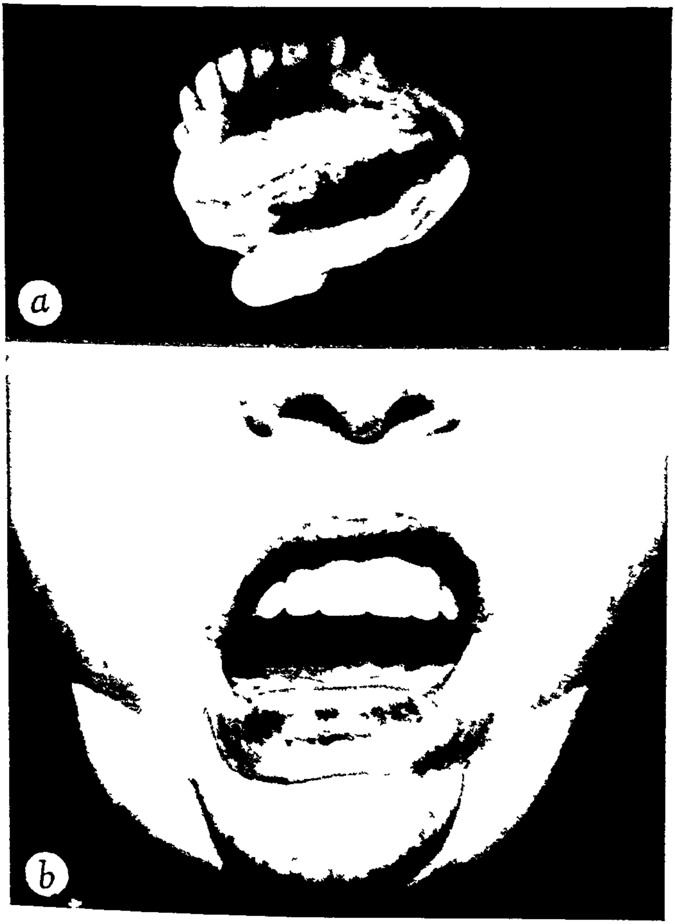

Fig 8 (Same patient as in Fig 6) a—Prosthesis employed to hold lower lip and chin forward (Downward extension on this prosthesis fits into skin-lined pocket illustrated in Fig 5-c) b—Prosthetic appliance shown in Fig 8-a fits almost as well as any full lower denture without the use of springs By using the muscles of expression about the mouth the patient can open her mouth and can masticate very soft food

tension on the proposed prosthetic device To prevent obliteration of the pocket by scar tissue, it is essential that the cavity be lined with a split-thickness skin graft (see Fig 5-c)

An acrylic prosthetic device with lower artificial teeth to occlude with the patient's upper teeth or denture is prepared by a dentist, this appliance is made to fit over the tissues in the floor of the mouth and has an extension down into the skin-lined pocket in the inlaid tube flap (see Fig 8-a)

In the three cases in which I have used this technique, I have found that the prosthetic appliance can be seated securely and that it fits almost as well as does the ordinary full lower denture (see Fig 8-b) The tube flap of skin eliminates extensive scarring behind the lower lip and chin and leaves the lower lip and chin nicely contoured (see Fig 9) and freely movable with no tendency to displace the dental appliance The patient can learn to masticate soft food by use of the muscles of expression about the mouth, and because the appliance is self-retaining, the patient can also learn to talk satisfactorily

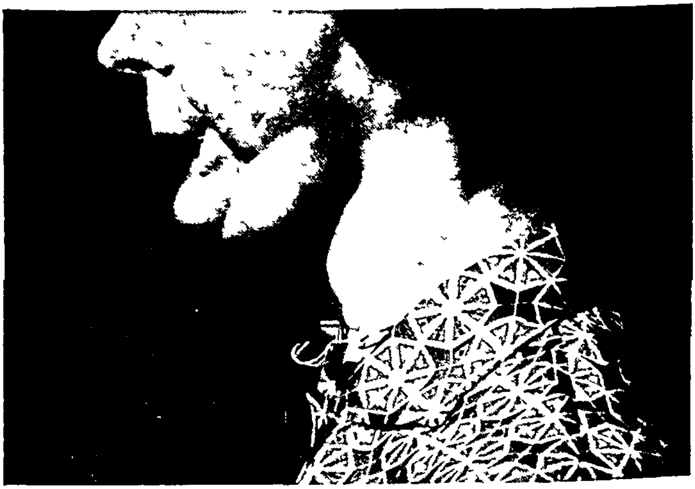

Fig 9 (Same patient as in Fig 6) Postoperative result

PROGRAM FOR AMERICAN MEDICAL ASSOCIATION SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY, 109th ANNUAL MEETING

June 15-17, 1960, Miami Beach, Fla

Wednesday, June 15, 1960

Joint Session of the Section on Laryngology, Otology and Rhinology, Section on the Diseases of the Chest, and Section on Radiology

1 Panel Discussion on Tumors of the Trachea and Bronchi

Moderator Dr Paul H Holinger, Chicago 11, Ill

Endoscopist Dr Herbert W Schmidt, Rochester, Minn

Radiologists Dr Barton R Young, Philadelphia, Pa,
Dr Wendell G Scott, St Louis, Mo

Surgeons Dr Robert J Jensik, Chicago 4, Ill,
Dr Robert W Jamplis, Palo Alto, Calif

2 Panel on Disseminated Diseases of the Chest

Moderator Dr Harold O Peterson, Minneapolis, Minn

Internist Dr James A Wier, Denver, Colo

Chest Physician Dr David Radner, Chicago, Ill

Endoscopist Dr Walter H Maloney, Cleveland 6, O

Radiologists Dr Eugene F Van Epps, Iowa City, Ia,
Dr Clyde A Stevenson, Spokane, Wash

Surgeons Dr Donald L Paulson, Dallas, Tex,
Dr William Tuttle, Detroit, Mich

Thursday, June 16, 1960

Audiology Its Progress and Problems

Kenneth O Johnson, Ph D, Executive Secretary, American Speech and Hearing Association, Washington 6, D C, (By Invitation)

External Otitis Specific Therapy Based on Accurate Diagnosis

Edley H Jones, Vicksburg, Miss

Address of Guest of Honor

Louis H Cleif, St Petersburg 2, Fla

Nasal Tip Surgery

Sam H Sanders, Memphis 4, Tenn.

Address of Section Chairman

Paul H Holinger, Chicago 11, Ill

Problems and Complications in Head and Neck Surgery

George A Sisson, Syracuse 10, N Y , and Norman E Johnson

The Long-Term Care of the Laryngectomized Patient

George F Reed, Massachusetts Eye and Ear Infirmary, Boston, Mass

Friday, June 17, 1960

Cystadenoma of the Parotid and of the Larynx

Arthur J Kuhn, Hammond, Ind

Effect of Premarin on the Ground Substance

Maurice Schiff, Capt (MC) USN, U S Naval Hospital, Oak Knoll, Calif

Cervico-Facial Congenital Anomalies

Richard T Farrior, Farrior Clinic, Tampa 6, Fla

Incidence of Hearing Impairment in a Medical Center Population

Edmund P Fowler, Jr , M D , and Thomas H Fay, Ph D , New York 32, N Y

Otometric Operations

George D Albers, Grand Rapids 3, Mich

Five Year Results of Stapes Mobilization and Current Results with Vein Plug Stapedeoplasty

C M Kos, University Hospitals, Iowa City, Ia

Fenestration of the Oval Window

John J Shea, Jr, Memphis 4, Tenn

Use of Ear Canal Skin in Myringoplasty

William F House, Los Angeles 57, Calif

Problems with Tympanoplasty

Edward C Brandow, Jr, Albany 10, N Y

LADY EWING MEMORIAL

"The death of Lady Ewing of Manchester, England, in July, 1959, ended a life of devoted service to the cause of the education of deaf children The world-wide reputation of the Department of Education of the Deaf at Manchester University came about mainly through her own genius as a teacher and her skill in imparting her knowledge to others From its opening in 1919 until her retirement in 1949, she inspired successive groups of students with her own enthusiasm and conveyed to them something of her steadfast adherence to the principles which she showed were sound

"It was perhaps fitting that, as she had started, so her life's work ended From 1912 to 1919 she demonstrated what could be done with young deaf children at the Royal Residential Schools for the Deaf in Manchester, and her closing years were spent in proclaiming the need for the earliest possible training of young deaf children and in giving help to these who were willing to undertake this work

"Her work won world-wide renown and her advice and help were eagerly sought not only throughout the United Kingdom, but in the Commonwealth and elsewhere as well Recognition of her work came with honorary degrees and other distinctions Her friends and former students now feel that this recognition should take a more permanent form and that some suitable memorial to her life and work should be established They believe that many people, especially those whom she helped directly and indirectly, would be glad to contribute to the cost of its foundation It has accordingly been decided to establish a Research Scholarship to be known as the Irene Ewing Research Scholarship

"It is believed that such a Research Scholarship would most fittingly preserve the memory of one who, in our generation, probably did more in her lifetime than any other single person to advance the cause of the education of deaf children"

Upon receipt of the above information from England, a group of Lady Ewing's friends in America felt that they would like to share in the establishment of this memorial We would like to offer others in the United States and Canada the opportunity of honoring one who has contributed so much to the education of deaf children throughout the world

Contributions from the United States and Canada may be mailed to Clarke School for the Deaf, Northampton, Mass Attention Miss Numbers

Checks should be made payable to Lady Ewing Memorial Fund

THE MOUNT SINAI HOSPITAL

An intensive Postgraduate Course in Rhinoplasty, Reconstructive Surgery of the Nasal Septum and Otoplasty will be given July 16, 1960, through July 29, 1960, by Dr Irving B Goldman and staff at the Mount Sinai Hospital in affiliation with Columbia University

Candidates for the course should apply to Registrar for Postgraduate Medical Instruction, The Mount Sinai Hospital, 5th Avenue and 100th Street, New York 29, N Y

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(Secretaries of the various societies are requested to keep this information up to date)

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President Dr Erling W Hansen, 90 So Ninth St, Minneapolis, Minn
Executive Secretary Dr William L Benedict, Mayo Clinic, Rochester, Minn
Meeting Palmer House, Chicago, Ill, October, 1960

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President Dr J J Longacre, 1503 Carew Tower, Cincinnati, O
President-Elect Dr D C Samuel Pruzansky, D D S, 840 So Wood St, Chicago, Ill
Secretary Treasurer Dr Spriestersbach, Ph D, Department of Otolaryngology, University Hospital, Iowa City, Ia
Meeting

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Meeting Palmer House, Chicago, Ill, October, 1960

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Secretary Dr F Johnson Putney, 1712 Locust St, Philadelphia 3, Pa
Treasurer Dr Charles M Norris, 3401 Broad St, Philadelphia, Pa
Meeting Lake Placid Club, Essex Co, N Y, May 20, 1961

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Secretary Dr Lyman G Richards, Wellesley Hills, Mass
Treasurer Dr Francis E LeJeune, New Orleans La
Editor, Historian, and Librarian Dr Edwin N Broyles, Baltimore, Md
Meeting Lake Placid Club, Essex Co, N Y May , 1961

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President Elect Dr John R Lindsay, Chicago Ill
Secretary Dr C Stewart Nash, 700 Medical Arts Bldg, Rochester 7, N Y
Treasurer Dr K M Day, 121 University Pl, Pittsburgh, Pa
Annual Meeting Lake Placid Club, Essex Co, N Y May 23 24 25 1961

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Secretary Dr Walter E Heck, San Francisco, Calif
Representative to Scientific Exhibit Dr Walter H Maloney, Cleveland, Ohio
Section Delegate Dr Gordon F Harkness, Davenport, Ia
Alternate Delegate Dr Dean M Lierle, Iowa City, Ia
Meeting Miami Beach, Fla, June 13-17, 1960

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Vice-President Dr Lawrence R Boies
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Vice President Dr Kenneth Hinderer, 402 Medical Arts Bldg, Pittsburgh, Pa
Secretary Dr Louis Joel Feit 66 Park Ave, New York 16, N Y
Treasurer Dr Arnold L Caron, 36 Pleasant St, Worcester, Mass

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President Dr Roland M Loring, 25 E Washington St, Chicago, Ill
Secretary Dr Robert M Hansen, 1735 N Wheeler Ave, Portland 17, Ore
Annual Clinical Session Chicago, Ill (Definite time and place to be announced later)
Annual Meeting October 1 1960, Belmont Hotel, Chicago, Ill

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Treasurer Dr F Johnson Putney Philadelphia, Pa
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Annual Meeting Palmer House Chicago Ill, October 1, 1960

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Treasurer Dr Joseph C Miceli Brooklyn, N Y
Secretary Dr Samuel M Bloom 123 E 83rd St, New York 28, N Y
Meeting Chicago, Ill, October 1960

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President-Elect Dr Leland H Prewitt Ottumwa Ia
Vice President Dr D A Skinner Newark O
Secretary Treasurer Dr Daniel S DeStio 121 S Highland Ave Pittsburgh 6 Pa
Annual Meeting Palmer House Chicago Ill October 8, 1960

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SOCIETE CANADIENNE D'OTOLARYNGOLOGIE

President Dr Gordon H Francis, 925 W Georgia St, Vancouver, B C
Secretary Dr Donald M MacRae, 324 Spring Garden Road, Halifax, Nova Scotia
Meeting Jasper Park Lodge, Jasper National Park, Alberta, June 10 12, 1960

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Vice-President Dr Linden Wallner, 122 So Michigan, Chicago, Ill
Secretary-Treasurer Dr Robert Lewy, 25 East Washington St, Chicago 2 Ill
Meeting First Monday of each month, October through May

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Secretary Dr M L Battles
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Secretary Treasury Prof Dr Antonio Carrascosa
Pro Secretary of the Interior Prof Dr Carlos P Mercandino
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Secretary
Meeting

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Secretary Dr Chevalier L Jackson, 3401 N Broad St, Philadelphia 40, Pa, U S A
Meeting

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President-Elect Dr Dick H Underwood
Secretary Dr James T Robison, 4620 J C Nichols Parkway, Kansas City, Mo
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Secretary Treasurer Dr Wendell C Irvine
Chairman of Ophthalmology Section Dr Carroll A McCoy
Secretary of Ophthalmology Section Dr Philip D Shanedling
Chairman of Otolaryngology Section Dr Robert W Godwin
Secretary of Otolaryngology Section Dr Francis O'N Morris
Place Los Angeles County Medical Association Bldg, 1925 Wilshire Blvd, Los Angeles, Calif
Time 6 30 P M last Monday of each month from September to June, inclusive—Otolaryngology Section 6 30, first Thursday of each month from September to June, inclusive—Ophthalmology Section

LOUISIANA MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

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Secretary Dr Edley H Jones, 1301 Washington St, Vicksburg Miss
Meeting Edgewater Gulf Hotel Edgewater Park Miss, April 8 9, 1960

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Assistant Secretary-Treasurer Dr William F Murrah Jr Exchange Bldg, Memphis, Tenn
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President Dr Paul Holinger, 700 No Michigan Blvd, Chicago, Ill
Executive Secretary Dr Chevalier L Jackson, 3401 No Broad St, Philadelphia 40, Pa, U S A
Meeting Seventh Pan American Congress of Oto Rhino Laryngology and Broncho Esophagology
Time and Place

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SIXTH INTERNATIONAL CONGRESS ON DISEASES OF THE CHEST

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Mid Winter Clinical Convention annually, the last two weeks in January at Los Angeles, Calif

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Place Army and Navy Club, Washington D C

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Secretary Treasurer Dr J F Birrell 14 Moray Place, Edinburgh
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Vice President Dr David Stack, Spartanburg S C
Secretary Treasurer Dr Roderick Macdonald Rock Hill S C
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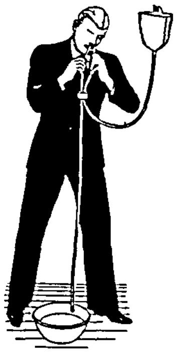

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CANCER OF THE EAR

A Report of 150 Cases *†‡

JOHN S LEWIS M D,

New York, N Y

Cancer is the end result of mutational processes, which giving rise to new races of cells, do not have the capacity to mature or to differentiate completely in response to regulatory host mechanisms [1] Cancer of the ear has the same biological characteristics as cancer found at other sites in the human body Cancer involvement of an organ of special sense, with the proximity of dura and brain, cranial nerves, temporomandibular joint and zygoma presents a formidable operation, for not only may hearing be lost and balance disturbed, but the incidence of postoperative intracranial infection complicates a hazardous surgical resection, therefore, it seems important at this time to review the diagnosis and surgical managament of the various types of cancer of the ear, as seen in 150 patients over a period of 15 years

The objectives of this report are to analyze these cases of aural cancer, with particular reference to etiology and pathology, to classify them according to therapy indicated, and to describe the surgical procedure employed for each classification

An early, excellent review of the literature was presented

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*Read at the meeting of the Eastern Section American Laryngological Rhinological and Otological Society Inc Philadelphia Pa Jan 8 1960

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‡From the Head and Neck Service Memorial Center New York

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by Peele and Hauser in 1941.[2] According to these authors, Wilde and Roudot, Schwartze, Lucae, Kidd, and others, in the period around 1775, were the first to discuss carcinoma of the middle ear. In 1883, Politzer[3] gave a comprehensive description of cancer of the ear, as manifested in his patients. Kretschmann,[4] in 1886, gave a comprehensive description of reported cases, and included four of his own. Between 1804 and 1899, 121 otitic tumors were recorded, according to Zeroni[5], and Newhart[6] found 34 cases of carcinoma of the middle ear reported between 1899 and 1917 (only eight of the 34 cases being reported in American medical journals). In 1921, Broders[7] made a statistical analysis of 63 cases of epithelioma of the ear, which was the first comprehensive article published in the English language. Yates[8] in 1936, published his findings covering a thorough search of the literature; he listed 14 cases reported between 1917 and 1924, and 24 cases between 1924 and 1936. In 1942, Figi and Hempstead[9] reported 48 malignant tumors of the middle ear and mastoid from 1922 to 1941, as well as 500 others involving the pinna or external auditory canal. Morrison,[10] in 1946, reviewed 13 cases of malignant tumors of the ear, including the external ear. Grossman, Donelly and Snitman,[11] in 1947, reported six cases of squamous cell carcinoma of the middle ear and mastoid. Garnett Passe[12] reported a case of primary carcinoma of the Eustachian tube in 1948, and reviewed two other cases in the literature. In 1950, Towson and Shofstall,[13] in an excellent review, analyzed seven cases of ear cancer, from the Jefferson Hospital, Philadelphia. Mattick and Mattick,[14] in 1951, discussed their experience with ten cases of cancer of the middle ear and mastoid. In 1954, Figi and Weisman[15] describing a wide experience with the surgical management of ear cancer and chemodectoma at the Mayo Clinic, cited 124 cases of cancer of the middle ear and mastoid covering the period from January, 1907, through December, 1951.

In 1954 Parsons and Lewis,[16,17] encouraged by the partial success of Ward, et al.,[18] and Campbell, et al.,[19] in resecting the temporal bone piecemeal for carcinoma, demonstrated the feasibility of a combined intracranial, extracranial approach in a one stage *en bloc* subtotal resection of the temporal bone for carcinoma involving the middle ear and mastoid. In 1958 these same authors reported successful temporal bone resections in 27 cases with an expected fair salvage rate.

Meanwhile, in 1955 Miller[20] analyzed 88 cases of carcinoma of the external auditory meatus. He emphasized the unpredictability of these lesions as to local extent and recurrence regardless of histologic type, and demonstrated the lymphatic drainage of the area.

DEFINITION

Cancer of the ear is a term covering a multiplicity of lesions, ranging from cancer of the skin of the auricle to malignant tumor which erodes the petrous portion of the temporal bone. The problems of the various lesions differ considerably, except in cases of advanced lesions (where the temporal bone is invaded); for these, radical measures are mandatory, whatever the site of origin may be. Cancer of the pinna and cancer of the auditory canal are designated as "early" (below 2.5 cm.) and "advanced" respectively, because of the difference in therapy required. Cancers of the middle ear, of the mastoid, and of the petrosa, at any stage, require radical surgery and are grouped in a common therapeutic approach.

INCIDENCE

According to published reports, cancer of the ear is a rare condition, and even a busy otolaryngologist may see, at most, only one or two ear cancers in his entire career. Figi and Weisman,[15] in analyzing the records of the Mayo Clinic for 45 years (January, 1907, through December, 1951), found only 124 cases of cancer of the middle ear and mastoid. By way of contrast, more than 13,000 patients were treated for cancer of the stomach at that clinic in the same period. Among 212,000 cases of aural disease seen between 1905 and 1924, at the Manhattan Eye, Ear and Throat Hospital, Robinson[21] found a diagnosis of tumor in only 48 cases, or a ratio of 1:4000. Schall[22] found that in a 12-year period, at the Massachusetts Eye and Ear Infirmary, only 15 patients out of 90,040 with pathological conditions of the ear, had neoby Peele and Hauser in 1941[2] According to these authors, Wilde and Roudot, Schwartze, Lucae, Kidd, and others, in the period around 1775, were the first to discuss carcinoma of the middle ear In 1883, Politzer[3] gave a comprehensive description of cancer of the ear, as manifested in his patients Kretschmann,[4] in 1886, gave a comprehensive description of reported cases, and included four of his own Between 1804 and 1899, 121 otitic tumors were recorded, according to Zeroni[5], and Newhart[6] found 34 cases of carcinoma of the middle ear reported between 1899 and 1917 (only eight of the 34 cases being reported in American medical journals) In 1921, Broders[7] made a statistical analysis of 63 cases of epithelioma of the ear, which was the first comprehensive article published in the English language Yates[8] in 1936, published his findings covering a thorough search of the literature, he listed 14 cases reported between 1917 and 1924, and 24 cases between 1924 and 1936 In 1942, Figi and Hempstead[9] reported 48 malignant tumors of the middle ear and mastoid from 1922 to 1941, as well as 500 others involving the pinna or external auditory canal Morrison,[10] in 1946, reviewed 13 cases of malignant tumors of the ear, including the external ear Grossman, Donelly and Snitman,[11] in 1947, reported six cases of squamous cell carcinoma of the middle ear and mastoid Garnett Passe[12] reported a case of primary carcinoma of the Eustachian tube in 1948, and reviewed two other cases in the literature In 1950, Towson and Shofstall,[13] in an excellent review, analyzed seven cases of ear cancer, from the Jefferson Hospital, Philadelphia Mattick and Mattick,[14] in 1951, discussed their experience with ten cases of cancer of the middle ear and mastoid In 1954, Figi and Weisman[15] describing a wide experience with the surgical management of ear cancer and chemodectoma at the Mayo Clinic, cited 124 cases of cancer of the middle ear and mastoid covering the period from January, 1907, through December, 1951

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Lodge[25] and his associates consider that cancers of the middle ear and of the ear canal are comparable to Marjolin's ulcer, with its chronic sanious discharge producing cellular irritation that develops into cancer Chronic otitis externa and chronic otitis media have also been forerunners of cancer in some of our patients, although in most cases there was no history of pre-existing infection Coachman[29] has reported a patient with squamous cell carcinoma of the middle ear secondary to cholesteatoma, and Sparks[30] had one with carcinoma, who had long-standing otitis externa Barnes[31] believes that a large number of cancers probably originate in the lining of mastoid cavities, such lining having become altered by prolonged suppuration Hyperkeratosis, too, has frequently appeared to be a beginning of cancer of the ear

Towson and Shofstall[13] and Fredricks[32] consider trauma and chronic irritation as etiological factors They feel that chronic eczema, dermatoses, frostbite, keratosis, sebaceous cysts, papilloma, cutaneous horns, psoriasis, lupus vulgaris, xeroderma pigmentosum, burns, razor cuts may all contribute to the development of cancer Trauma caused by the pressure of eyeglasses is included among these factors

Ackerman and del Regato[33] reported that out of 32 of their patients with ear cancer, only three were women, however, in our patients, as will be demonstrated, the sex incidence varied with the anatomical site of the primary lesion

Driver and Cole[26] found 64 3 years to be the median age of patients with cancer of the ear, and Broders[7] found it to be 61 years In our patients, the median age for all was 63 years, 72 years was the median age for those with cancer of the auricle, and 55 years for those with cancer of the auditory canal, of the middle ear, and of the mastoid

PATHOLOGY

In 1956, Schenck[34] presented an excellent classification of the various pathological types of malignant ear tumors Acplasms Furstenberg[23] found cancer of the middle ear in only two out of 40,000 patients in the Department of Otolaryngology at the University of Michigan Medical School Tod,[24] quoting from the records of the London Hospital, where 200,000 cases are seen annually, found only one case Lodge and his co-workers[25] found six cases of cancer of the temporal bone reported in a population of one million people, and estimated that 0 006 per thousand of living persons suffer from aural cancer at a given time Mattick and Mattick[14] reported a diagnosis of intrinsic cancer of the ear in ten out of 35,000 tumor cases at the Roswell Park Memorial Institute in Buffalo Towson and Shofstall[13] point out that the proportion of cancer of the middle ear and mastoid to all otologic pathological conditions is 1 5000 to 1 20,000

In their report of a 25-year study, Driver and Cole[26] note that the ear was involved in 5 5 per cent of all the patients with skin cancer Labarde and Via[27] state that 8 per cent of all skin cancer originates from the auricle Broders[7] had estimated some years earlier that 84 per cent of aural cancer involved the auricle, 12 per cent the external auditory meatus, and 1 5 per cent the middle ear and mastoid In the group of 150 patients discussed here, the origin of the cancer was the auricle in 60 per cent, the external auditory canal in 28 per cent, and the middle ear and mastoid in 12 per cent

Conway and Howell[28] note the high incidence of auricular cancer in blond persons, and do not report a single case of it in a series of 100 Negro patients Not a single case of cancer of the auricle in a Negro occurred among the author's patients, although two Negro patients had cancer of the middle ear

ETIOLOGY

Sex, age, heredity, and chronic infection are the main factors that influence the development of cancer of the ear Cancer of the auricle primarily affects the aged, most often the male, and is most likely stimulated by environmental factors Actinic rays of the sun, frostbite, insect bites and ceruminous glands. Two others fungated into the middle ear from a parotid primary.

II. Mesenchymal Tumors

Sarcoma

Cases of embryonal rhabdomyosarcoma of the temporal bone have been reported by Blanchard and House,[43] Karatay,[44] and Holman.[45] According to Stobbe and Dargeon[46] the median age of patients with this tumor is 6.1 years. The tumor is always locally invasive, metastasizes distantly by blood and lymph routes but does not involve the regional lymph nodes. Only one of the patients in the present author's series, a five-year-old boy, was found to have this condition.

Soft parts sarcomas are not usually expected in this anatomical site. One of the rare cases in this series was a spindle cell sarcoma arising in the middle ear of a 43-year-old white female.

III. Malignant Tumors of Questionable Origin

Malignant Melanoma

A comprehensive report of 36 patients with melanoma of the external ear, treated between 1928 and 1934, is presented by Sylvan and Hamberger[47] from the Radium Hemmett in Stockholm. The median age on admission from this group of patients (consisting of two children ten and 12 years old respectively, and 34 adults) was 55 years. Of the 36 patients, 21 were male and 15 female. Two additional cases of melanoma of the ear are reported in the literature: one by Friedmann and Radcliffe,[48] and the other by Cordes and Masing.[49]

Our series includes two cases of melanoma arising from the posterior surface of the auricle, and measuring less than 2 cm. One of these patients had proven positive neck nodes on admission. Another arose in the auditory canal and had neck node metastases. A nine-month-old infant had a melanoma of the middle ear. This was undoubtedly due to a transplacental implant from the mother, who subsequently died of widespread melanomatosis.

cording to this classification, the present author has grouped the various types of tumors found in his patients, as listed below

I Epithelial Tumors

A Malignant Surface Epithelial Tumors

1 Epithelioma

a Squamous Cell Carcinoma

Broders,[7] Towson and Shofstall,[13] Driver and Cole,[26] Conway and Howell,[28] Brunner,[35] Eller and Eller,[36] all consider squamous carcinoma the most common type of cancer found in the ear. Although in our own patients only one-third of the lesions arising on the auricle were squamous cell, 75 per cent of the advanced cancers of the auditory canal and middle ear were of this histologic type. Eleven per cent of these tumors had metastasized to the neck nodes by the time of the patient's admission.

b Basal Cell Carcinoma

In conformity with the type of cancer most often found elsewhere on the skin surface of the body, two-thirds of the cancers in this author's patients, arising on the auricle, were basal cell carcinomas. About 50 per cent of the early, and 11 per cent of the advanced auditory canal lesions were of this type. In five of the patients, the lesions fungated from the auricle, and invaded the middle ear, mastoid, and squamous portion of the temporal bone.

B Malignant Glandular Epithelial Tumors

1 Adenocarcinoma

This tumor may arise from sebaceous glands or ceruminous glands of the ear canal. Schall,[22] Furstenberg,[23] Lukens,[3] Grabschied,[18] Fraser,[39] Thorell,[40] and Fabre,[41] all reported cases of this type of cancer. In 1941, Warren and Gates[4] described the first case of carcinoma of the ceruminous glands. In four of the author's patients the lesion developed in the auditory canal, and two were diagnosed as originating in the arcinoma We also have one case of malignant melanoma rising on the posterior surface of the auricle

Symptoms The majority of our patients were asympto-atic, however, in most cases, the lesions when present, were bvious to the patient or were found by the examining physi-ian A few of the lesions became ulcerated and occasionally ot infected with subsequent bleeding and pain

Treatment Local medical treatment in the form of salves nd caustics had been applied, before admission, to most of the lesions for periods ranging up to five years Five patients had been given X-ray treatment, two had been subjected to electrocauterization, two had been surgically treated and had had a recurrence, and one case had had both surgery and radium treatment

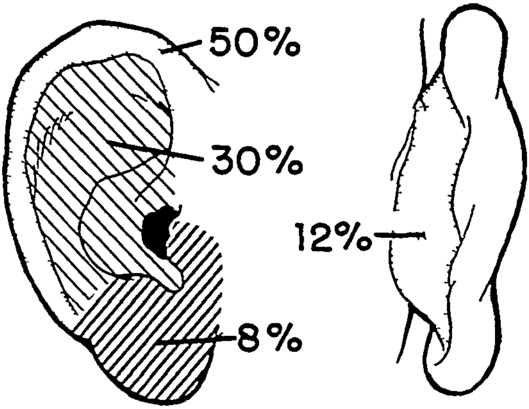


Fig 1 Distribution of lesions arising from the auricle

Some authors advocate radiation for patients with ear cancer[26 33], however, radiation chondritis and necrosis of underlying bone may be serious complications Chemosurgery, preferred by Mohs,[50] is not the treatment of choice The procedure that has proven uniformly successful is wide excision, without regard to cosmetic effect The good results, as reported by Fredricks[32] and Conway and Howell,[28] give

CLASSIFICATION BY THERAPEUTIC MANAGEMENT

Early Cancer of the Auricle (70 Cases)

These skin lesions, by definition, are cancers that are confined to the pinna, that have not spread to adjacent structures and measure under 2.5 cm. Seventy of our adult patients were afflicted by 73 tumors of this type. The right ear was involved more frequently than the left, and in three cases, both ears were affected coincidentally (see Table I).

TABLE I

Early Cancer of the Auricle (Under 2.5 cm.)

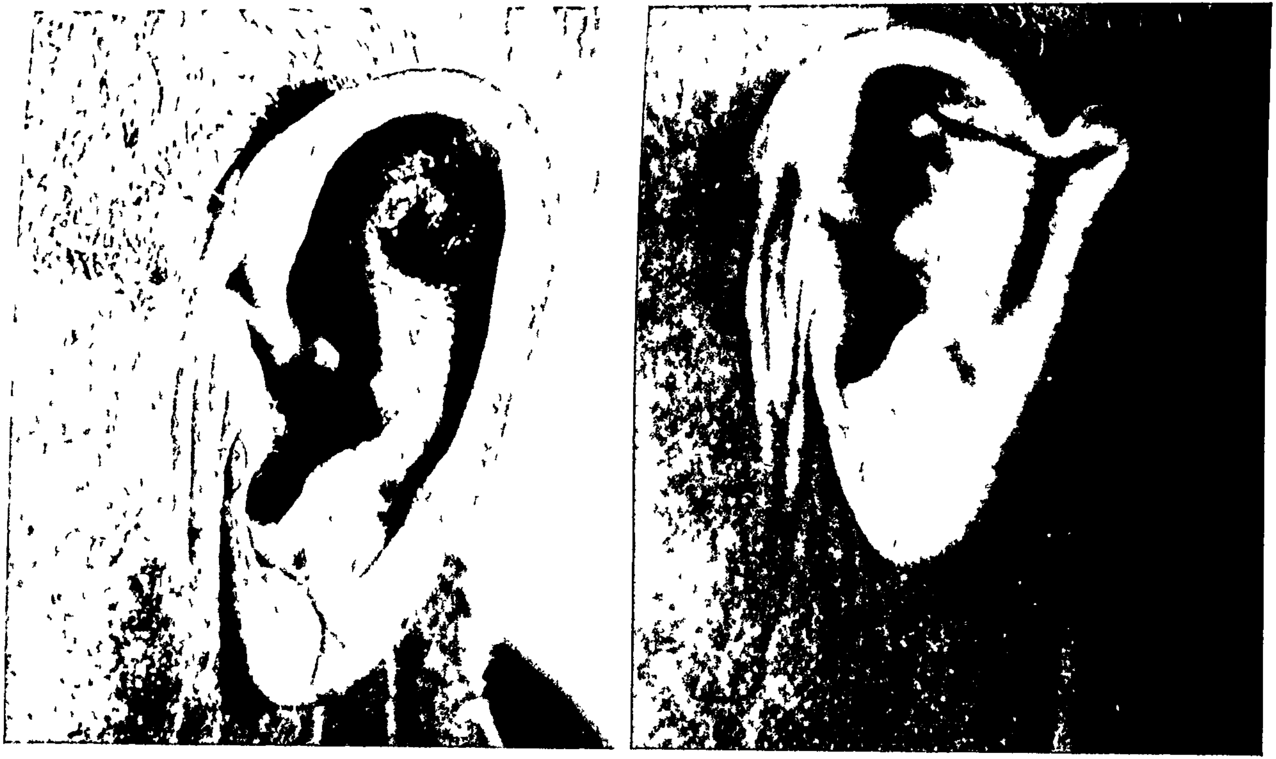
Pathological Type	No.	Median Age	Male	Female	Surgery	Radiation	Untreate
Basal Cell	46	73	0	4	46	0	0
Squamous Cell	23	71	0	1	23	0	0
Melanoma	1	55	1	0	1	0	0
Total	70	70	1	5	70	0	0

Age and Sex. Cancer of the skin seems to affect older people predominantly. In our group of 70 patients with this disease, the median age was 70 years. The youngest patient was 45 years old, and the oldest was 89.

This condition rarely affects females. Of all our patients with early cancer of this type, 92 per cent were males.

Location. We found the most common site of origin to be the helix in our patients, 50 per cent of all the lesions occurring at that location. The central region of the pinna (concha, triangular and scaphoid fossae, antihelix and its crus) was the second most frequent site, and accounted for 30 per cent of all the lesions. Twelve per cent of all the lesions arose from the posterior surface of the auricle, and the remainder was located on the concha, lobule, and pre-auricular region, in that order (see Fig. 1).

Pathology. In 67 per cent of our cases, the early lesions on the external ear were basal cell carcinomas. (These lesions are locally invasive and slow-growing, and do not, as a rule, metastasize.) In 30 per cent, the lesions were squamous cell



a Preoperative b Postoperative

Fig 3 Full thickness excision of cancer of the central portion of the auricle

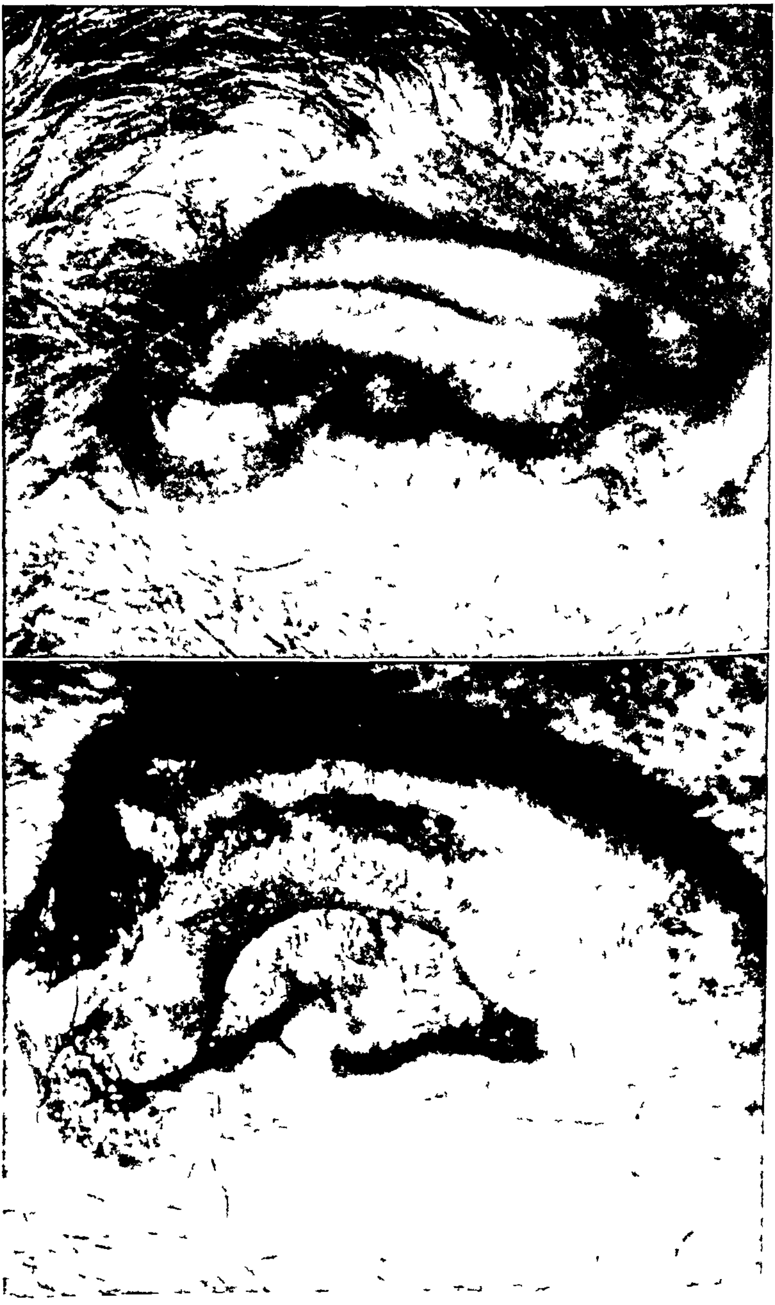


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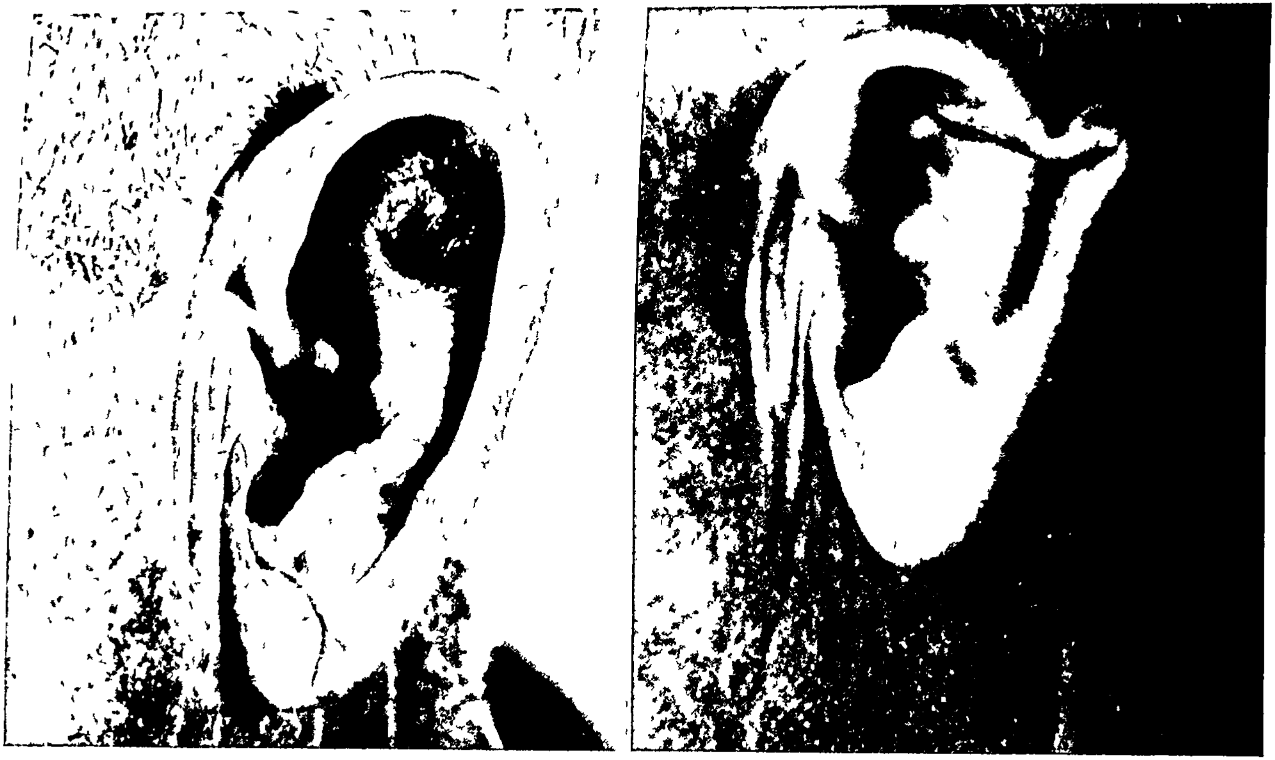
Fig. [illegible] — Excision of cancer of the helix



a Preoperative

b Postoperative

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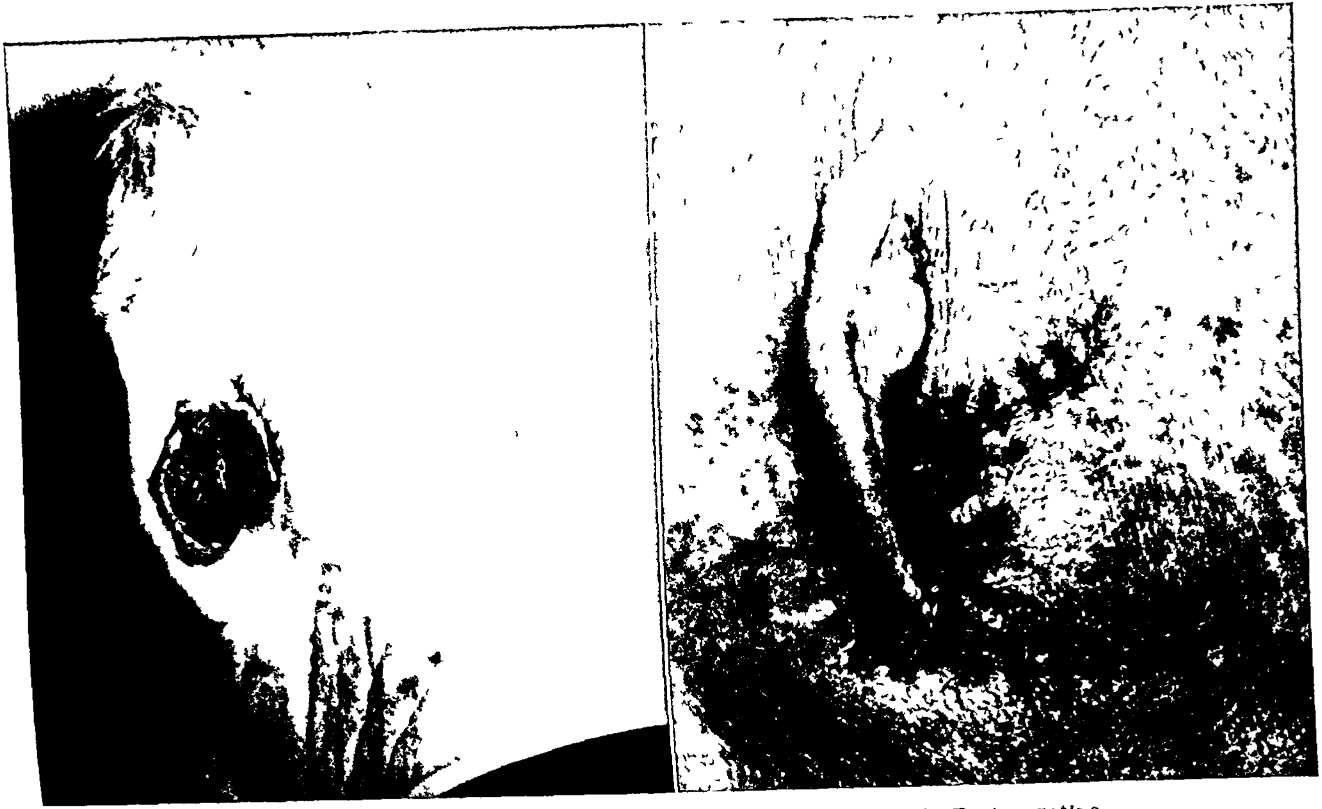

a Preoperative

b Postoperative

Fig 1 Excision of lesion of posterior surface of ear with closure by scalp flap

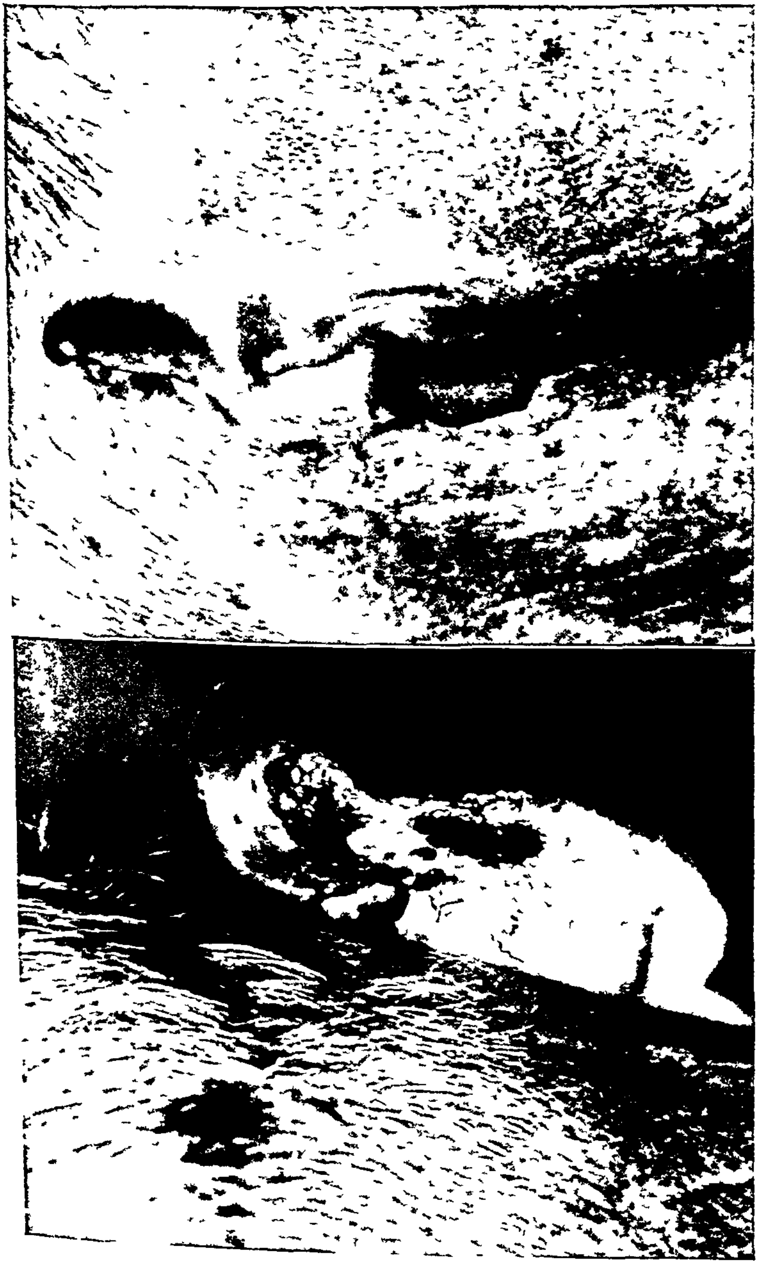

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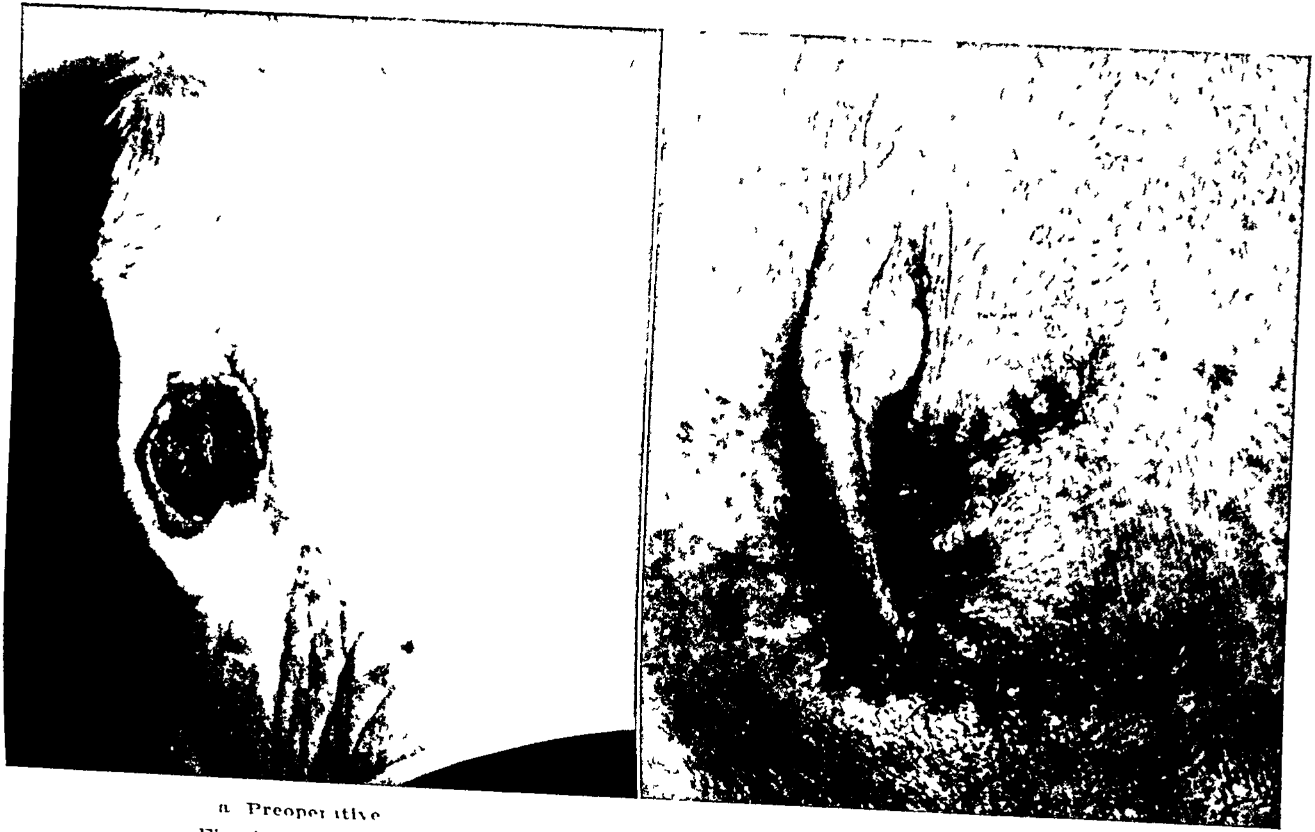
Fig 5 Excision of major portion of auricle with extensive cancer



a Preoperative b Postoperative

Fig 1 Excision of lesion of posterior surface of ear with closure by scalp flap

group of 61 patients was 70 years Because of the advanced age of these patients, 80 per cent were either lost to follow-up or died of other diseases For these reasons, this group cannot be analyzed statistically for a five-year salvage rate. Those patients who were followed appeared to be uniformly free of recurrent cancer, during the period of follow-up

Advanced Cancer of the Auricle (20 Cases)

Once the cancer extends beyond the limits of the auricle and involves the middle ear, temporal bone, parotid gland, and mandible, serious problems arise Of our entire group of 150 patients, 20 suffered from this type of advanced disease and were treated by various methods, as discussed below (see Fig 6 and Table II)

TABLE II

Advanced Cancer of the Auricle

Pathological Type	No	Median Age	Male	Female	Surgery	Radiation	Untreat
Basal Cell	12	70	8	4	7	3	2
Squamous Cell	7	73	6	1	5	0	2
Melanoma	1	55	1	0	1	0	0
Total	20	70	15	5	13	3	4

Age and Sex The median age for this group was 72 years, with about the same distribution as in the group with early lesions

Fifteen of the 20 patients were male and five were female

Pathology Seven out of this group of patients had squamous cell carcinoma, of these, two had neck nodes requiring a homolateral neck dissection Twelve others had basal cell carcinoma, one of which metastasized to neck nodes One case was a melanoma with cervical metastasis

Symptoms All the patients gave a history of a sore developing on the auricle and progressing for a period of one year or more, the median period was 40 months Eight patients had had previous limited surgery or X-ray treatment, support to the excellent prognosis in patients treated by this method

Sixty-seven per cent of our patients were treated by wedge excision and primary closure Both skin and cartilage were removed, giving very satisfactory cosmetic results (see Fig 2) Twenty per cent were treated by wide local excision and full-thickness skin graft, in these cases, the lesions did not lend themselves readily to primary closure, as those of the concha (see Fig 3) In three cases, the posterior lesions were excised and a temporal rotated scalp flap was made to cover the defect (see Fig 4) In three cases, a wedge excision with homolateral neck dissection was performed, as a primary method of treatment The larger lesions may at times require sacrifice of a major portion of the auricle, as evidenced in one case (see Fig 5)

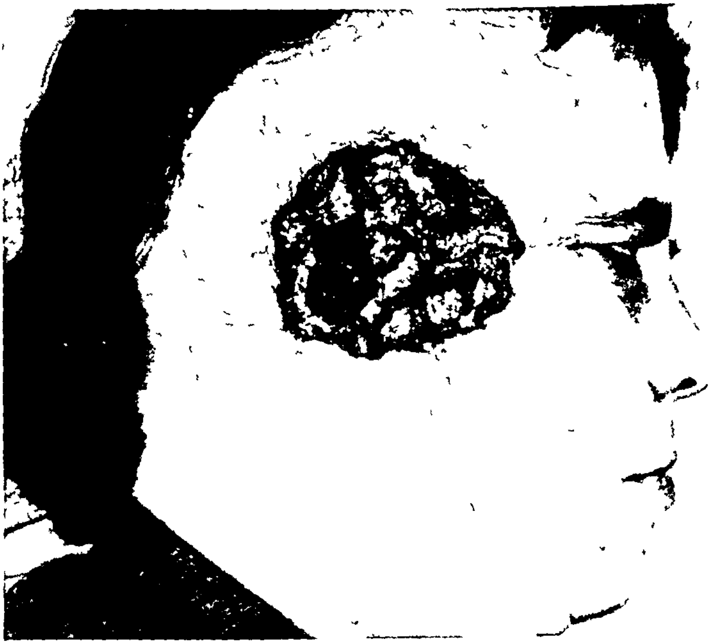


Fig 6 Advanced cancer involving the entire pinna and adjacent areas

Results As indicated previously, the median age for this membranous portion of the auditory canal, and 35 had advanced lesions invading the middle ear, mastoid, and contiguous structures

Age and Sex The youngest of these patients was 24 years old, the oldest was 75, and the median age for the entire group was 55 years

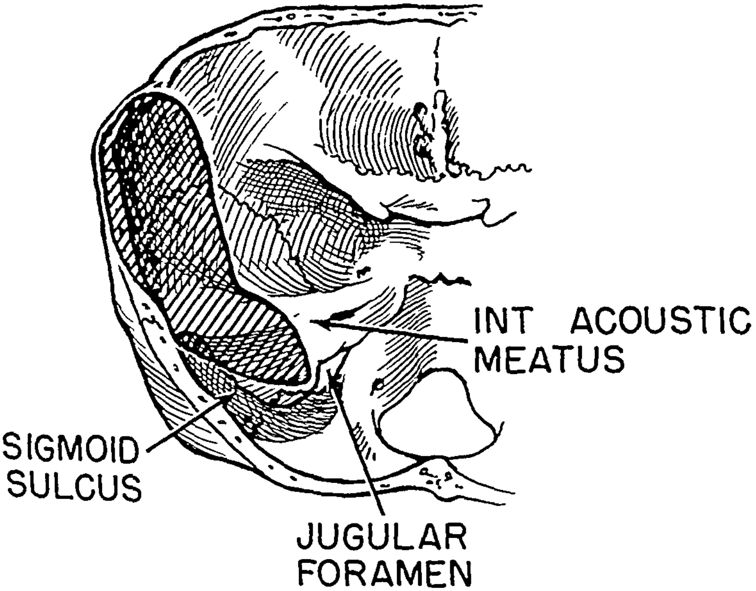


Fig 7 Extent of bone removed in subtotal resection of temporal bone

Twenty-four patients were female, and 18 were male This proportion is the reverse of that found in patients with cancer of the auricle, as previously indicated

Pathology In 69 per cent of our patients, the cancer originated in the ear canal and was diagnosed as squamous cell epithelioma Of all the early cancers among our patients, only 27 per cent were of this type, and of the advanced cancers, 77 per cent were squamous cell carcinomas In three of the latter group, proven positive neck nodes were present at the time of admission Only 20 per cent of all our patients had basal cell carcinomas The basal cell carcinomas com-

or both Pain did not appear to be a factor until bone erosion occurred, leading to secondary infection

Treatment Of the 20 patients with advanced cancer, three refused treatment, one was 90 years old (who died before any treatment was administered), three were given palliative X-ray therapy because of feebleness and advanced age, and the remaining 13 were treated by radical surgery

Excision of the auricle, mastoidectomy, resection of the ear canal, and parotidectomy with skin graft were done in eight of the 13 surgical cases Four of these eight patients also had dissection of the homolateral neck, as part of the operation, and a third had radon seeds implanted into the residual disease, on the dural plate In the five other surgical cases, excision of the auricle and subtotal resection of the temporal bone were carried out, by the technique of Parson and Lewis[16] In two of these cases, the defect was replaced by a rotated scalp flap

Results The three patients who were given X-ray therapy died within six months to two years following treatment

Of the eight patients treated by excision of the auricle, mastoidectomy, resection of the ear canal, and parotidectomy with skin graft, six died of disease within a period of eight months The seventh patient has been free of disease for five years, and the eighth was lost to follow-up after staying free of disease for four years

Of the five patients subjected to excision of the auricle and subtotal resection of the temporal bone (see Fig 7), two have gone free of disease for five years, two (recently operated) have been free of disease for periods up to two years, and one died two months after surgery, due to a brain hemorrhage One of these seven patients developed paralysis of the vagus nerve, and required tracheotomy and a feeding tube for two months after surgery

Cancer of the External Auditory Canal (42 Cases)

In 42 of our patients, the cancer originated in the external ear canal Seven cases had early lesions confined to the endaural incision much like coring an apple, and the entire ear canal is removed The defect is grafted with a split-thickness graft, sutured to the endaural incision and maintained in place by a stent

Three of the six patients treated by this method have been free of disease for four years and two others have had no recurrence for three years (see Table III)

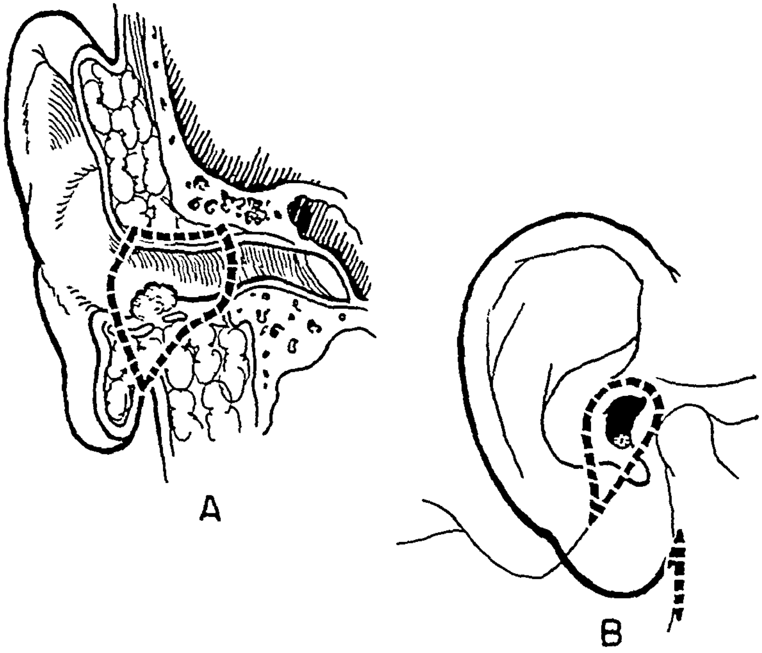


Fig 8-a Extent of excision of soft tissue for early auditory canal lesion
Fig 8-b Area of excision lateral view

Of the 35 patients with *advanced* lesions, 14 were subjected to a radical mastoidectomy, and to a parotidectomy in addition, as required The same procedure, together with a homolateral dissection of the neck, was used for two patients of the 14 in whom proven positive neck nodes were present One of these two patients had a recurrence following neck dissection and mastoidectomy, was given irradiation, and has prised 50 per cent of the early ear canal lesions, and 11 per cent of the advanced lesions Four of the 42 patients had adenocarcinoma, which, in two appeared to arise from the ceruminous glands One patient of the 42 had advanced malignant melanoma, and died of disease two years after surgery (see Table II)

Symptoms and Signs The patients with early cancer reported occasional pain in the canal, that persisted for periods up to six months Otorrhea, deafness, pain and occasional vertigo were the usual symptoms in the advanced cases, and reported in that order of frequency These symptoms were reported as lasting from four months to three-and-a-half years, and for six months on the average Five of the patients gave a history of chronic otitis of many years' duration Palsy of the facial nerves was present in four patients In most instances, an ulcerated bleeding lesion was found filling the ear canal, and often involving the middle ear

Positive X-ray findings were present in only 50 per cent of the patients, at the time of biopsy Of these, 30 per cent showed evidence of chronic mastoiditis, and in 12 per cent, a tumor and destruction of the bone were present The remaining 8 per cent showed a previous mastoid defect and chronic inflammation

Treatment Nine out of the 42 patients had been treated by irradiation, prior to admission Three others had had a mastoidectomy followed by Roentgen therapy, two had had mastoidectomy alone, and the remainder had had no treatment at all Six patients with *early* lesions involving only the membranous canal were treated by a sleeve resection of the ear canal Another was treated elsewhere The incision employed in this procedure is indicated in Figs 8-a and 8-b

The incision consists of the upper limb of a parotidectomy incision (as described by Martin[21]) which is deepened so as to expose the inferior portion of the external auditory canal Care is taken to avoid the facial nerve, and it may be necessary to expose it in a deep-seated lesion, so that it may be retracted and avoided in the subsequent surgery The remaining portion of the auditory canal is excised through an cause of their uniqueness and discussion by the author in another paper

Age and Sex The age span was nine months to 76 years, with a median age of 55 years

Nine patients were female and nine were male Both mastoid conditions involved the males

TABLE IV

Advanced Cancer of the External Auditory Canal

thological Type	No	Median Age	Male	Female	Surgery	Radiation	Untreate
sal Cell	4	47	3	1	4	0	0
uamous Cell	27	55	9	18	23	4	0
lenocarcinoma	3	54	3	0	3	0	0
elanoma	1	74	1	0	1	0	0
tal	35	55	16	19	31	4	0

TABLE V

Cancer of the Middle Ear and Mastoid

thological Type	No	Median Age	Male	Female	Surgery	Radiation	Chemotherapy
uamous Cell	12	57	6	6	9	3	0
elanoma	2	32	1	1	0	1	1
mbryonic Rhabdomyosa	1	5	1	0	1	0	0
pindle Cell	1	43	0	1	1	0	0
denocarcinoma	2	45	1	1	2	0	0
otal	18	55	9	9	13	4	1

Pathology The pathology in this group of 18 patients was distributed as follows 12 had squamous cell carcinoma (these included both patients with primary cancer in the mastoid), two patients (one infant and one elderly male) had malignant melanoma, one had spindle cell sarcoma, and one (a five-year-old child) had embryonal rhabdomyosarcoma Two patients had adenocarcinoma extending from the parotid gland.

Symptoms Of this entire group of patients, 40 per cent gave a history of otorrhea of one to 50 years' duration, in some cases dating back to early childhood The symptoms most often reported were bleeding from the ear (lasting six been free of disease for five years The remainder of the patients (13 cases) who had been operated on by this method were failures, were given X-ray treatment, and died of the disease within a period of three months to two years In this last group, there were two patients with adenocarcinoma, and one with melanoma, the others all had squamous cell carcinoma Of the entire group of 35 patients, one other had a radical mastoidectomy at another hospital, and has had no recurrence for 18 years Both of the successfully treated patients, one male and the other female, had squamous cell carcinoma

TABLE III

Early Cancer of the Auditory Canal

Pathological Type	No	Median Age	Male	Female	Surgery
Basal Cell	4	51	0	4	4
Squamous Cell	2	57	1	1	2
Adenocarcinoma	1	48	1	0	1
Total	7	55	2	5	7

Four patients, out of a group of 35, were treated by irradiation, and died within six months after therapy was instituted Thirteen patients were treated by subtotal resection of the temporal bone, have been free of disease up to the present time, and one has had no recurrence for more than five years Two out of this group of 13 patients died of complications of surgery

In three patients out of the group of 35, a temporal craniotomy was performed and because the disease was found to involve the dura, brain, and base of the skull, postoperative irradiation was also administered All three patients succumbed to disease within a year after treatment (see Table IV)

Cancer of the Middle Ear and Mastoid (18 Cases)

This group of 18 patients consisted of two with primary cancer in the mastoid, and 16 with primary cancer in the middle ear (see Table V) Glomus tumors are omitted because of their uniqueness and discussion by the author in another paper

Age and Sex The age span was nine months to 76 years, with a median age of 55 years

Nine patients were female and nine were male Both mastoid conditions involved the males

TABLE IV

Advanced Cancer of the External Auditory Canal

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sal Cell	4	47	3	1	4	0	0
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enocarcinoma	3	54	3	0	3	0	0
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denocarcinoma	2	45	1	1	2	0	0
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Pathology The pathology in this group of 18 patients was distributed as follows 12 had squamous cell carcinoma (these included both patients with primary cancer in the mastoid), two patients (one infant and one elderly male) had malignant melanoma, one had spindle cell sarcoma, and one (a five-year-old child) had embryonal rhabdomyosarcoma Two patients had adenocarcinoma extending from the parotid gland.

Symptoms Of this entire group of patients, 40 per cent gave a history of otorrhea of one to 50 years' duration, in some cases dating back to early childhood The symptoms most often reported were bleeding from the ear (lasting six weeks to one year), pain (for periods of two to 12 months), and progressive deafness Vertigo was reported by two patients, and facial palsy by three Pain and swelling over the mastoid process were reported by both patients with cancer of the mastoid

On admission, one patient, with malignant melanoma, had proven positive neck nodes, bone destruction was evident on X-ray examination in 40 per cent of this group, 50 per cent showed pathological changes suggesting chronic inflammation, one had only a mastoid defect following surgery, and in one case, the X-ray findings were negative

Treatment and Results Nine of these 17 patients had had no previous treatment, nine had had a mastoidectomy, or a mastoidectomy and Roentgen therapy

On admission, subtotal resection of the temporal bone was performed on 13 patients Of these 13, four are still alive, free of disease—one, operated on for spindle cell sarcoma, has survived for six years, one for four years, and two for three years Nine were failures Three patients developed meningitis after surgery One patient died of pneumonia associated with Xth nerve paralysis

One other of the group of 18 patients was found to be unresectable due to involvement of the brain and base of the skull, was given postoperative betatron irradiation and ended in failure

Four patients of the group of 18 were treated by irradiation only, and died of disease The infant with melanoma was treated with nitrogen mustard and also succumbed to the cancer

DISCUSSION

For practical purposes cancer of the ear has been divided into *A* Cancer of the auricle, *1* early, and *2* advanced, *B* Cancer of the external canal, *1* early, and *2* advanced, *C* Cancer of the middle ear and mastoid

Though seldom seen in office practice, the management of an ear cancer case is a dramatic episode for any otologist. Based on the wide clinical measurement of Towson and Shofstall,[13] cancers of the middle ear and mastoid are indeed rare, and occur in 1:5,000 to 1:20,000 otologic pathological conditions. Cancer of the auricle comprises about 5.5 per cent to 8 per cent of all skin cancers. The author, in assaying a group of 150 cases, has found the origin of the cancer was the auricle in 60 per cent, the external canal in 28 per cent, and the middle ear and mastoid in 12 per cent. Broders[7] reported a high proportion of cancer originating on the auricle (84 per cent) which may represent a truer indication of lesions seen at a general hospital, as against the experience at a cancer institution (where referred treated failures are more commonly seen). No case of auricular carcinoma has been reported in a Negro, although the author has cited two Negro patients with cancer of the middle ear. Sex, age, heredity and chronic infection are the main etiological factors. Cancer of the pinna affected males in 92 per cent of the early cases and 75 per cent of advanced cases. By contrast, cancer of the auditory canal was more common in females, and was distributed equally between the two sexes in the middle ear. The median age of those with cancer of the auricle was 70 years, that of the ear canal and middle ear and mastoid was 55 years. Cancer involving the Eustachian tube was invariably an extension from the nasopharynx and has been excluded from this study. Consistent with experience elsewhere on exposed skin surfaces of the body, two-thirds of the cancers of the auricle were of the basal cell type. Almost one-third were of the squamous cell variety and in two cases neck node metastases were present on admission. By contrast, three-fourths of the widely invasive lesions of the auditory canal and middle ear were of the squamous cell type and 11 per cent of these tumors had metastasized to neck nodes by the time of the patient's admission. Adenocarcinoma originated in the auditory canal, and in two of four cases of this series, appeared to arise in the ceruminous glands. Malignant melanomas were located on the posterior surface of the auricle, the auditory canal and the middle ear with a tendency to cervical metastases. Melanoma at this site appears to be most aggressive regardless of the size of the primary lesion. An additional embryonal rhabdomyosarcoma of the middle ear in a five-year-old boy is included in this study A spindle cell sarcoma arising in the middle ear of an adult white female adds to the collection of this rare histologic type

Pain is an uncommon symptom in ear cancer until infection and bony invasion takes place The diagnosis of cancer of the auricle is obvious to the examiner and confirmed always by biopsy Chronic otorrhea, hemorrhage, mastoid swelling, facial paralysis, tinnitus, deafness and vertigo are symptoms and signs of advanced ear canal middle ear and mastoid lesions Roentgen evidence of temporal bone destruction comes late and augurs poorly for resectability of an ear cancer

The importance of classification by therapeutic approach must be emphasized Each anatomical site and extent of the primary lesion is a test of the versatility and experience of the cancer surgeon Radiation and chemosurgery are fraught with complications of necrosis and infection and were not used primarily as therapeutic modalities by the author

Early lesions of the pinna (2 5 cm or less) are usually found on the helix and are best excised by means of a wedge or V-shaped type of incision Central lesions are widely excised and amenable to skin grafting of the defect Posterior lesions lend themselves readily to a rotated skin flap A margin of 1 cm should afford uniformly successful results, although follow-up in this group is difficult because of advanced age of the patients Local recurrence may necessitate sacrifice of the entire auricle Advanced cancer of the auricle requires sacrifice of the entire pinna and adjacent temporal bone including mastoid Almost half of the operated cases in this group (13 cases) are free of disease for periods up to five years The three irradiated cases were failures

Early lesions of the auditory canal were treated by a sleeve resection of the auditory canal with a replacement of the canal with a split-thickness skin graft maintained in place by a stent All of the cases (seven) are free of disease for periods up to five years Advanced cancer of the auditory canal with invasion of the middle ear and mastoid responded best to radical surgical attack Subtotal resection of the temporal bone was the surgical procedure of choice Eight of 31 operated cases are free of disease for periods up to five years, seven cases (23 per cent) are three year cures The four irradiated cases were failures

Cancer of the middle ear and mastoid requires temporal bone resection which was utilized in 13 operated cases Four of these (31 per cent) are three year cures The four irradiated cases were failures Combining advanced ear canal and middle ear and mastoid operated cases, (44 cases) the three year salvage is 25 per cent

Temporal bone resection is a unique combination of intracranial and extracranial surgical *en bloc* resection of cancer-ridden bone The initial exposure of the dura and petrous pyramid is through a temporal craniotomy The path of least resistance for cancer extending from the middle ear is through the thin roof of the tympanum into the middle cranial fossa Once dura and petrous bone are exposed, it becomes at once apparent to the operator whether the disease is limited to the temporal bone or has extended intracranially and to the base of the skull At this point a decision may be made whether subtotal resection of the petrous and mastoid is practical or whether the disease is unresectable Occasionally large segments of involved dura may be resected and replaced with temporal fascia Removal of about 40 cubic centimeters of cerebrospinal fluid will allow the dura and lateral sinus to be retracted from the petrous, so that a Stryker saw and chisels may be introduced to fracture the involved temporal bone from the base of the skull Hypotensive agents contribute greatly to the reduction of blood loss, and the blood pressure is kept at about 90 systolic throughout the procedure Fracture of the petrosa is made lateral to the carotid canal and internal auditory meatus The use of a split thickness skin graft over the operative defect permits rapid healing and reduces the danger of meningitis Equilibrium is lost for a period of five to 20 days after surgery due to sacrifice of the labyrinth, and lateral tarsorrhaphy compensates somewhat for the facial paralysis

Thirty-four cases of cancer involving the temporal bone were subjected to this procedure Fourteen of these are free of disease for periods of six months to five years Eleven of the 34 have survived for three years or more (32 per cent three year salvage) and three have survived more than five years Thirteen cases appeared to be primary in the middle ear and four of these (31 per cent) are three year cures Four cases (12 per cent) of the 34 died of postoperative cerebral hemorrhage or infection of the brain and meninges

SUMMARY

1 Considering the frequency of other affections of the ear, cancer occurs extremely rarely

2 The auricle most often gives rise to basal cell carcinoma Squamous cell carcinoma more frequently arises from the auditory canal and middle ear and may metastasize to regional lymph nodes by a rich lymphatic network Other rare tumors have been classified in this series and are of such rarity as to be curiosities

3 Because of the difference in therapeutic approach, cancer of the external ear has been classified as being early and advanced, ear canal as early and advanced, and middle ear and mastoid as a therapeutic entity The surgical approach for small lesions of the pinna involves some sacrifice of the cosmetic appearance, but is remarkably successful Small lesions originating in the membranous canal are best removed by a sleeve resection with primary skin grafting Advanced lesions of the auricle, ear canal and middle ear where temporal bone is involved require radical surgery The most promising procedure for these lesions in the author's experience is a combined intracranial, extracranial subtotal resection of the temporal bone *en bloc* This operation, while hazardous, upholds the principles of cancer surgery in that cancer-laden bone with a wide margin of normal tissue is removed in a single segment The three year salvage rate for this type of procedure is approximately 32 per cent

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The board of editors announces the founding of *The Journal of Auditory Research*, an interdisciplinary non-profit quarterly devoted to the scientific study of hearing Publication will cover the fields of psychoacoustics, otology, audiology, neurophysiology of audition, speech and communications, auditory aspects of human engineering, musicology, instrumentation for hearing research, and all other aspects of audition Special policies will include quick but thorough editing, rapid publication, and lowest possible cost to subscribers Mr J Donald Harris, of the Medical Research Laboratory, New London, Conn, will be editor of the new journal, and Dr James Jerger, of Northwestern University, will be associate editor The Editorial Policy Board consists of Drs Norton Canfield, Raymond Carhart, Stacy Guild, Henry L Haines, Fred Kranz, Al Liberman, and E Glen Wever The new journal will be published by the C W Shilling Auditory Research Center, Inc, of Groton, Conn Initial publishing costs have been defrayed by a grant from the Beltone Institute for Hearing Research

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submitted to the pathologists in 54 (67 5 per cent) of the cases Twenty-nine of these patients are alive, one with recent local recurrence, 17 died of the disease and eight died of disease not related to the neoplasm The data obtained from an analysis of the patients exhibiting carcinoma of this classification in this series suggests that adenocarcinoma of salivary gland origin is a relatively slow-growing malignant neoplasm with a progressive tendency for local recurrence and a definite trend toward spread to the regional lymph nodes and multiple distant metastases to lung and bone if not completely eliminated by the initial treatment employed This neoplasm accounted for 41 (73 per cent) of the total number of recurrences in the 80 patients in this series and, in addition, exhibited distant metastases in 13 (24 per cent) of the 54 patients afflicted with this tumor

TABLE I

Carcinoma of the Salivary Glands—A Follow Up Survey—80 Cases
Classification of Tumors and Frequency of Occurrence According to Salivary Gland Involved

	Parotid	Submaxillary	Sublingual	Accessory Salivary
Malignant mixed tumor	1	0	0	0
Carcinoma	6	1	1	0
Undifferentiated carcinoma	0	0	0	1
Mucoepidermoid carcinoma	7	2	0	1
Epidermoid carcinoma	5	1	0	0
Adenocarcinoma	29	5	1	5
Carcinoma simplex	9	2	0	0
Papillary carcinoma	1	0	0	0
Cylindroma (malignant)	2	0	0	0
Total	60	11	2	7

Mucoepidermoid carcinoma occurred in ten (12 5 per cent) patients in this series Seven are alive, one with distant metastatic disease, and three are dead of the neoplastic disease A total of four recurrences occurred among three of the patients, one of whom also exhibits the metastases previously mentioned In addition, generalized metastatic disease developed in one patient and pulmonary metastases in another Both of these patients are dead It has been my impression

CARCINOMA OF SALIVARY GLAND ORIGIN

A Follow-Up Survey*

FRANK D LATHROP, M D ,†
Boston, Mass

Eighty patients with microscopically proven carcinoma of salivary gland origin arising in the principal salivary glands or accessory salivary gland tissue have received definitive treatment at the Lahey Clinic during the 15-year period from January 1, 1942, through December 31, 1956 The neoplasm was situated in the parotid gland in 60 patients, the submaxillary gland in 11 patients, the sublingual glands in two patients, and in seven patients the malignant lesion arose in accessory salivary gland tissue located in the lip, cheek, palate, tongue, or floor of the mouth Follow-up information was obtained in every case and, in each instance, at a date later in 1959 than that of the year during which the patient's treatment was first instituted during the 15-year period Thus, for those patients in this series whose treatment was initiated most recently the follow-up period was three years

PATHOLOGY

Nine pathological diagnoses were made of the malignant neoplastic disease found in this group of patients These diagnoses have been divided into five groups in order to demonstrate the frequency with which a carcinoma of a particular classification affected the salivary glands in this series (see Table I)

Adenocarcinoma was the tumor most frequently encountered A pathological diagnosis of adenocarcinoma or one of its subclassifications—carcinoma simplex, papillary carcinoma, cylindroma—was made of the pathological material

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submitted to the pathologists in 54 (67.5 per cent) of the cases. Twenty-nine of these patients are alive, one with recent local recurrence, 17 died of the disease and eight died of disease not related to the neoplasm. The data obtained from an analysis of the patients exhibiting carcinoma of this classification in this series suggests that adenocarcinoma of salivary gland origin is a relatively slow-growing malignant neoplasm with a progressive tendency for local recurrence and a definite trend toward spread to the regional lymph nodes and multiple distant metastases to lung and bone if not completely eliminated by the initial treatment employed. This neoplasm accounted for 41 (73 per cent) of the total number of recurrences in the 80 patients in this series and, in addition, exhibited distant metastases in 13 (24 per cent) of the 54 patients afflicted with this tumor.

TABLE I

Carcinoma of the Salivary Glands—A Follow-Up Survey—80 Cases
Classification of Tumors and Frequency of Occurrence According to Salivary Gland Involved

	Parotid	Submaxillary	Sublingual	Accessory Salivary
Malignant mixed tumor	1	0	0	0
Carcinoma	6	1	1	0
Undifferentiated carcinoma	0	0	0	1
Mucoepidermoid carcinoma	7	2	0	1
Epidermoid carcinoma	5	1	0	0
Adenocarcinoma	29	5	1	5
Carcinoma simplex	9	2	0	0
Papillary carcinoma	1	0	0	0
Cylindroma (malignant)	2	0	0	0
Total	60	11	2	7

Mucoepidermoid carcinoma occurred in ten (12.5 per cent) patients in this series. Seven are alive, one with distant metastatic disease, and three are dead of the neoplastic disease. A total of four recurrences occurred among three of the patients, one of whom also exhibits the metastases previously mentioned. In addition, generalized metastatic disease developed in one patient and pulmonary metastases in another. Both of these patients are dead. It has been my impression that mucoepidermoid carcinoma of the salivary glands is a neoplasm of a relatively low grade of malignancy, but an analysis of the ten cases in this series does not substantiate this impression since recurrent disease as well as distant metastases occurred in 30 per cent of the patients.

A diagnosis of malignant mixed tumor was made in only one instance. Carcinoma of an unspecified nature was diagnosed in eight additional patients, however, and these have been included in this classification of malignant mixed tumor. This has been done because Foote and Frazell,* in their discussion of this tumor in their excellent monograph, state that the major portion of such a tumor is malignant, and an appreciable number of these do not exhibit areas of benign mixed tumor when routinely sectioned and, consequently, are diagnosed as carcinoma. Thus, nine patients (11.25 per cent) are included in this classification in this series of 80 patients. Five of these patients are alive and four are dead, one of disease of an unrelated nature. The tumor recurred on six occasions in five of the patients, and three of these exhibited distant metastatic disease at the time of their death.

Epidermoid carcinoma was encountered in only six patients (7.5 per cent); percentagewise, however, it caused more deaths than any other neoplasm in this series. Half of the patients were dead of malignant disease within one year and three months after treatment; furthermore, only one of the three patients in whom the neoplasm recurred following their initial treatment at the Lahey Clinic is alive without further development of neoplastic disease, while two of the six patients exhibited distant metastases at the time of their death.

Undifferentiated carcinoma occurred in only one patient (1.25 per cent) in this series. This patient is alive and free of disease and has had no recurrence. It is impossible to arrive at any conclusion regarding the degree of malignancy of this tumor in view of the fact that it was encountered in only one instance.

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*Foote, F. W., Jr., and Frazell, E. L.: Tumors of the Major Salivary Glands. Atlas of Tumor Pathology, Section IV, Fascicle 11, 149 pp. Armed Forces Institute of Pathology, Washington, D. C., 1954.

OVER-ALL RESULTS

Analysis of the data obtained from this survey discloses that 27 of the 80 patients have died of the neoplastic disease for which they received treatment, while 42 are alive and without evidence of recurrent or metastatic disease. Two patients are alive with disease, one of whom exhibits a local recurrence and the other multiple distant metastases. Nine other patients have died of unrelated disease without recurrence of or metastases from the original neoplasm. In addition, a total of 55 recurrences developed at one time or another in 37 of the 80 patients comprising this series (see Table II).

TABLE II

Over-All Results

	Cases	Per Cent
Dead—		
Of neoplasm	27	33.7
Of other disease	9	11.3
Alive—		
With neoplasm	2	2.5
Without neoplasm	42	52.5
Total	80	
Recurrence	37	

Of the patients whose deaths were attributed to other diseases, seven were treated for carcinoma of the parotid gland and one each for carcinoma of the submaxillary gland and an accessory salivary gland. All but two of the patients had survived for three to nine years without evidence of recurrent malignant disease. The remaining two patients died within five months after surgical excision of the neoplasm (see Table III).

When the data derived from this study are considered in terms of the salivary gland involved by the malignant process, it is seen that malignant neoplasms of the submaxillary gland are associated with a higher death rate than those of the parotid, sublingual or accessory salivary glands. Seven of the patients with carcinoma of the submaxillary gland died that mucoepidermoid carcinoma of the salivary glands is a neoplasm of a relatively low grade of malignancy, but an analysis of the ten cases in this series does not substantiate this impression since recurrent disease as well as distant metastases occurred in 30 per cent of the patients

A diagnosis of malignant mixed tumor was made in only one instance Carcinoma of an unspecified nature was diagnosed in eight additional patients, however, and these have been included in this classification of malignant mixed tumor This has been done because Foote and Frazell, in their discussion of this tumor in their excellent monograph, state that the major portion of such a tumor is malignant, and an appreciable number of these do not exhibit areas of benign mixed tumor when routinely sectioned and, consequently, are diagnosed as carcinoma Thus, nine patients (11 25 per cent) are included in this classification in this series of 80 patients Five of these patients are alive and four are dead, one of disease of an unrelated nature The tumor recurred on six occasions in five of the patients, and three of these exhibited distant metastatic disease at the time of their death

Epidermoid carcinoma was encountered in only six patients (7 5 per cent), percentagewise, however, it caused more deaths than any other neoplasm in this series Half of the patients were dead of malignant disease within one year and three months after treatment, furthermore, only one of the three patients in whom the neoplasm recurred following their initial treatment at the Lahey Clinic is alive without further development of neoplastic disease, while two of the six patients exhibited distant metastases at the time of their death

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*Foote F W Jr and Frazell E L Tumors of the Major Salivary Glands Atlas of Tumor Pathology Section IV Fascicle 11 149 pp Armed Forces Institute of Pathology Washington D C 1954

In two cases the carcinoma originated in the sublingual glands. One of these patients is dead of the neoplastic disease, and the other is alive without evidence of disease (see Table IV).

FIVE-YEAR RESULTS

Sixty-seven patients have had follow-up studies for at least five years from the date of initial treatment at the Lahey Clinic, for carcinoma of salivary gland origin. The tumor was situated in the parotid gland in 49 patients, the submaxillary gland in ten, the sublingual gland in two and in accessory salivary glands in six patients.

TABLE IV

Total Results According to Gland Involved

	Parotid Cases	%	Submaxillary Cases	%	Sublingual Cases	%	Accessory Salivary Cases	%
Dead—								
Of neoplasm	18	30	7	64	1	50	1	14.3
Of other disease	7	12	1	9	0		1	14.3
Alive—								
With neoplasm	2	3	0		0		0	
Without neoplasm	33	55	3	27	1	50	5	71.4

Eleven of the patients who had been treated for carcinoma of the parotid gland are dead of the disease. Another died of cancer of the stomach nine months prior to the termination of the five-year period. Thirty patients are alive and have exhibited no evidence of recurrent or metastatic malignant disease for five years or more. Six of these had had recurrence of the carcinoma but following additional treatment have had no further recurrence of the malignant process for at least five years. Twenty-four of the 30 patients have never had recurrent disease. In two other patients recurrences developed before the end of the five-year period and after further therapy they are alive but have been free of malignant disease for less than five years. Five other patients were alive at the termination of the five-year interval but presented either local recurrences or metastatic disease.

of the disease, while only three are alive without disease One patient died of unrelated disease

Although carcinoma of the parotid gland occurred more than five times as frequently as that of the submaxillary gland, only 18 patients died of the disease, while 33 are alive and clinically free of recurrence or metastases Seven of the 60 patients in this group have died of unrelated disease and two are alive with persistent disease, one with local recurrence of the neoplasm and the other with generalized metastases

TABLE III

Dead of Other Disease (No Recurrence)

Cause of Death	Interval After Operation		
	Years	Months	Days
Parotid Gland			
Cerebral thrombosis	9	9	19
Acute myocardial infarction	9	8	21
Coronary thrombosis	7	1	5
Bronchopneumonia	6	3	10
Cancer of stomach	4	3	3
Papillary carcinoma of thyroid	3	1	10
Congestive heart failure	0	4	21
Submaxillary Gland			
Coronary thrombosis	0	5	0
Accessory Salivary Glands			
Cirrhosis of liver	3	2	23

Malignant tumors of aberrant tissue origin are generally considered to carry a graver prognosis than do those arising from normally placed tissue Tumors of salivary gland origin which are situated in the tongue, cheek, lip, palate or floor of the mouth are often referred to as being of aberrant salivary gland origin This is a misnomer, since these tumors do not arise from aberrant structures but from normally situated accessory salivary glands which are constant anatomical components of the mucous membrane of these regions In seven patients in this series a malignant neoplasm of salivary gland origin was located either in the lip, cheek, palate, tongue or floor of the mouth Five are alive without evidence of persistent disease, and two are dead One of these died of the carcinoma and the other died of cirrhosis of the liver from a neoplasm of the upper lip Both of these responded satisfactorily to surgical excision (see Table V)

TEN-YEAR RESULTS

Thirty patients in this survey have had follow-up studies for at least ten years from the date of initial treatment for neoplastic disease at the Lahey Clinic The tumor was located in the parotid gland in 22 patients, the submaxillary gland in five, the sublingual glands in one and an accessory salivary gland in two

Of those patients who had been treated for carcinoma of the parotid gland, 11 died of the disease and one of unrelated disease The latter patient had had no recurrence of the original primary adenocarcinoma and had died of cerebral thrombosis nine years and ten months after excision of the neoplasm Five patients who are alive have had no recurrence of the neoplastic process during this period Recurrence had developed in four patients and all four have been alive for at least ten years but not free of disease during the ten-year period One of these patients has since died of acute myocardial infarction just three months short of ten years' freedom from the disease The remaining patient is alive but presents local recurrence of the carcinoma

Only one of the five patients with carcinoma of the submaxillary gland had no recurrence of the tumor and was alive and clinically free of neoplastic disease ten years or more after initial treatment at the Lahey Clinic Another patient has been alive ten years, but the tumor had recurred which necessitated further treatment and, thus, has not been free of malignant disease for ten years The remaining three patients died of carcinoma of the submaxillary gland

Only one patient with carcinoma of the sublingual glands has had follow-up studies for ten years or longer, a recurrence has never developed in this case

Two patients with carcinoma of accessory salivary glands are included in this group One of these had a recurrent neoplasm when originally treated at the Lahey Clinic, and

Four of the ten patients treated for carcinoma of the submaxillary gland are dead of the disease. Another patient died of coronary thrombosis five months after a local recurrence had been excised. Four patients are alive. In two of these the original neoplastic disease had not recurred during the five-year interval, and in the other two recurrent malignant disease had developed but had been eradicated by additional therapy. Both of these patients have been alive for more than five years but have been free of the disease for less than five years. One patient was alive with disease at the end of five years but has since died of pulmonary metastases.

TABLE V

Five Year Follow-Up Results According to Gland Involved 67 Cases

	Parotid		Submaxillary		Sublingual		Accessory Salivary	
	Cases	%	Cases	%	Cases	%	Cases	%
Dead—								
Of neoplasm	11	23	4	40	1	50	1	17
Of other disease	1	2	1	10	0		1	17
Alive, no recurrence	24	49	2	20	1	50	2	33
Recurrence—								
Alive without disease five years after treatment	6	12	0		0		2	33
Alive without disease less than five years	2	4	2	20	0		0	
Alive with neoplasm	5	10	1	10	0		0	

One of two patients with carcinoma of the sublingual gland is dead of the disease, while the other patient is alive without evidence of neoplastic disease.

Of the six patients with carcinoma of the accessory salivary glands, one is dead of the disease, and another died of cirrhosis of the liver without further evidence of malignant disease. Four patients have been alive five years or longer without malignant disease. In two of these recurrent or metastatic disease had never developed after their initial treatment at the clinic. In the other two patients additional neoplastic disease had developed, one a local recurrence and the other metastases to the lymph nodes of the submaxillary triangle from a neoplasm of the upper lip Both of these responded satisfactorily to surgical excision (see Table V)

TEN-YEAR RESULTS

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Only one of the five patients with carcinoma of the submaxillary gland had no recurrence of the tumor and was alive and clinically free of neoplastic disease ten years or more after initial treatment at the Lahey Clinic Another patient has been alive ten years, but the tumor had recurred which necessitated further treatment and, thus, has not been free of malignant disease for ten years The remaining three patients died of carcinoma of the submaxillary gland.

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Two patients with carcinoma of accessory salivary glands are included in this group One of these had a recurrent neoplasm when originally treated at the Lahey Clinic, and

this patient died of cerebral infarction one week after excision of the lesion. This patient has been included among those who died of malignant disease in the over-all statistical results. The other patient had had metastases to the submaxillary triangle from an adenocarcinoma of the upper lip and has had no further recurrence for more than five years (see Table VI).

TABLE VI

Ten Year Follow Up Results According to Gland Involved 20 Cases

	Parotid Cases	%	Submaxillary Cases	%	Sublingual Cases	%	Accessory Salivary Cases	%
Dead—								
Of neoplasm	11 —	50.0	3 —	60	0		1 —	50
Of other disease	1 —	4.5	0		0		0	
Alive, no recurrence	5 —	22.7	1 —	20	1 —	100	0	
Recurrence—								
Alive without disease ten years after treatment	0		0		0		0	
Alive without disease less than ten years	4 —	18.1	1 —	20	0		1 —	50
Alive with neoplasm	1 —	4.5	0		0		0	

RECURRENCE

Forty-six of the patients in this series had had no treatment before coming to the Lahey Clinic, while 34 patients had received treatment elsewhere and exhibited recurrent carcinoma of salivary gland origin. In 34 of the 46 patients who had had no previous treatment the neoplasm was located in the parotid gland, six patients had a malignant tumor of the submaxillary gland, one a malignant tumor of the sublingual gland and, in the remaining five patients, the malignant neoplasm originated in accessory salivary gland tissue. The 34 recurrent tumors involved the parotid gland in 26 cases, the submaxillary gland in five, the sublingual gland in one and the accessory salivary glands in two patients (see Table VII).

A recurrence of the carcinoma, either local or metastatic to the regional lymph nodes, developed in 17 (approximately 37 per cent) of the 46 patients who received their initial treatment at this clinic—in the parotid gland in 11 cases, in the

submaxillary gland in four, and in accessory salivary glands in two In contrast, further recurrent disease developed in 20 (approximately 59 per cent) of the 34 patients who were first treated at the Lahey Clinic for recurrent carcinoma of the salivary glands The additional recurrent neoplastic disease was situated in the parotid gland in 17 patients, the submaxillary gland in two and the sublingual gland in the remaining patient (see Table VIII)

TABLE VII

Status of Malignant Neoplasm at Initial Examination

Gland	Neoplasm Primary	 Recurrent
Parotid	34	26
Submaxillary	6	5
Sublingual	1	1
Accessory salivary	5	2
Total	46	34
	80	

TABLE VIII

Patients Exhibiting Recurrence After Treatment at Lahey Clinic

Gland	Neoplasm Primary Cases	 Per Cent	 Recurrent Cases	 Per Cent
Parotid	11	32	17	65
Submaxillary	4	67	2	40
Sublingual	0		1	100
Accessory salivary	2	40	0	
Total	17		20	
		37		

These 37 patients in whom recurrent carcinoma developed presented a total of 55 recurrences Forty-two of these occurred in the parotid gland, but only 15 (27 per cent) occurred in those patients whose initial treatment was for primary carcinoma as compared to 27 (49 per cent) which developed in patients whose original treatment was for recurrent disease In three of the patients who had been treated initially for primary carcinoma more than one recurrence developed,

this patient died of cerebral infarction one week after excision of the lesion. This patient has been included among those who died of malignant disease in the over-all statistical results. The other patient had had metastases to the submaxillary triangle from an adenocarcinoma of the upper lip and has had no further recurrence for more than five years (see Table VI).

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Of other disease	1 —	4.5	0		0		0	
Alive, no recurrence	5 —	22.7	1 —	20	1 —	100	0	
Recurrence—								
Alive without disease ten years after treatment	0		0		0		0	
Alive without disease less than ten years	4 —	18.1	1 —	20	0		1 —	50
Alive with neoplasm	1 —	4.5	0		0		0	

RECURRENCE

Forty-six of the patients in this series had had no treatment before coming to the Lahey Clinic, while 34 patients had received treatment elsewhere and exhibited recurrent carcinoma of salivary gland origin. In 34 of the 46 patients who had had no previous treatment the neoplasm was located in the parotid gland, six patients had a malignant tumor of the submaxillary gland, one a malignant tumor of the sublingual gland and, in the remaining five patients, the malignant neoplasm originated in accessory salivary gland tissue. The 34 recurrent tumors involved the parotid gland in 26 cases, the submaxillary gland in five, the sublingual gland in one and the accessory salivary glands in two patients (see Table VII).

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TABLE VI

Ten Year Follow Up Results According to Gland Involved 20 Cases

	Parotid Cases	%	Submaxillary Cases	%	Sublingual Cases	%	Accessory Salivary Cases	%
Dead—								
Of neoplasm	11	50.0	3	60	0		1	50
Of other disease	1	4.5	0		0		0	
Alive, no recurrence	5	22.7	1	20	1	100	0	
Recurrence—								
Alive without disease ten years after treatment	0		0		0		0	
Alive without disease less than ten years	4	18.1	1	20	0		1	50
Alive with neoplasm	1	4.5	0		0		0	

RECURRENCE

Forty-six of the patients in this series had had no treatment before coming to the Lahey Clinic, while 34 patients had received treatment elsewhere and exhibited recurrent carcinoma of salivary gland origin. In 34 of the 46 patients who had had no previous treatment the neoplasm was located in the parotid gland, six patients had a malignant tumor of the submaxillary gland, one a malignant tumor of the sublingual gland and, in the remaining five patients, the malignant neoplasm originated in accessory salivary gland tissue. The 34 recurrent tumors involved the parotid gland in 26 cases, the submaxillary gland in five, the sublingual gland in one and the accessory salivary glands in two patients (see Table VII).

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Status of Malignant Neoplasm at Initial Examination

Gland	Neoplasm Primary	Neoplasm Recurrent
Parotid	34	26
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Total	46	34
	80	

TABLE VIII

Patients Exhibiting Recurrence After Treatment at Lahey Clinic

Gland	Neoplasm Primary Cases	Neoplasm Primary Per Cent	Neoplasm Recurrent Cases	Neoplasm Recurrent Per Cent
Parotid	11	32	17	65
Submaxillary	4	67	2	40
Sublingual	0		1	100
Accessory salivary	2	40	0	
Total	17		20	
	37			

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TABLE IX

Total Number of Recurrences After Treatment at Lahey Clinic

Gland	Neoplasm	
	Primary	Recurrent
Parotid	15	27
Submaxillary	8	2
Sublingual	0	1
Accessory salivary	2	0
Total	25	30

Only 15 of these 37 patients in whom recurrence of the neoplastic process developed after their first treatment at the Lahey Clinic are alive in spite of additional therapy for the recurrence. Thirteen are alive and free of malignant disease while the other two are alive but exhibit either local recurrent or metastatic malignant disease. Twenty-two of the 37 patients are dead, two of unrelated disease and 20 of the malignant tumor (see Table X).

METASTASES

Metastases or extension of the malignant process to structures other than the regional lymph nodes occurred in 21 patients (26.3 per cent) in this series and only in those patients with carcinoma of the parotid or submaxillary glands. No apparent relationship of a significant degree appeared to exist between a pathological diagnosis and the incidence of metastatic disease percentagewise, however, there was a definite relationship between the incidence with which metastases occurred and whether or not the neoplasm had recurred locally. Only three of the patients in whom distant metastases developed had not had a local recurrence of the neoplastic disease, and in one of these the original neoplasm had not resolved as a result of the therapy employed.

TABLE X

Status of 37 Patients Treated for Recurrence of Neoplasm Following Initial Treatment at Lahey Clinic

	Cases	Per Cent
Alive—		
Without further recurrence	13	35.1
With persistent or metastatic neoplasm	2	5.4
Dead—		
Of neoplasm	20	54.1
Of other disease	2	5.4

TABLE XI

Metastases or Extension to Structures Other Than Regional Lymph Nodes

Gland	Neoplasm			
	Primary		Recurrent	
	Cases	Per Cent	Cases	Per Cent
Parotid	8	24	7	27
Submaxillary	2	33	4	80
Sublingual	0		0	
Accessory salivary	0		0	
Total	10		11	
		21		

Of 15 patients (25 per cent) with carcinoma of the parotid gland in whom distant metastases occurred, eight had had no treatment for the malignant disease prior to that instituted at the Lahey Clinic (see Table XI). Thirteen of the 15 patients had had local recurrence of their disease, while two patients who had had no treatment before that instituted at the Lahey Clinic had not developed recurrent disease. In one of these two, recurrence had not developed at any time before the metastatic disease was discovered, in the other patient the primary carcinoma had never responded satisfactorily to treatment. Distant metastatic disease developed in six (54.5 per cent) of 11 patients with carcinoma of the submaxillary gland, and only one of these patients had not had a local recurrence of the malignant tumor The malignant tumor in one patient was recurrent when the patient was first seen, and treatment of this lesion proved unsuccessful

Pulmonary metastases occurred most commonly in this series, there being nine instances of metastases to the lungs Metastatic disease involving bone was next in frequency with six such metastatic lesions Metastases or direct extension to the brain occurred on three occasions and to the scalp and liver in one instance each The case of metastasis to the liver is of particular interest as it occurred 16 years after excision of a recurrent carcinoma of the submaxillary gland, without further local recurrence Five patients exhibited generalized metastatic disease (see Table XII)

TABLE XII

Location of Metastases

Lungs			9
Bone			6
	Ribs	2	
	Pelvis	2	
	Skull	1	
	Spine	1	
Scalp			1
Liver			1
Brain			3
Generalized			5

SUMMARY AND CONCLUSIONS

Eighty patients have been treated at the Lahey Clinic for carcinoma of salivary gland origin during the 15-year period from January 1, 1942, through December 31, 1956 Follow-up information regarding the patient's status in 1959 has been obtained in every instance and has been correlated insofar as possible with the day and month of the year the patient first received definitive treatment at the Lahey Clinic Thus, every patient who is now alive has had follow-up studies for three years or longer

Forty-four patients, two of whom exhibited either persistent or metastatic disease, were alive at the time this follow-up survey was completed—a survival rate of 55 per cent. Thirty-six patients were dead, 27 of the malignant neoplastic disease for which they had been treated and the remaining nine patients of other disease, without evidence of recurrent or metastatic disease.

On the basis of this survey, carcinoma of the submaxillary gland must be considered to have much greater lethal potentiality than carcinoma of the parotid gland. Sixty-four per cent of those patients with carcinoma of the submaxillary gland died of the neoplastic process as compared to 30 per cent of the patients with carcinoma of the parotid gland. In addition, the incidence of distant metastases was higher in carcinoma of the submaxillary gland than carcinoma of the parotid gland. Distant metastatic disease ultimately developed in six of the 11 patients (54.5 per cent) with carcinoma of the submaxillary gland, as compared to 15 (25 per cent) of the 60 patients with carcinoma of the parotid gland who exhibited such metastases. The increased incidence of distant metastases with carcinoma of the submaxillary gland apparently was not related to the type of neoplasm diagnosed in this series since there was no significant difference in this incidence among the various tumor classifications. Rather, the incidence of metastases appeared to be related to the incidence of recurrent disease since the percentage of recurrent disease was greater with carcinoma of the submaxillary gland than with carcinoma of the parotid gland.

The malignant neoplasm recurred much more frequently among those patients whose initial treatment at the Lahey Clinic was for recurrent carcinoma. Further recurrence developed in 59 per cent of these patients as compared with a recurrence rate of 37 per cent for the patients initially treated for primary carcinoma of the salivary glands.

Recurrence developed in 11 of 34 patients who received treatment for primary carcinoma of the parotid gland, whereas additional recurrent disease developed in 17 of 26 patients who had recurrent carcinoma of the parotid gland when first treated at the Lahey Clinic. Only three of the 11 patients who had recurrent disease after treatment of their primary carcinoma are alive and free of disease at this time—a salvage rate of 27 per cent. Six of the 17 patients in whom additional recurrence developed after treatment for their original recurrent neoplastic disease are alive and free of disease at this time—a salvage rate of 35 per cent.

Recurrent disease developed in four of the six patients who were treated for primary carcinoma of the submaxillary gland. Only one patient is now alive and free of disease. Further recurrent disease developed in two of five patients who received treatment for recurrent carcinoma of the submaxillary gland when first seen at the Lahey Clinic. Only one of these patients is alive without disease at this time. Thus only two of the six patients treated for recurrent malignant disease of the submaxillary gland are now alive and free of disease. One patient who was treated for a recurrent carcinoma of the sublingual gland had further recurrence and is now dead of malignant disease. Two other patients who were treated for primary carcinoma of the accessory salivary glands and in whom the neoplasm recurred are now alive and free of disease.

Thus, of the 37 patients who had recurrent malignant disease following their original treatment for carcinoma of salivary gland origin during the 15-year period from January 1, 1942, through December 31, 1956, only 13 patients are now alive and free of the malignant process—a survival rate of 35 per cent.

Since all but nine of the carcinomas in this series were situated in the parotid or submaxillary glands, it would seem that the survival rate should be higher than that obtained in this series. Tumors located in these situations are recognizable as such early in their course and are readily accessible to adequate therapy; thus, it would seem that the survival rate of 57.5 per cent for primary carcinomas of these glands obtained in this series could be increased if adequate treatment were instituted early in the course of the disease.

CARCINOMA OF THE TONSIL[*]

JOHN F. DALY, M.D.,[†]
and
MILTON FRIEDMAN, M.D.,[‡]
New York, N. Y.

Cancer of the tonsil accounts for 1.5 to 3 per cent of all cancers of the body. This figure, however, does not indicate the magnitude or importance of the problem for the otolaryngologist whose patients represent a selected population having symptoms in the tonsillar region. There are two parts to the clinical problem of cancer of the tonsil: first is the diagnosis of the early or occult lesion. Because the area is relatively insensitive, early symptoms of cancer of the tonsil are vague and indefinite. Soreness or a sticking sensation on one side of the throat, otalgia or fullness in the upper neck may be the first indication of a new growth. When these symptoms occur in a male past 50 years of age, they are of particular significance because of the higher incidence of cancer of the tonsil in this group. Slight changes in the appearance of the tonsil, redness about a crypt, or a thickened area which bleeds easily should be regarded with suspicion and biopsy specimens taken. Lesions diagnosed at this early stage are curable in a high percentage of cases.

The second part of the problem of cancer of the tonsil is the extensive lesion which presents no difficulty in diagnosis but is a challenge in management. This is a responsibility shared by the otolaryngologist and radiation therapist. Treatment should be based on an accurate estimate of the extent of the disease which can be best accomplished by staging the tumor. This report covers our experience in attempting to stage a series of 30 cases of cancer of the tonsil.

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ANATOMY

The anatomy of the tonsillar region is familiar to every otolaryngologist The tonsillar bed was his cradle and on its pillars he cut his first surgical teeth The glosso-palatine fold, the pharyngo-palatine fold, better known to us as the anterior and posterior pillars, the upper and lower poles and the superior constrictor muscle of the pharynx, are its boundaries This is the anatomical bed for the tonsil, however, cancer does not respect precise anatomical boundaries, it spreads to the adjacent structures where it may grow in bulk, at times even larger than at the site of origin In some lesions it may be difficult or impossible to determine whether the tumor started in the tonsil and spread to the anterior pillar, tongue or palate, or whether it had its origin in one of these sites and later involved the tonsil, however, in most instances the site of origin can be determined The importance of classifying tumors according to site of origin is based on the observation that tumors arising in different sites appear to be biologically different, they behave differently and respond to therapy differently Cancer of the tonsil is biologically different from cancer arising in the anterior pillar, and each should be considered as a separate clinical entity The former tends to be more radio-sensitive, the latter is more radio-resistant The 30 cases in this report are all cancers which arose primarily in the tonsil

The tonsil has a rich blood supply and a well developed lymphatic drainage, this, plus the constant muscular activity of the area provides conditions conducive to early metastasis Undoubtedly, these factors account in part, for the 70 to 80 per cent of clinically palpable nodes when the patient is first seen

PATHOLOGY

Tumors arise either from the epithelium covering the tonsil or from the lymphatic tissue Consistent with many reported series, 80 per cent of the 30 tumors are of epithelial origin Three others were lymphoepithelioma and three were lymphosarcoma We have accepted the method of reporting which groups lymphoepithelioma with lymphosarcoma

STAGING OF CANCER OF THE TONSIL

The necessity for staging in the clinical study of cancer has had widespread acceptance throughout the medical world, and its importance is indicated by the number of national and international committees that are working toward forming acceptable criteria for staging Behind this movement is the basic conviction that reports of cure rate for any form of therapy are not meaningful unless the stage of the lesion has been considered

TABLE I

Carcinoma of the Tonsillar Area

Survival rates reported in literature showing spread of cure rates

	Author	Period	No of Cases	Per Cent Survived Five Years
1	Berven	1925 1945	107	16 2
2	Cade and Ledlie	1939	82	21
3	Coutard	1920 1926	33	18
4	Duffy	1921-1928	49	20
5	Ennuyer and Bataini	1919 1954	534	18
6	Jacox	1930 1958	65	32
7	Martin and Sugarbaker	1930 1935	107	18
8	Parshall and Stenstrom	1926 1950	72	21
9	Paterson	1940 1944	52	12
10	Scanlon et al	1958	46	43
11	Sheline et al	1931 1950	56	19
12	Teloh	1952	142	5
13	Walker and Schulz	1936 1945	39	15
14	Wood and Aird	1934 1945	80	29

The advantages of staging are many It would avoid widespread disparity in reported cure rates of cancer of the tonsil which may be due in part to the difference in distribution of early, moderately advanced and advanced lesions in each series (see Table I), it provides a more accurate basis for comparing results from different institutions, it makes it possible to summate the experience of many clinics, furthermore, the very process of staging disciplines the clinician to make a more thorough investigation of each lesion The fruit

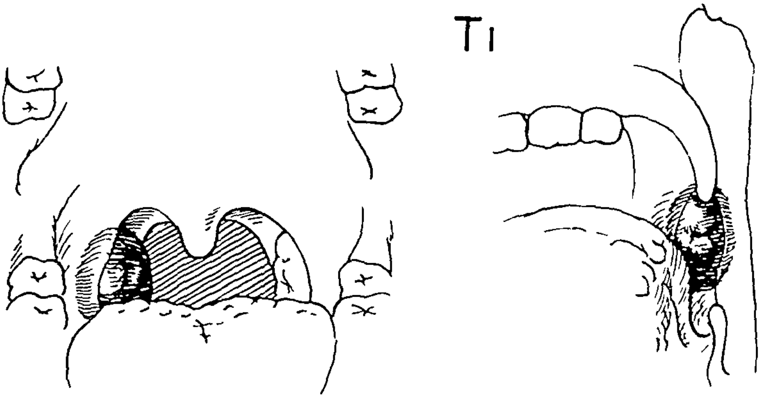


Fig 1-a T1 Confined to the tonsillar bed and less than 3 cm diameter Exophytic and bulky type

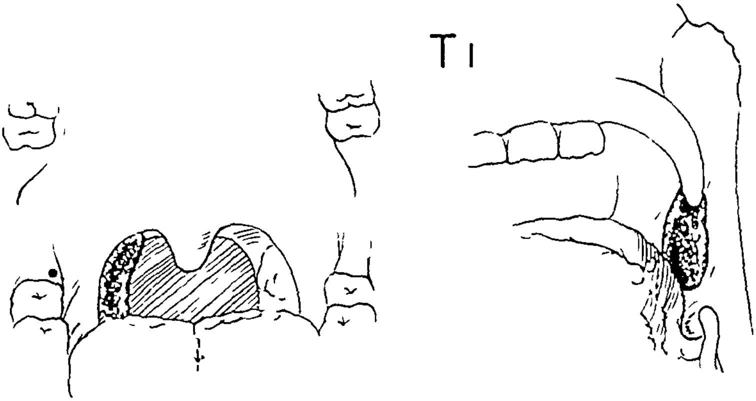


Fig 1-b T1 Ulcerative or granular, flat

of such efforts is a more sound clinical judgment with respect to treatment and prognosis

DESCRIPTION OF STAGING OF THE PRIMARY TUMOR

We have undertaken to stage cancer of the tonsil using the system proposed by the International Union Against Cancer, which is generally referred to as the T N M system

T1 Confined to the tonsillar bed and less than 3 cm diameter (see Fig 1)

a Exophytic, bulky

b Ulcerative or granular, flat

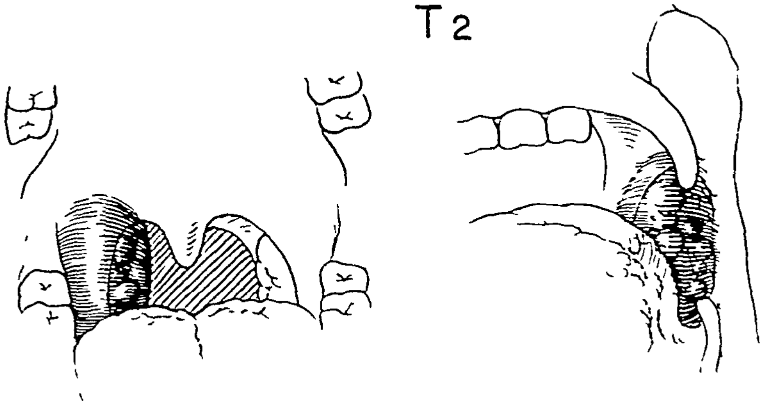


Fig 2-a T2 Minimal extension to adjacent structures approximately 3 to 5 cm diameter Exophytic bulky tumor with predominant extension to hypopharynx

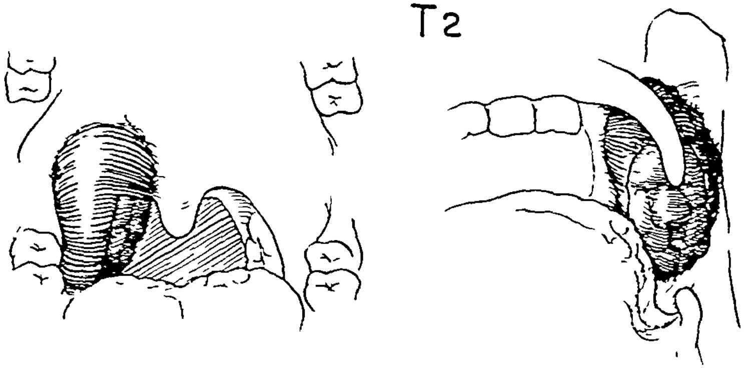


Fig 2-b T2 Exophytic bulky tumor with predominant extension to palate and toward the nasopharynx

T2 Minimal extension to adjacent structures approximately 3 to 5 cm diameter (see Fig 2)

a Exophytic, bulky with extension to hypopharynx

b Exophytic, bulky with extension to palate and toward the nasopharynx

c Ulcerative, flat, with extension into the glossotonsillar sulcus

d Ulcerative, flat, with extension into the tonsillar pillars

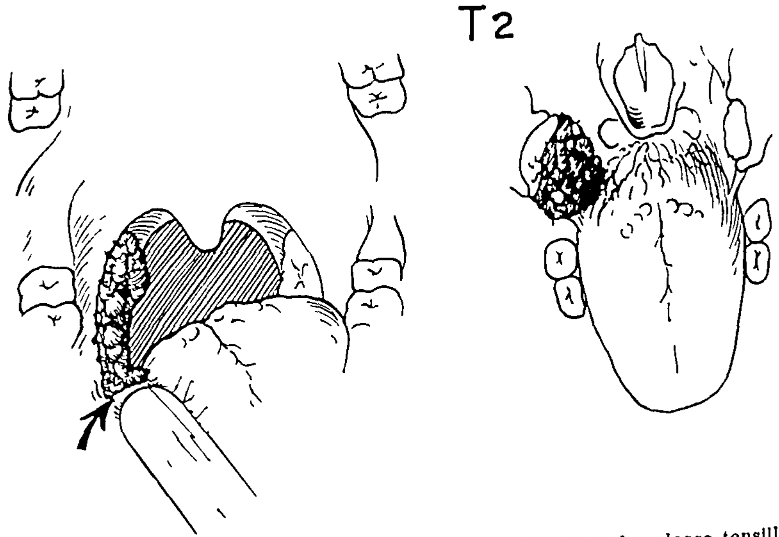


Fig 2-c T2 Ulcerative and flat with extension into the glosso tonsillar sulcus

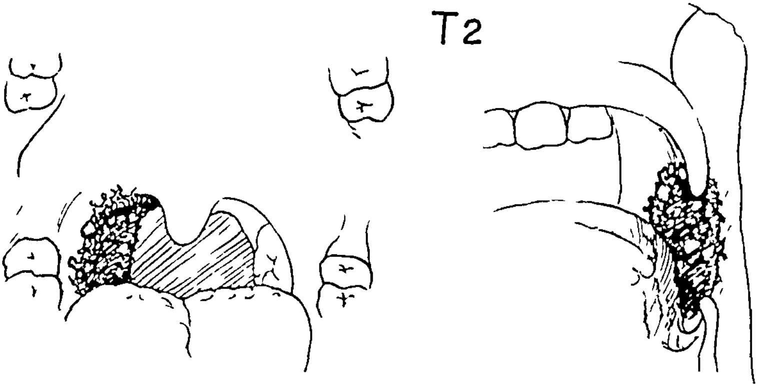


Fig 2-d T2 Ulcerative and flat with extension into the tonsillar pillars

T3 Large or extensive lesion with moderate infiltration into surrounding structures, larger than 5 cm (see Fig 3)

a Exophytic, bulky with extension into nasopharynx and hypopharynx, and forward displacement of the soft palate

b Bulky tumor with submucosal extension into the palate

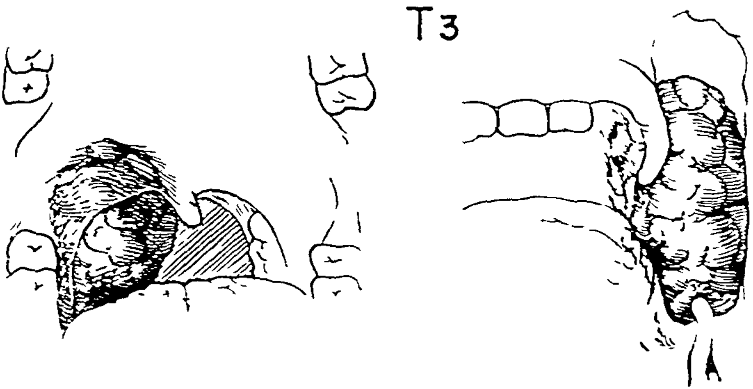


Fig 3-a. T3 Large or extensive lesion with moderate infiltration into surrounding structures larger than 5 cm Exophytic bulky tumor extending into nasopharynx and hypopharynx, and with forward displacement of the soft palate

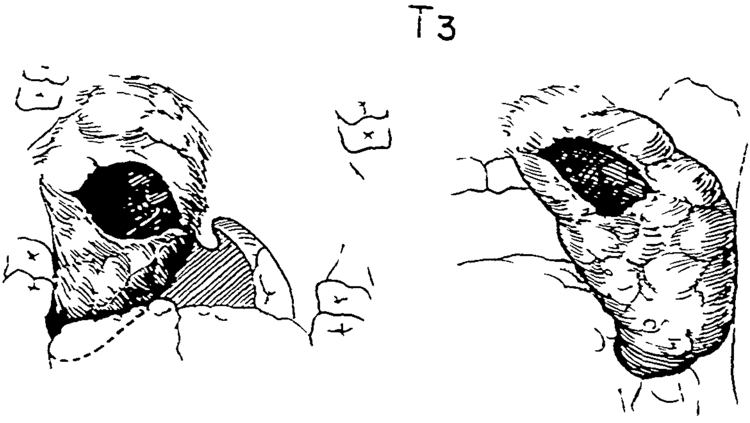


Fig 3-b T3 Bulky tumor extending submucosally into the palate

c Massive tumor with narrow base and moderately infiltrating adjacent structures

d Ulcerative lesion with moderate invasion of adjacent structures

T4 Massive lesion (see Fig 4)

a. Excavating necrotizing lesion with large crater

b Extensive infiltration of tongue with deep linear fissure

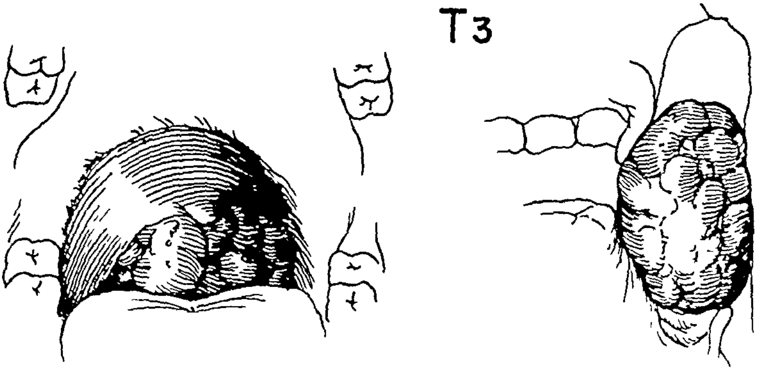


Fig 3-c T3 Massive tumor but with moderate infiltration of adjacent structures at the base of the tumor

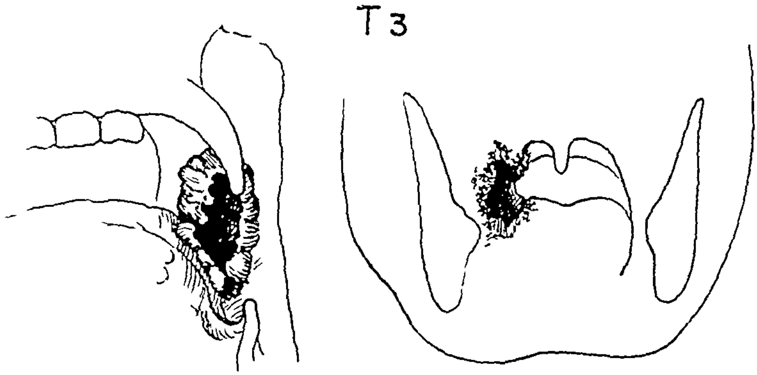


Fig 3-d T3 Ulcerative lesion with moderate invasion of adjacent structures

c Extension across faucial arch, involvement of both tonsils

DESCRIPTION OF METASTATIC CERVICAL NODES

Cervical nodes are classified into four categories from N0 to N3 as follows

N0—No node

N1—Single small to moderate size (less than 3 cm) or two adjacent small nodes (less than 2 cm each)

N2—Large, moveable node (more than 3 cm) or multiple unilateral nodes (more than 2 cm)

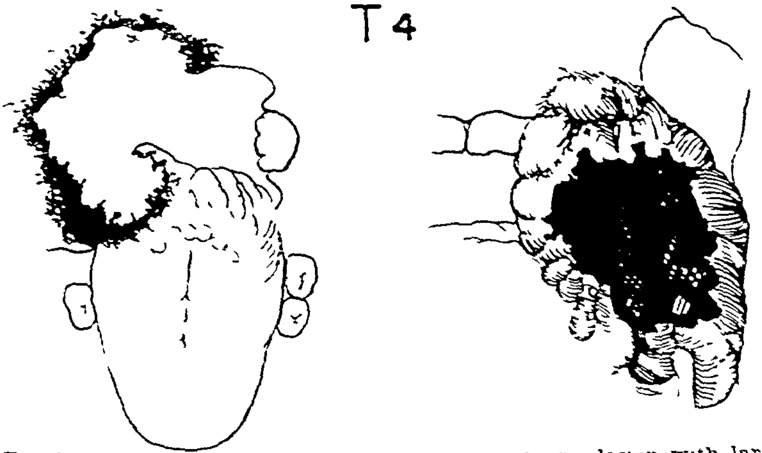


Fig 4-a T4 Massive tumor Excavating necrotizing lesion with large crater

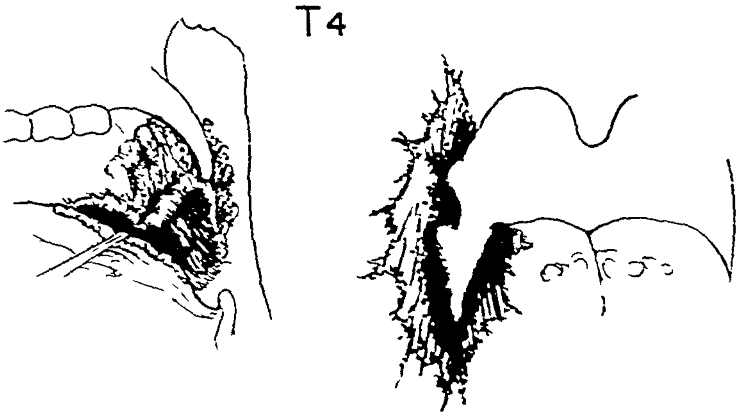


Fig 4-b T4 Extensive infiltration of tongue with deep linear fissure

N3—Fixed large unilateral node or nodes or bilateral nodes

DESCRIPTION OF DISTANT METASTASIS

Distant metastasis is designated as M, followed by a description of the site

DESCRIPTION OF STAGES OF CANCER OF THE TONSIL

The T N M categories make it possible to describe any malignant lesion These categories are analyzed and separated into groups indicating the stage of the lesion as early, moderately advanced, advanced, and incurable The stages are designated Stage I through Stage IV, as follows

Stage I—T1NO, T2NO The cure rate should be relatively high

Stage II—T1N1, T2N1, T3NO, T3N1 Cure rates are expected in moderate percentages

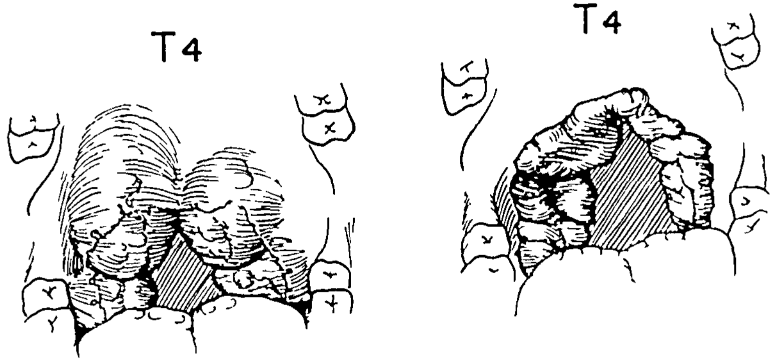


Fig 4-c T4 Extension across faucial arch involvement of both tonsils

Stage III—T1N2, T2N2, T3N2, T4NO, T4N1 Cure rate is extremely low, but aim of treatment should be a cure

Stage IV—T4N2 and all combinations of T with N3, also M (distant metastasis) Incurable except for rare unexpected case

The stages are represented on the T N diagram (see Fig 5) with heavy lines The distribution of the 30 cases is shown by the black dots

Classification and staging, expressed in its simplest terms, is an attempt at speaking a common language An early lesion fits in the definitions of Stage I, a moderately advanced lesion is Stage II, an advanced lesion is Stage III, and far advanced or lesions with a hopeless prognosis are Stage IV All combinations with M would be considered incurable and are placed in Stage IV

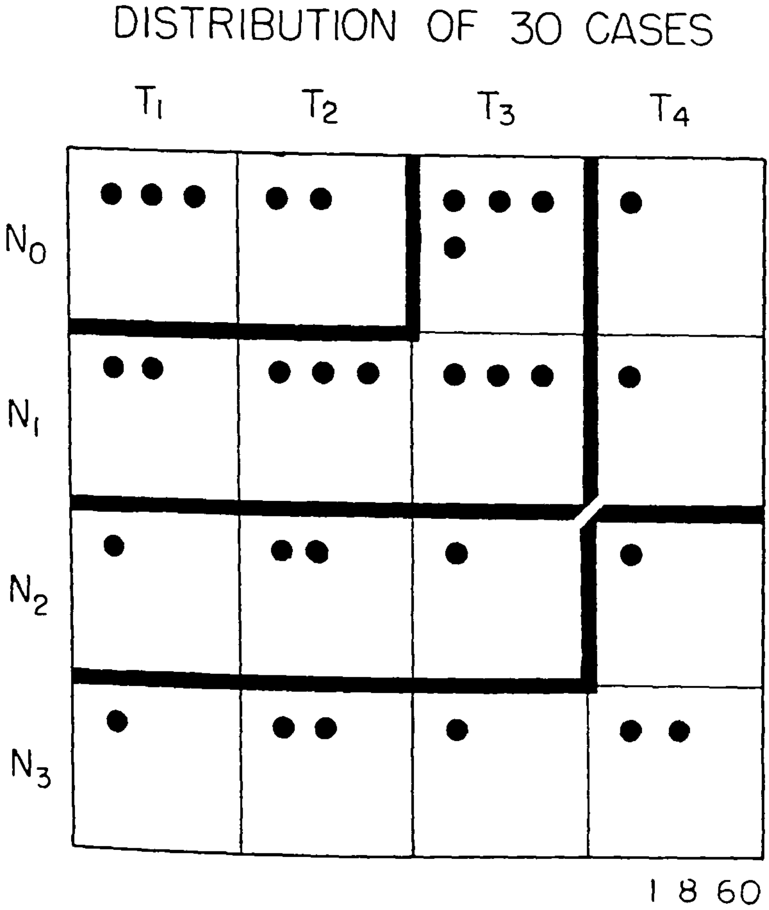


Fig 5 Distribution of the 30 malignant tumors of the tonsil in this series It includes 24 epitheliomas three lymphoepitheliomas and three lymphosarcomas

TREATMENT

a Radiation Therapy

Supervoltage radiation, either with an X-ray machine or cobalt bomb, has been one of the advances in modern treatment of cancer New techniques, particularly rotation, have further improved the effectiveness of radiation All 30 patients in this report have been treated with a two million volt X-ray machine using the rotation technique (see Fig 6)

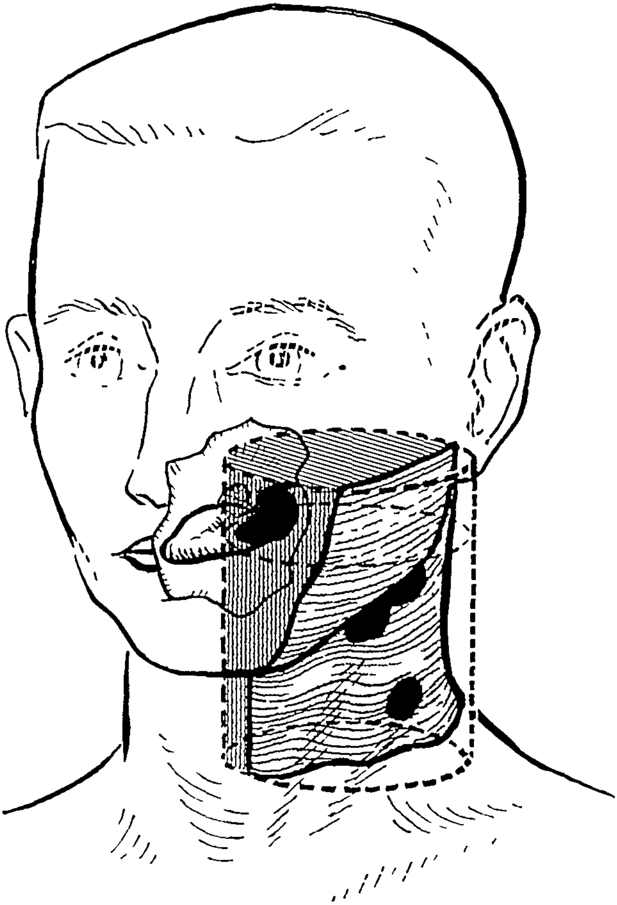


Fig 6 Distribution of irradiation achieved by supervoltage X-rays and the rotation technique The partially immersed cylinder of intense irradiation includes the primary tumor and neck nodes in continuity

This technique delivers a concentrated dose of radiation in the region of the tumor and the neck nodes with minimal irradiation effect on the adjacent normal tissues For cancer of the tonsil the shape of the volume of intense irradiation is a cylinder which includes the tonsillar region and the upper neck nodes in continuity The accuracy of the rotation is and neck at the level of the lesion

Cancer of the tonsil is one of the more radio-sensitive cancers of the upper respiratory tract, checked on an X-ray film inserted in a phanthom of the head, and consequently lends itself to treatment with external radiation alone Most lesions can be destroyed with tumor dosage ranging from 5500 to 6500 Roentgens in three to five weeks An occasional squamous cell carcinoma may be resistant and require a larger dose

The skin reaction is mild as a result of supervoltage X-rays Mucous membrane reaction is confined to one side of the pharynx by virtue of the rotation technique, this markedly reduces the discomfort of the patient which, in the past, has often been a limiting factor

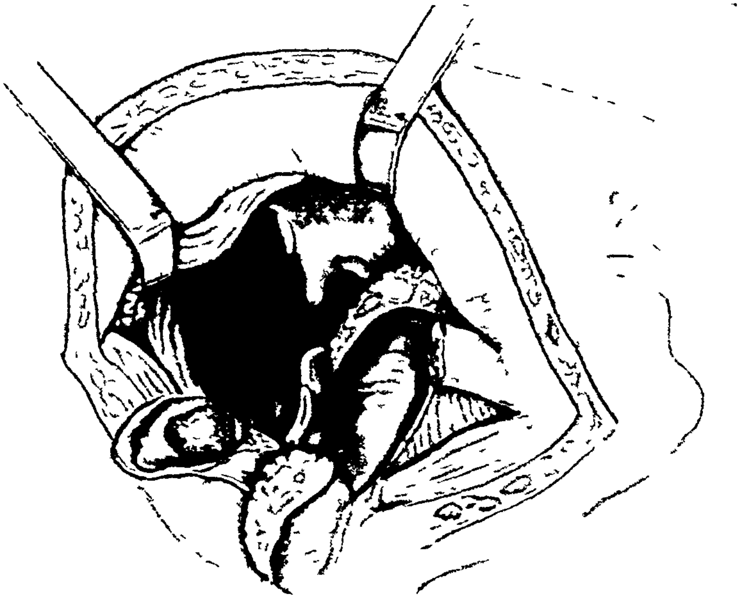


Fig 7 Surgical resection of tonsillar region (modified from Ennuyer)

b Surgery

Surgery has an important but secondary role to play in the management of cancer of the tonsil The indications for surgery are three *1* failure of radiation therapy to control the primary tumor, or recurrence of the primary lesion, *2* cervical nodes which are palpable following radiation, particularly in the more radio-resistant carcinomas and *3* osteomyelitis of the mandible or non-healing radiation ulceration of the tonsillar region The sloughing ulceration and osteomyelitis produces intractable pain and dysphagia which makes it impossible for the patient to maintain his nutrition The complications of radiation therapy can be as fatal as the cancer itesIf Death may come from a sudden massive hemorrhage A wide surgical excision relieves the patient of the foul slough and the intractable pain and restores his well-being

The area excised includes the primary site, portion of the mandible, palate, pharynx, pterygoid muscles, floor of mouth and tongue (see Fig 7)

Closure is accomplished by using the skin and soft tissues of the neck The pharyngeal defect is too large for a primary closure and is allowed to granulate in

It is remarkable that the external closure holds without fistula formation, in spite of the fact that the tissues have been heavily irradiated Two factors that determine whether the closure will be maintained are *1* the period of time elapsing between the completion of radiation and the surgery and *2* the total amount of radiation given to the area

If the surgical excision is done within the first year and the radiation tissue dose had been less than 6000 Roentgens with supervoltage X-rays, secondary wound breakdown will be infrequent The cosmetic and functional results are acceptable (see Figs 8-a and 8-b)

RESULTS

Our cases are small in number and have not matured, hence, reporting our experience will not have the weight of a larger series observed for a longer period of time, however, our series has the virtue of presenting a single form of therapy managed by one group Although final judgments cannot be made concerning the effectiveness of supervoltage rotation technique, a review of our experience at this time is

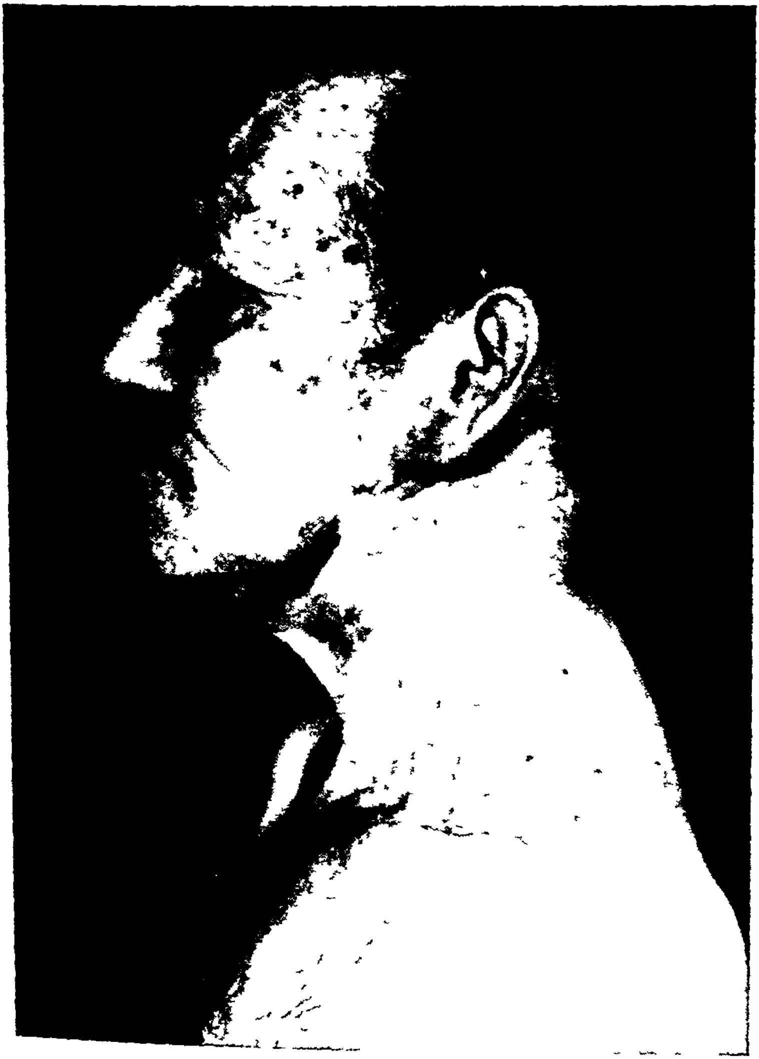


Fig 8-a Postoperative photograph (lateral view)

of some value The course of each patient is recorded on the flow chart (see Fig 9)

The results among the 22 patients with epithelioma and lymphosarcoma and lymphoepithelioma of the tonsil, treated more than two years ago, were first studied (see Fig 10) Though the number of cases is too small to be significant,

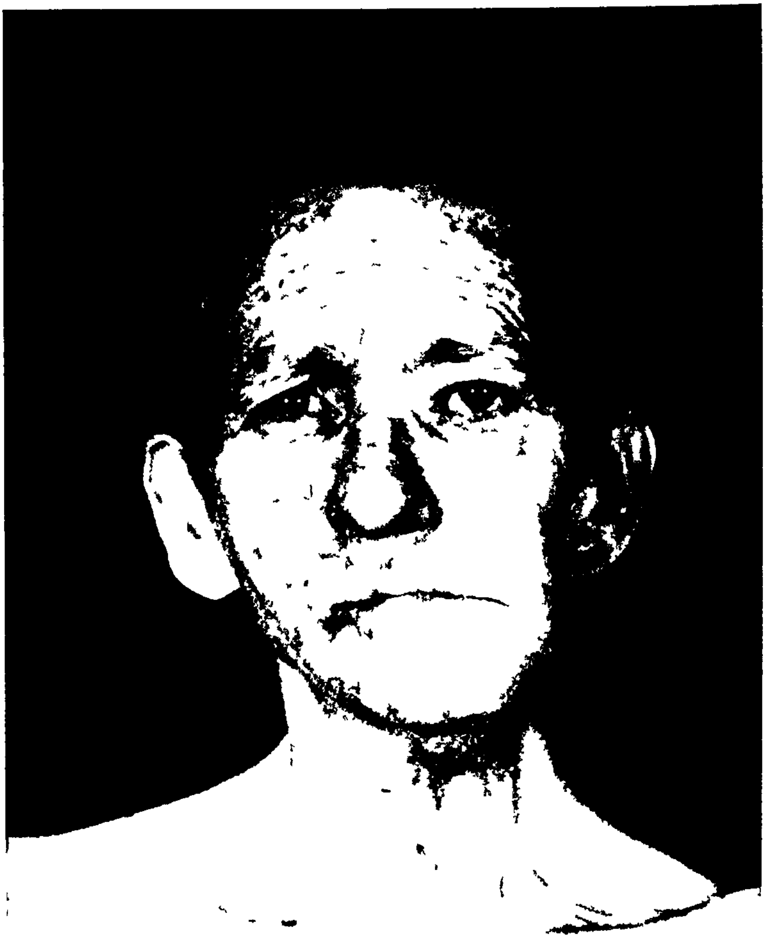


Fig 8-b Postoperative photograph (front view)

there appears to be a correlation between stage and prognosis In the Stage I cases, the two year arrest rate was 100 per cent, Stage II, 66 per cent, Stage III, 33 per cent and in Stage IV none was arrested In the past it has been assumed that in defining progressive stages of disease, metastatic nodes constituted a more advanced stage than no nodes Our opin-

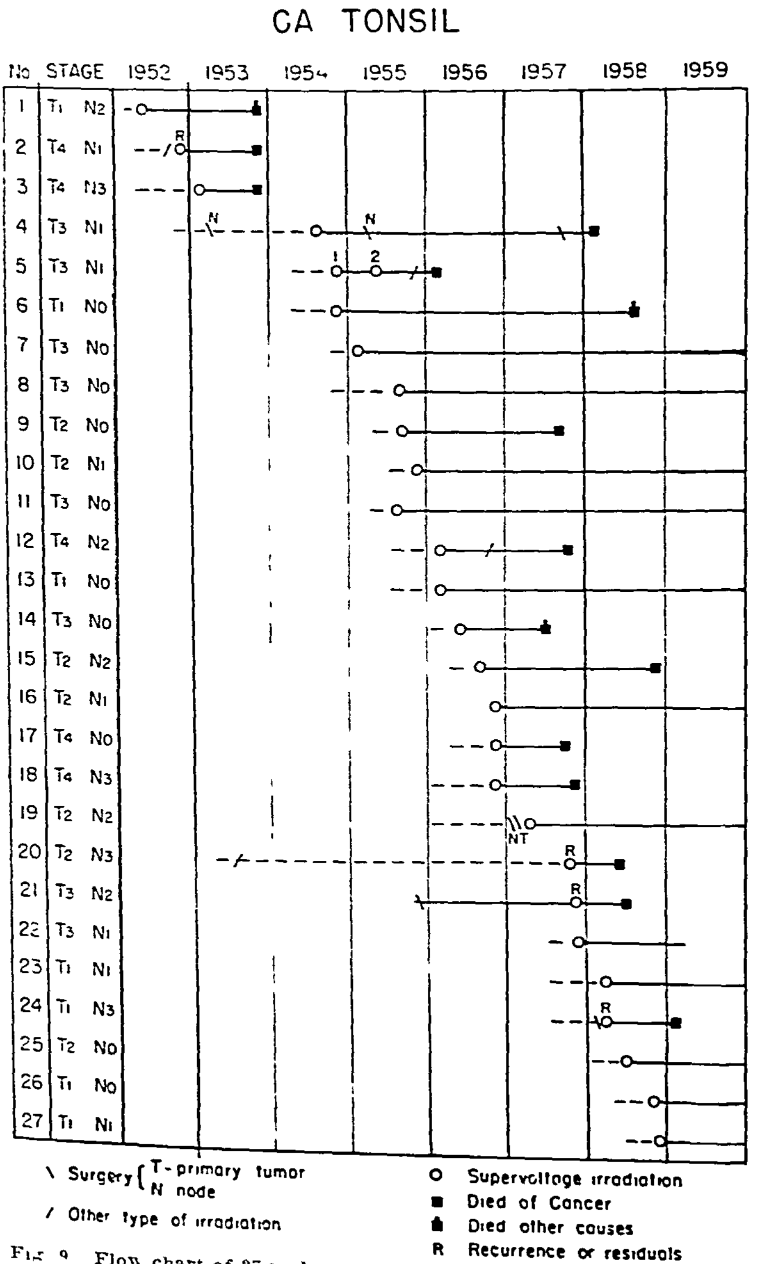
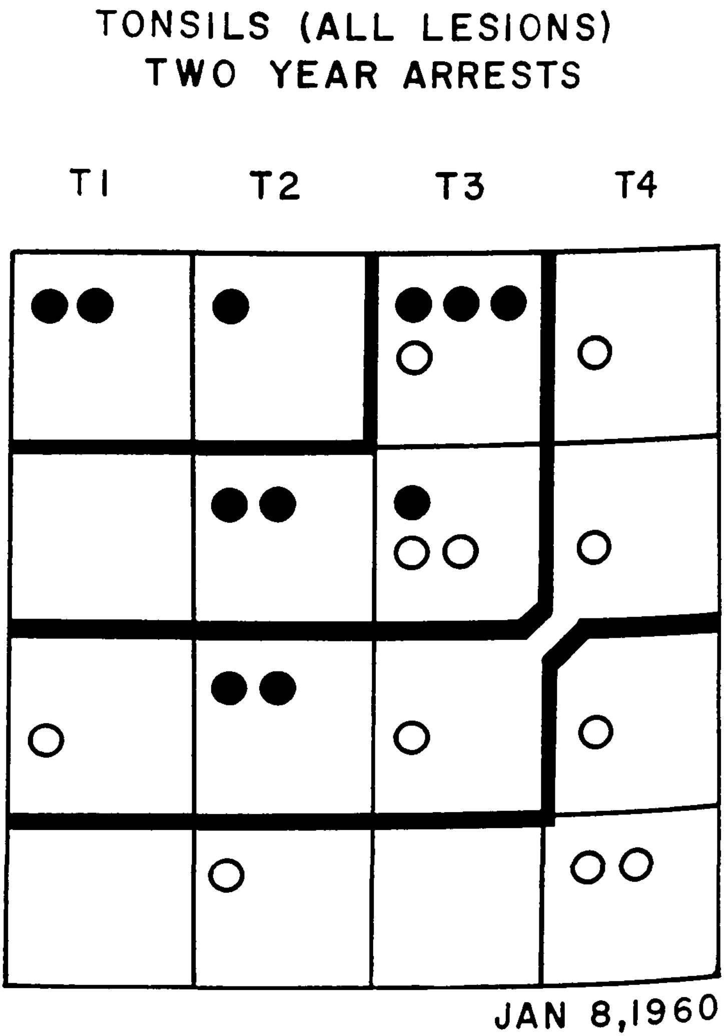
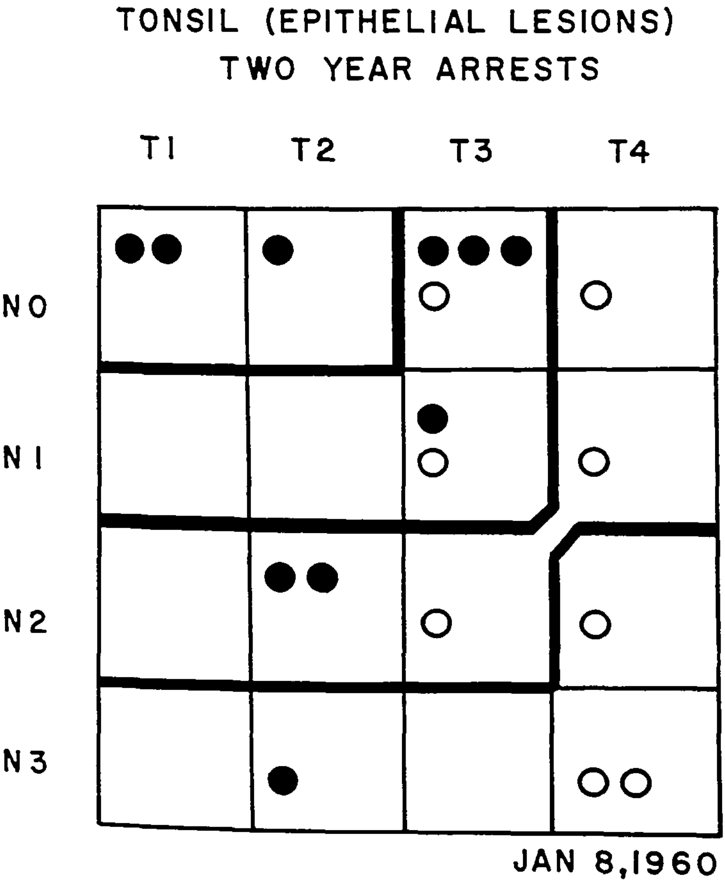


Fig. 9. Flow chart of 27 malignant tumors in this series. Three patients treated less than one year ago are not depicted. The dotted line represents duration of symptoms prior to irradiation. Three patients who died of other causes are considered as cancer deaths. Case 22 could not be traced after 15 months and is counted as dead. Eleven patients are free of disease January 8, 1960.



STAGE	I	3/3	100%
"	II	6/9	66%
"	III	2/6	33%
"	IV	0/4	0%

Fig 10 Study of results in 22 cases treated more than two years ago. All cases epitheliomas lymphosarcomas and lymphoepitheliomas are included Solid dot represents tumor arrest circle represents death Note the good correlation between stage and tumor arrest Note that a massive primary tumor without nodes (T4 NO) may have a worse prognosis than a smaller tumor with small nodes (T2 N1)



STAGE	I	3/3	100%
"	II	4/6	66%
"	III	2/5	40%
"	IV	0/4	0%

Fig 11 Study of results among 18 epitheliomas The solid dots represent two-year arrests the circles deaths Once again is seen the good correlation between stage and tumor arrest

ion, that a massive primary tumor without nodes (T4, N0) has a worse prognosis than a smaller primary with small nodes (T2, N1), seems to be upheld by the analysis in Fig 10

The 18 epitheliomas were analyzed (see Fig 11) This analysis again showed the correlation between stage and prognosis

A final summary of the 29 patients in this series (all histologic types), treated more than one year ago, is presented in Table II The three year apparent arrest rate is 44 per cent

TABLE II

Survival in All Cases of Malignant Lesions of the Tonsil

Final summary of all cases in this series (all histologic types), at risk more than one year

Year of Initial Treatment	New Cases	Years Arrested 1	2	3	4	5
1952	2	2	–	–	–	–
1953	1	–	–	–	–	–
1954	3	3	2	2	–	–
1955	5	5	5	4	4	1
1956	7	6	3	2	1	–
1957	4	2	1	–	–	–
1958	5	4	–	–	–	–
1959	2	2	–	–	–	–
Cases at Risk	29	27	22	18	11	6
Number Arrested		24	11	8	5	1
Per Cent Arrested		89	50	44	45	16

January 8 1960

and the five year rate is 16 per cent, however, these figures are not statistically significant because of the small number of cases

From the analysis of this small series of cases treated with supervoltage rotation technique, we have not demonstrated a significant improvement in the cure rate, however, our clinical experience convinces us that supervoltage rotation technique is an improvement over conventional methods by virtue of its sparing of tissues and the lessening of discomfort to the patient

CONCLUSIONS

1 Cancer of the tonsil is a clinical entity, biologically dif-

ferent from cancer arising in adjacent sites, such as tonsillar pillar and retromolar fossa

2 A method of staging cancer of the tonsil has been described, following the T N M system recommended by the International Union Against Cancer

3 Treatment of cancer of the tonsil is primarily by radiation therapy, surgery plays an important but ancillary role in the management of failures and complications of radiation therapy

4 Supervoltage rotation irradiation has the advantage of reducing the severity of radiation reactions and provides an efficient means of delivering the necessary tumor dose to the primary tumor and metastatic neck nodes in continuity

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CRITERIA FOR OTOSCLEROSIS SURGERY AND FURTHER EXPERIENCES WITH ROUND WINDOW SURGERY*†‡

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INTRODUCTION

Since the inception of Lempert's[1] fenestration procedure for correction of hearing impairment incurred by otosclerosis many changes in the corrective procedure have come about We are no longer restricting our efforts to by-passing the pathology produced by otosclerosis Our efforts have turned to replacing the pathological structures with artificial ossicular chains and stapedial footplates

The limitations inherent in the fenestration procedure are no longer a problem The new techniques evolved from Rosen's[2] original concept are not limited by the complete absence of an ossicular continuity or by the restrictions that accompany a derangement of the phase relations between the oval and round windows and the transmission characteristics of the tympanic membrane and tympanic cavity The moderately limited success that accompanies fenestration surgery has been established by time and experience Whereas the success that accompanies oval window surgery, whether it be stapedial mobilization or stapedial replacement is still to be determined and tested by time

Theoretically the success of fenestration surgery was handicapped by changes in middle ear physiology which had no

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chance of elimination Because the drum-head and ossicular chain are by-passed the average patient is left with a 20-25 db air-bone gap On the other hand, oval window surgery offers the possibility of nearly complete, if not complete, restoration of middle ear function On the one hand the defects created by fenestration surgery were fixed, and the surgeon was forced to accept the limited success In the case of oval window surgery, however, new techniques and improvement in the materials used to substitute defects in the ossicular chain offer a real chance of complete correction of the middle ear deficiency created by otosclerosis

INDICATIONS FOR SURGERY IN OTOSCLEROSIS

When fenestration surgery was the most commonly used method of correcting the hearing defect produced by otosclerosis, certain criteria were used to determine the suitability of the cases

It is generally recognized that the upper level of normal hearing for everyday speech is 15 db at the principle speech frequencies 500, 1000 and 2000 c/s It is also generally accepted that when an average of these three frequencies reaches 30 db the individual will have enough difficulties hearing and understanding everyday speech to require some kind of assistance In view of the magnitude of the fenestration procedure and the limitations placed upon the patient by the postoperative care and restricted activity, fenestration surgery was seldom done unless the anticipated result assured a threshold of at least 30 db at the speech frequencies Practically this means that unless the surgeon can be sure of reaching a hearing level that will require no amplification to assure practical hearing for speech he will seldom perform fenestration surgery The usual criteria for fenestration surgery were a preoperative air-bone gap of 30 db or more and bone conduction hearing levels no higher than 20 db at 500 and 1000, and 30 db at 2000 c/s

Obviously these criteria limited the number of otosclerotic patients who were suitable for surgery which in turn severely limited the usefulness of fenestration surgery for otosclerosis

Obviously if temporal bone surgery was to correct all the problems created by otosclerosis, methods, whose indications were less restricted and whose contraindications were not a problem, must be developed

Rosen's technique later amplified by Shea[3] seemed to produce better hearing levels and better air-bone gap closures with few of the disadvantages of fenestration surgery, however, we believe that the usefulness of oval window surgery has been severely restricted by the use of criteria or indications originally established for fenestration surgery

If the full benefit of this modern technique is to be realized the question of criteria must be carefully reviewed with the advantages and disadvantages of oval window surgery in mind We believe the criteria used for selecting patients for fenestration surgery are not suited to oval window surgery and that skillful surgeons should extend their efforts beyond the limits imposed on the basis of past experience with fenestration surgery The principles and concepts of fenestration surgery are not comparable to those of oval window surgery, and before criteria for selection of patients are established the therapeutic possibilities offered by oval window surgery must be thoroughly investigated and understood

We draw your attention to some of our recent thinking about this problem Several important questions are immediately brought to mind first, should there be a maximum air conduction threshold above which surgery should not be attempted? Second, should there be a maximum bone conduction threshold above which surgery should not be attempted? Third, should a hearing aid be used to supplement the benefits of surgery?

Theoretically, regardless of the maximum air conduction and bone conduction loss, if there is an air-bone gap, reestablishing the ossicular function should produce a better hearing level, at least equal to the true sensory-neural hearing level Measurements of high air conduction thresholds and particularly high bone conduction thresholds are restricted because of equipment limitations When air conduction levels of 85-95 db are encountered it is frequently impossible to obtain bone conduction responses with our present equipment. It is entirely possible that many of these cases have air-bone gaps of as much as 35-40 db. Assuming that this is true, it is quite probable that oval window surgery will close this gap and make it possible for these patients to wear a hearing aid. Results of this kind are much more important to these patients than to patients with a 30 db gain whose threshold levels are 35-45 db.

These broader concepts open up greater possibilities for the rehabilitation of impaired hearing patients than have ever been possible before.

CLINICAL PROGRESS OF OTOSCLEROSIS

Otosclerosis can produce a pure conductive hearing loss with maximum hearing levels of 50-60 db in the speech frequencies. Otosclerosis also can produce a mixed type hearing loss whose maximum levels are really not thoroughly established. Whether otosclerosis produces a pure sensory-neural hearing loss is still a controversial subject. Those who believe this condition exists are hard put to explain its causal relations. A better understanding of the sensory-neural and mixed hearing loss found in patients who obviously have clinical otosclerosis will undoubtedly lead to new methods for correcting the impairment found in these individuals.

Diagnosing otosclerosis, when no bone conduction responses are found and there is a profound loss by air conduction, is very difficult. If the patient gives a history of a slowly progressive hearing loss the most likely diagnosis is otosclerosis. Other possibilities such as ototoxic drugs, viral disease, Meniere's syndrome, auditory nerve tumors and syphilis must be considered; but these conditions are rare compared to otosclerosis.

It has been known for several years that the effects of otosclerosis on the temporal bone can be seen by X-ray. Recently Compere[4] has emphasized this again. In our opinion, if profound hearing loss is not accompanied by increased density in the temporal bone demonstrated by X-ray the diagnosis of otosclerosis is highly improbable.

When a diagnosis of otosclerosis is made in a patient with a profound hearing loss (greater than 80 db) we believe the middle ear should be explored These explorations have led to a number of interesting observations that need further study

There are no important contraindications to middle ear explorations Patients with profound hearing loss are in dire distress and help of any kind is important to them If there is even a slight chance that surgery will improve the hearing level, middle ear surgery should be carefully considered

SURGICAL AND HISTOPATHOLOGICAL OBSERVATIONS IN ADVANCED OTOSCLEROSIS

The following are a few of the interesting observations we have made during the past three years

First, some of these patients have normal appearing stapes, normal oval and round windows and when an opening is made in the footplate the perilymph appears to be normal The hearing remains unchanged after surgery Unless the diagnosis of otosclerosis based on X-ray findings is incorrect in these cases the loss must be due to pure sensory-neural pathology If otosclerosis is the precipitating cause the site of the pathology must be outside the middle ear Bruhl in 1910 and Guild in 1954 reported histologic evidence of otosclerosis in areas other than the middle ear in the temporal bones of patients with profound hearing loss

Several patients have been observed with normal middle ear structures, but when pulsating pressure is applied to the stapes no round window reflex is seen There is no substantiated explanation for this, but it appears that some sort of obstruction must be blocking the continuity of motion between the two windows

Two patients were seen with normal oval and round windows, but the perilymph was thick and gelatinous Siebenmann reported finding cell free exudate in the cochlea of a patient with otosclerotic closure of the cochlear aqueduct in 1911

Some patients have been found with all degrees of stapes fixation from easily mobilized ligament fixation to complete obliteration of the oval window by otosclerosis The round window of these patients may be normal, constricted with a normal or calcified membrane, or totally obliterated along with part of the scala tympani, by otosclerosis We have observed no patient with a normal stapes and oval window, who showed closure of the round window

The histopathology of stapes fixation in otosclerosis is well documented in the literature starting with the first observations of Valsalva[8] in 1735 Nager and Fraser[9] published the histologic findings of six cases showing otosclerotic closure of both windows in 1938 They point out that closure of both windows is found only in patients who have total or almost total hearing loss They point out also that there are very few of these cases In general these cases show diffuse otosclerosis with some very active areas It is rare, indeed, for otosclerosis to invade the labyrinthine spaces In the six cases reported by Nager and Fraser, five showed closure of the cochlear aqueduct All cases showed total closure of the round window

Troltsch[10] in 1873 reported a 16-year-old profoundly deaf case with a fixed stapes and a constricted round window niche The round window membrane was white, thickened and completely calcified

Habermann[11] reported a case with total otosclerotic closure of the round window but no involvement of the stapes or oval window This case demonstrated a closed connective tissue tract leading from the tympanic cavity lining to the scala tympani The patient showed almost total loss of hearing in one ear and minimal loss in the other ear. The totally deaf ear showed complete closure of both windows Habermann concluded from this that the round window was of little importance to hearing

Goodhill, et al ,[12] was able to show a loss of approximately 10 db for bone conduction and air conduction in the speech frequencies when the round window in animals was closed artificially Wever and Lawrence[13] had great difficulty producing a hearing loss in cats by blocking the round window They concluded that in the presence of normal middle ear structures, blocking the round window would affect the high tones more than the low tones Massive blockage of the round window raised the threshold only 20 db in any of the frequencies tested

We have not observed any patients with otosclerotic closure of the round window only The effect of round window closure on the pure tone threshold and the speech discrimination score is not known

SURGICAL TECHNIQUES

One year ago a preliminary report[14] was published on oval and round window surgery in extensive otosclerosis As indicated in this report the first cases were done through an endaural incision This approach made it possible to use the dental drill to remove the thick otosclerotic bone at the windows Subsequently, local anesthesia transcanal techniques were developed We felt that opening both windows at the same time resulted in excessive acoustic trauma and loss of perilymph, therefore, we changed to a two-stage procedure the oval window was opened first and then the round window a month or more later Since the original report five two-stage procedures have been completed

The first stage operation may be exploratory only, depending upon the findings As indicated above, we may find no window pathology to massive involvement of both windows If minimal fixation is found with a blue footplate, the head and crura of the stapes are removed, the footplate is then shattered and polyethylene strut is placed between the incus and footplate which may or may not be covered with gelfoam

If massive otosclerosis is found, it is removed with a dental drill, and the open oval window is covered with either a vein graft of gelfoam, after which a polyethylene strut is placed between the lenticular process of the incus and the oval window

The above method is a standard procedure for re-establishing the ossicular chain continuity in all degrees of conductive or mixed otosclerotic hearing loss

At the second stage of the operation the round window was opened Local anesthesia with a transcanal approach was used Round window closure can vary from calcification of the round window membrane to complete obliteration of the niche, the membrane, and basal part of scala tympani In some cases the round window niche appears to be filled in, but usually a fibrous tract remains extending from the tympanic mucosa to the scala tympani This fibrous tract can be used by the surgeon as a guide for drilling away otosclerotic bone to reopen the scala tympani In other cases a smooth depression in the promontory is all that remains of the round window In these cases the landmarks to the scala tympani are completely obliterated

When opening the round window or scala tympani with a bur, it is important to stay inferior (toward the hypotympanum) to avoid the scala media and medial (toward the brain stem) to avoid the scala vestibuli, since it is on the medial side of the scala tympani throughout most of the basal portion of the cochlea The round window was considered open when perilymph escape was noted which transmitted pulsation movements from the oval window

The new round window opening has been closed with gelfoam, vein graft and middle ear mucosa We do not feel this is an important factor since the endosteum probably quickly seals over the opening

CASE REPORTS

Case 1 (See Fig 1) This patient is a 42-year-old baker with a hearing loss for 15 years X rays show bilateral petrosal sclerosis Both ears had approximately the same loss In the right ear the preoperative speech reception threshold was 87 db with a discrimination score of 18 per cent There was a definite air bone gap with bone conduction response at 15 db for 250 c/s 30 db at 500 c/s, 50 db for 1000 c/s and no response for bone above this

In January 1959 the oval window was found to be fixed with hard white otosclerotic bone This was thinned with a bur, fractured and depressed into the vestibule A polyethylene strut was inserted between the incus and the depressed footplate

Following surgery the bone conduction remained the same but the speech reception threshold improved to 62 db Speech discrimination producing a hearing loss in cats by blocking the round window They concluded that in the presence of normal middle ear structures, blocking the round window would affect the high tones more than the low tones Massive blockage of the round window raised the threshold only 20 db in any of the frequencies tested

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The above method is a standard procedure for re-establish-remained approximately the same at 16 per cent. One month later in February, 1959 the round window was opened In this case it was necessary to remove only a small amount of bone Clear perilymph escape was noted and this moved in opposite phase to incus pulsation There was a 20 db drop in hearing on the table

The hearing was depressed considerably at first and has gradually improved In December, 1959, the bone conduction was the same as preoperative and the speech reception threshold had improved to 52 db with a discrimination score of 26 per cent

Comment Opening the oval window was of definite benefit In this case the round window could be opened with little difficulty, however, it took a long time to regain the level attained by the oval window surgery alone

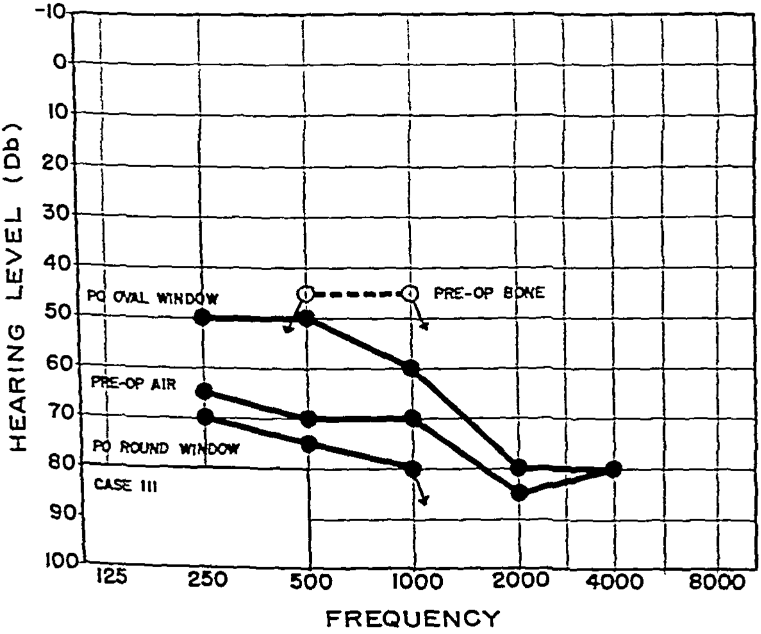


Fig 3

Case 2 (See Fig 2) A 54-year-old housewife with hearing loss for 20 years had a preoperative speech reception threshold of 97 db with bone conduction of 30 db at 500 c/s only In January 1959 a three-quarter solid footplate was removed and a P E strut and vein graft placed Postoperatively the pure tone and speech reception thresholds varied from test to test but finally stabilized at about 70 db Six months later in June 1959 the round window was opened Considerable bone had to be removed It is interesting that in the first hours after surgery the patient's hearing was subjectively improved and her tinnitus subsided

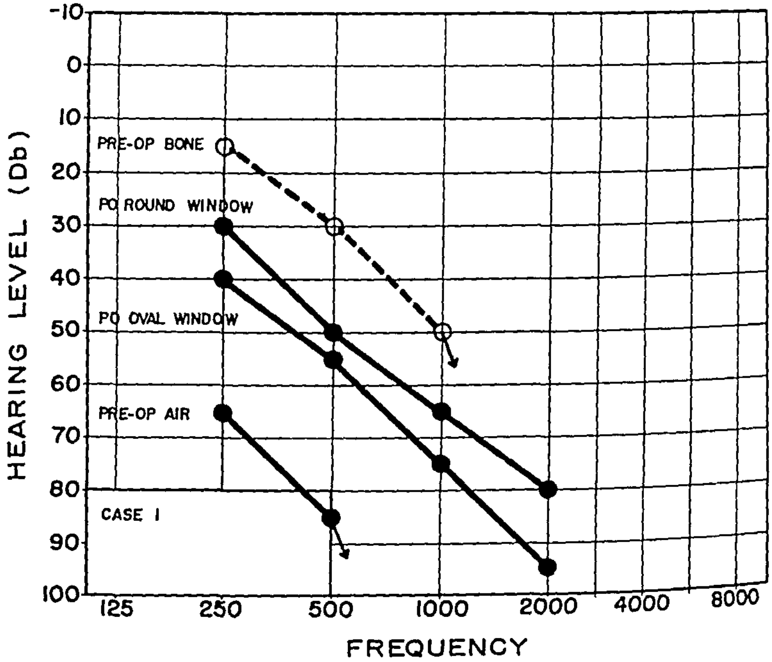
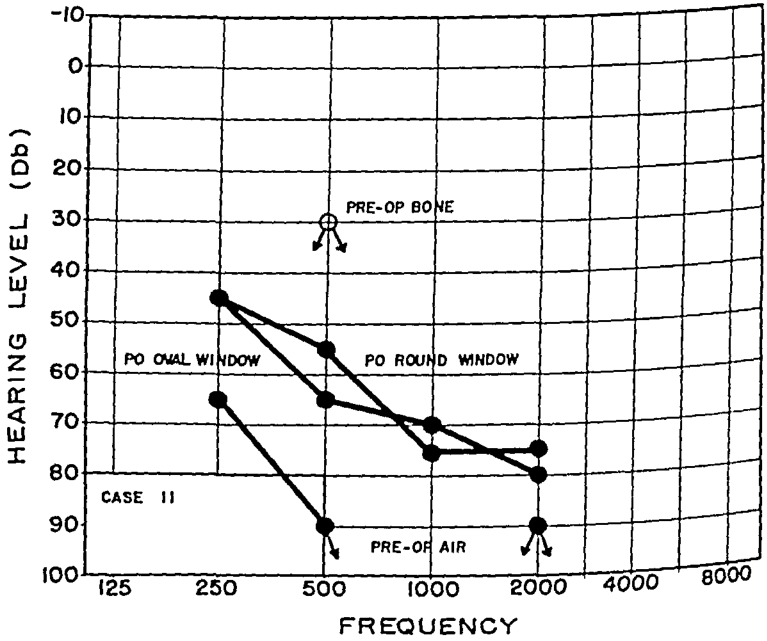

HEARING LEVEL (Db)
-10
0
10
20
30
40
50
60
70
80
90
100
PRE-OP BONE
PO ROUND WINDOW
PO OVAL WINDOW
PRE-OP AIR
CASE I
125
250
500
1000
2000
4000
8000
FREQUENCY

Fig 1


HEARING LEVEL (Db)
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PRE-OP BONE
PO OVAL WINDOW
PO ROUND WINDOW
CASE II
PRE-OP AIR
125
250
500
1000
2000
4000
8000
FREQUENCY

Fig 2

B C frequency at 500 c/s has remained the same but all response to air conduction has disappeared

Case 5 The patient is a 52-year-old carpenter He has had hearing loss for 40 years He had no response by air or bone conduction in the left ear In June, 1959, a solid footplate was removed with a drill Periosteum taken from the ear canal was placed over the oval window and a P E strut placed One month postoperative, he had a speech reception threshold of 90 db but 0 discrimination He now also had one response by B C of 50 db at 500 c/s In July, 1959, the round window was opened Fibrous tissue tract led from the tympanic mucosa to the former round window Perilymph with good transmitted movement from the oval window was noted The mucosa of the promontory was placed over the opening Postoperatively the patient has again lost all responses by A C and B C

COMMENT

In the above five patients there was definite improvement in hearing threshold after opening the oval window We have noted in this group and in other deaf patients on whom oval window surgery has been performed that it usually takes up to six months for the patient to transfer his aid from his formerly better ear to the operated ear

At first there is often a very narrow loudness tolerance (usually only 10 or 15 db between threshold and pain) This gradually improves so that the patient can wear an aid with comfort The reason for this phenomenon is unknown Usually, if he is wearing an aid on the unoperated ear, he finds it too loud and, therefore, unbearable to wear in the operated ear

Shortly after surgery the discrimination score is often worse than preoperative This, too, improves with time During the first postoperative months we usually advise these patients to have a set of earphones with an adjustable volume attached to the television When the patient finds through practice with his ear phones that his hearing is stabilized, his tolerance increased, and his discrimination "re-learned" in the operated ear a hearing aid evaluation is done to determine the aid that is now best suited for him

The results of these cases has led us to the conclusion that opening the oval window in conductive or mixed loss is potentially a worthwhile procedure regardless of the level of the bone conduction or air conduction The limiting factors in

By the third day, however, the hearing had returned to preoperative level and has remained here since

Case 3 (See Fig 3) The patient is a 32 year-old oil worker with a hearing loss of 12 years' duration Preoperatively in the left ear, he had an S R T of 68 with a discrimination score of 74 per cent B C was 45 db for 500 and 1000 c/s In January, 1959, a partially blue footplate was fragmented and a P E strut placed from the incus to footplate Three weeks postoperative the S R T was 48 db, but the discrimination score had dropped to 54 per cent There was no change in B C

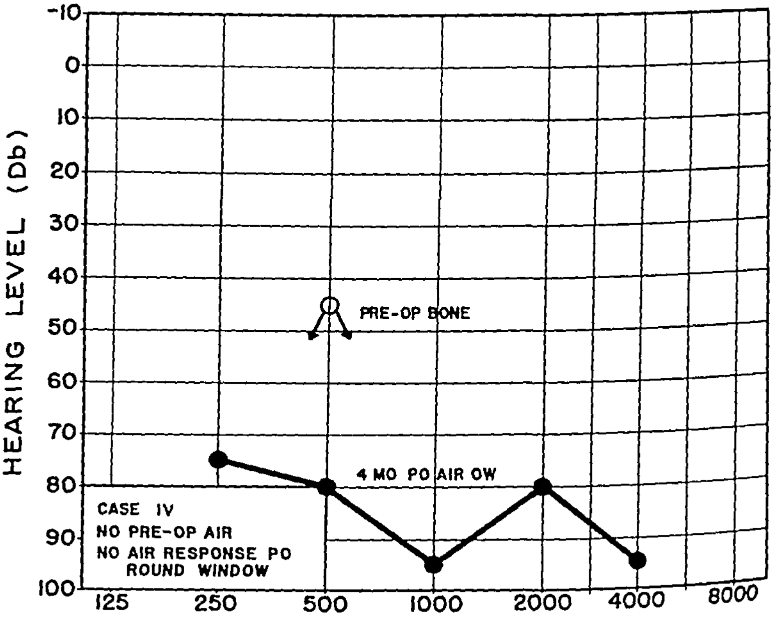

Fig 4

In February 1959, the round window was opened It was not firmly fixed but definitely had a calcified membrane Following the procedure the B C has remained the same, but the threshold has dropped to 85 db where it has remained

Case 4 (See Fig 4) The patient is a 57 year old furniture finisher He had a hearing loss of 15 years' duration Hearing test in the right ear gave no A C response and B C at 45 db for 500 c/s only In March, 1959, a completely solid footplate was thinned, fractured free from the oval window margin and depressed into the vestibule This was covered with gelfoam and a P E strut placed The hearing improved to a 55 db level by A C, but the B C remained unchanged The patient however continued to have an 0 discrimination score

Four months later in July of 1959 the round window was opened It was completely solid and considerable bone was removed along the basal coil before perilymph escape was noted Pulsation pressure from the oval window was well transmitted to this opening Postoperatively the

Apparently fixation of the oval window can be blamed for the amount of bone conduction loss commonly known as the Carhart notch[16] Closure of the round window accounts for possibly only 10 db of bone conduction loss These two figures then clearly represent the hydrodynamic changes in the cochlea caused by window closure, that can be measured by bone conduction audiometry.

The still completely unanswered question then is Why do patients with otosclerosis develop the sensory-neural loss of mixed and perceptive otosclerosis? Bone conduction measurement is the present method of evaluating this loss This is inadequate, because it does not separate the hearing loss due to hydrodynamic factors of otosclerosis, and it does not delineate the site of the sensory-neural lesion

There is much to be done, we have only begun the conquest of otosclerosis

CONCLUSIONS

1 At present there seems to be no maximum bone conduction thresholds to contraindicate oval window surgery in otosclerosis

2 The minimum air-bone gap that is practical to attempt to improve by oval window surgery has not yet been established This seems to be the prinicpal limitation on the indications for oval window surgery

3 Otosclerotic patients with stapes fixation and hearing loss beyond the limits of present audiometric measurements can be improved by oval window surgery, providing an unmeasurable air-bone gap is present Diagnostic procedures for these patients should be improved

4 Patients with extensive otosclerosis often have closure of the round window Opening this window with a drill through a transcanal approach has not proved beneficial and in three cases it has been detrimental We are, therefore abandoning round window surgery until more precise controlled methods can be developed selection of cases seems to be only the width of the air-bone gap, hearing level of the other (better) ear, age, and general condition of the patient

What about surgery on the round window? This is a potentially dangerous procedure since in three out of five of our cases the hearing was made worse The reason for this is obscure, but probably the scala media was surgically invaded since only obscure surgical landmarks are present

Previous case reports, animal work, and the first two cases reported in this study all indicate that the round window has little to do with hearing threshold, therefore, apparently the patient has little to gain and the chance of damage is great by surgery on this window For these reasons we are abandoning further surgery on the round window or scala tympani until better diagnostic methods and more precise surgical techniques are available

Several facets of otosclerosis have become increasingly apparent from clinical, radiological, and surgical study of otosclerosis

First, the reason for a conductive hearing loss in otosclerosis is stapes fixation in the oval window In general if the fixation is ligament in type (that is, without an actual bony bridge between the footplate and the oval window, but merely a thickening of the membrane) the amount of the air-bone gap tends to be less If there is bony fixation, the gap tends to be 30 db or more Once the annular ligament is bridged the audiogram does not tend to reflect the *amount* of the otosclerosis in the footplate or oval window

Second, fenestration surgery, or *complete release* of stapes ankylosis often improves the bone conduction This was attributed to a change in cochlear hydrodynamics by Juers[1]

It seemed reasonable that total closure of both windows would completely immobilize the cochlear fluids and, therefore, lead to total hearing loss By opening both of these windows it was hoped that dramatic hearing improvements could be obtained, this has not proven to be the case

VERTIGINOUS EPILEPSY*†

BERNARD J ALPERS, M D,
(By Invitation),

Philadelphia, Pa

That dizziness may accompany an epileptic seizure has long been known Whether it can in itself, without other features of epilepsy, constitute a seizure, is a much more difficult problem to solve Interest has recently been aroused in the problem by physiological stimulation of the cerebral cortex and by the occurrence of seemingly pure vertiginous seizures It seems advisable to attempt to evaluate the state of our present knowledge concerning the problem, though it is meager and inconclusive in many important respects

NOMENCLATURE AND CLASSIFICATION

The seizures in question have been referred to as "epileptic vertigo," "vestibular epilepsy," "vestibular seizures," and "vestibulogenic seizures" The term vertiginous epilepsy seems preferable, since it is non-committal regarding the origin of the seizures, and it refers to their outstanding characteristics

Vertiginous seizures may occur as an aura, preceding a seizure, or they may constitute the seizure itself Dizziness as an aura constitutes no diagnostic problem, its interest is largely physiological, as an indicator of the possible source of the cortical discharge When it occurs as an aura, it is followed by the typical features of an epileptic seizure which indicate its true significance It may occur in petit mal, grand mal or psychomotor seizures In his extensive study of auras, Gowers[1] found it to be present in 25 of 155 cases of

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*Read at the meeting of the Eastern Section of the American Laryngological Rhinological and Otological Society Inc Philadelphia Pa Jan 8 1960

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SIXTH INTERNATIONAL CONGRESS ON DISEASES OF THE CHEST

The Sixth International Congress on Diseases of the Chest will be held at the University of Vienna from August 29 to September 1, 1960

per cent and Hgb 14 9 Gm The leucocytes numbered 6 000, with a differential of neutrophils 30 per cent, lymphocytes 50 per cent, atypical lymphocytes 13 per cent, monocytes 47 per cent, eosinophiles 2 per cent and basophils 1 per cent The spinal fluid examination was negative Pneumogram revealed no evidence of tumor The only significant findings were revealed by the EEG, which showed evidence of general paroxysmal cerebral dysrhythmia with right temporal lobe discharges in the sleep record

CLINICAL FEATURES OF VERTIGINOUS EPILEPSY

That vertigo may appear frequently as an aura, to be succeeded by one of a variety of seizures, is well-known That it may occur, however, in pure or relatively pure form, as the only feature of a seizure, is not sufficiently recognized, and for this reason it may be helpful to review its clinical characteristics

The *incidence* of vertiginous epilepsy is probably rare when one considers the great variety of seizures and the many forms of epilepsy, both idiopathic and symptomatic There appear to be no reliable statistics of its occurrence

A group of 14 patients with epileptic vertigo has been reported by Pedersen and Jepsen,[2] but I have not included all of these in my discussion because in only six was there impairment of consciousness, while two had slightly impaired hearing Eight had abnormal EEG patterns Probably only those with impairment of consciousness should be included

The *clinical features* of this type of epilepsy are distinctive, but not in themselves diagnostic of the condition The central characteristic appears to be recurrent attacks of dizziness associated at time with lapses of consciousness, which, however, is not always present In its absence there may be complete amnesia for the episode, or there may be no impairment or loss of consciousness of which the patient is aware The lapse of consciousness covers only seconds, and is quickly over There is no fall, objects are not dropped during the episode, and no other associated symptoms are usually present The onset is sudden, without warning of any type The vertigo itself varies in degree, but in quality it is always associated with a sense of movement of the patient or his surroundings It may be typically paroxysmal in type Nau-

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In addition to the nature of the vertigo, impairment or loss of consciousness, or amnesia for the duration of the attack is an essential characteristic of the seizures. As in the case of a patient with petit mal, the patient with vertiginous seizures has a momentary lapse of consciousness. He may or may not be aware of this, but he may be seen to look blank or pale for the short duration of the attack. Where there are true vertiginous seizures, persistent questioning will reveal impairment of consciousness, though this may be denied by the patient.

The physical and neurological examinations in patients with the characteristics noted, are negative. This is true even if the vertiginous seizures are associated with brain disease, since the lesion responsible for the condition is so small that nothing is to be found neurologically.

The EEG may be normal, or there may be evidence of discharge from one or both temporal lobes in sleep EEG These may also be evidence of generalized paroxysmal cerebral dysrhythmia In any event, the EEG findings are important, for without them the diagnosis becomes quite difficult

Cortical Localization of Vertiginous Epilepsy The localization of the lesion associated with vertiginous seizures is not specific, but what evidence is at hand in human cases indicates that the process is in the temporal or temporo-parietal region The lesion has been found to be in the posterior half of the superior temporal gyrus, or at the parieto-temporal junction (Penfield and Kristiansen[3]) Electrical stimulation of these areas produced vertiginous experiences similar to those of spontaneous seizures (Penfield and Rasmussen[4], Penfield and Jasper[5], Penfield[6]) Penfield[6] reports that in seven of 108 patients who had exploration of the temporal lobe typical labyrinthine sensations were produced on cortical stimulation The area from which this sensation could be produced was in the posterior part of the first temporal convolution, adjacent to the transverse gyrus of Heschl In other instances the lesion responsible for the seizures has been found in the middle rather than in the posterior part of the temporal lobe

DIAGNOSIS OF VERTIGINOUS EPILEPSY

The diagnosis of a vertiginous aura offers no difficulties, nor is there any trouble in recognizing a vertiginous seizure when it occurs in conjunction with other types of seizures, both petit mal and grand mal and even psychomotor, there are great difficulties, however, in establishing the diagnosis in cases in which the vertiginous seizures occur in pure culture The problem becomes one of true vertiginous attacks, usually of brief duration, indistinguishable from vertigo due to other causes by the nature of the symptom itself Two features provide significant diagnostic clues to the true nature of the condition These are the absence of after-discharge, characterized by the fact that the vertigo is over with immediately, without evidence of gradually subsiding dizziness or other systemic symptoms The second feature consists of impairment or loss of consciousness or momentary amnesia associated with the seizure Nothing is to be found on physical neurological examination, but the very nature of the attacks indicates a small focal lesion for which a cause may or may not be found in the past history—evidence of head injury The EEG will show generalized or focal activity, or both, and the diagnosis will be heavily supported by positive EEG findings As in other forms of epilepsy, however, the EEG may be normal, but the diagnosis can be established if all other features of the condition are correct

Vertiginous epilepsy offers difficulties in recognition, but it is a possible clinical diagnosis, and it should probably be recognized more frequently The essential points in diagnosis consist of

1 The presence of vertigo This is true dizziness, and it differs in no way from dizziness due to other causes It may even be paroxysmal and thus confused with a peripheral type of vertigo

2 Impairment or loss of consciousness, or amnesia for the attack Without this it is difficult to see how the vertigo can be separated from an ordinary attack of dizziness It must be differentiated from syncope, but if this is done, and it can be determined that a seizure has been experienced, one of the most important steps in diagnosis has been established

3 The absence of auditory symptoms Tinnitus or other ear noises may be present, often as auras, but loss or impairment of hearing should be looked upon with caution, because of the bilateral representation of hearing and the great difficulty in determining hearing deficit of central origin

4 EEG changes While these are not always positive in the interseizure record of any form of seizure, the diagnosis in this and vertiginous type of seizure should not be made at the present time without support from the EEG, either in the form of focal or diffuse evidence of seizure activity

It is difficult to be sure, but it is possible that vertiginous seizures may be more common than is at present recognized

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POSTGRADUATE COURSE IN OTOLARYNGOLOGY.

Colby College, Waterville, Maine

The Department of Adult Education at Colby College is offering a short postgraduate course in otolaryngology on August 3, 4, and 5, 1960. This course is designed to review fundamental principles and present recent advances in this specialty through lectures, demonstrations and panel discussions by guest physicians of national prominence The broad content of the course should make it equally attractive to the practicing otolaryngologist and the recent graduate of an approved residency program

It will be conducted along the general line of the courses in Occupational Hearing Loss which have been given at the college for the past eight years The program will be under the general direction of Frederick Thayer Hill, M D, Medical Director of the Thayer Hospital, Waterville, Maine

The fee for the course is $75 and includes tuition, board and room Requests for further information should be made to William A Macomber, Director of Adult Education, Colby College, Waterville, Maine

ANTIMICROBIALS, TONSIL AND ADENOID BACTERIOLOGY, AND BLOOD CULTURE FINDINGS BEFORE AND AFTER TONSILLECTOMY AND ADENOIDECTMY[*]

PETER N PASTORE, M D ,[†]
and
MILES E HENCH, Ph D ,[‡]
(By Invitation),

Richmond, Va

In a recent publication[1] it was noted that chemotherapy and antibiotics apparently have not eliminated or reduced the need for tonsillectomy and adenoidectomy below the age of seven years The author called attention to the study by Rhoads, Sibley and Billings[2] who stated (p 880), "Our experience and that of others is that the elimination of the beta hemolytic streptococcus carrier state by antibiotic therapy often is only temporary When the pretreatment flora of the throat are restored, the micro-organisms that had been temporarily suppressed are often found to have gained resistance to the antibiotic that had been used in treatment Hence, the hazard of prolonged antibiotic therapy not followed by extirpation of the tonsils may be serious, even if the micro-organisms that were the original objects of the anti-bacterial attack are temporarily eliminated" The conclusions of their study, "Bacteremia Following Tonsillectomy" (review of pertinent reference literature, p 879), summarized by them, follows

> Blood cultures taken just after tonsillectomy were positive in 28 3 per cent of a group of 68 patients who received no antibiotic therapy prior to tonsillectomy The incidence of bacteremia was reduced to 5 9 per cent in a group of 20 subjects who received penicillin in a daily dose of

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[*] Read at the meeting of the Southern Section of the American Laryngological Rhinological and Otological Society Inc New Orleans, La Jan 16 1960

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> 600,000 to 800,000 units intramuscularly for four to ten days prior to tonsillectomy The incidence of bacteremia in a group of 29 patients who received 600,000 to 800,000 unit doses (half this dose for children) of procaine penicillin 12 to 18 hours and one hour prior to tonsillectomy or in a small group (seven) of patients receiving 900,000 to 1,200,000 units orally daily for five to seven days prior to operation was not reduced below that of the control group Beta hemolytic streptococci were obtained from blood cultures four times, pneumococci once, alpha hemolytic streptococci (green forming) or Gamma anhemolytic streptococci 28 times, and a combination of Beta hemolytic streptococci and Gamma anhemolytic streptococci twice The need for several days preoperative treatment with penicillin to prevent post tonsillectomy bacteremia is obvious
>
> In the control series of 68 patients who had no antibiotic treatment immediately preceding tonsillectomy, Beta hemolytic streptococci were present in 57 4 per cent of the cultures of the excised tonsils, although these micro organisms were found in only 28 26 per cent of throat cultures taken just before the operation Among patients receiving penicillin in single doses only the day before and the day of tonsillectomy, 31 03 per cent had Beta hemolytic streptococci in the excised tonsils, although these micro organisms were not present in the throat cultures taken just before tonsillectomy Beta hemolytic streptococci were found only once in the cultures from the excised tonsils of persons receiving penicillin intramuscularly each day for four to ten days prior to tonsillectomy Most of the Gram positive micro organisms except micrococci and Gaffkya tetragenus were greatly reduced in number by penicillin administered intramuscularly, but gram negative micro organisms, such as Klebsiella pneumoniae, Aerobacter aerogenes, and Escherichia coli, were found in increased numbers in cultures from the throats and excised tonsils of these subjects

As a result of their report, several questions of clinical interest were stimulated If bacteremia occurs frequently (28 3 per cent Rhoads) post-tonsillectomy without preoperative antimicrobial therapy, *a* how can it be recognized by clinical manifestations, and *b* should prophylactic anti-microbial therapy be administered routinely pre- and post-tonsillectomy to reduce such a high incidence of bacteremia?

The circumstances of a total physician-referred practice to a large medical center offered an excellent opportunity for the study of such questions This report is the result of tonsil and adenoid tissue bacteriology and blood culture observations on a consecutive series of 140 patients undergoing tonsillectomy and adenoidectomy by operator A during the period October, 1958, through June, 1959

In both the study and control groups, all of the patients came from within a radius of 150 miles of Richmond, Va These were all considered of the medical therapy resistant type Many of them had reached the stage where the attacks

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In both the study and control groups, all of the patients came from within a radius of 150 miles of Richmond, Va These were all considered of the medical therapy resistant type Many of them had reached the stage where the attacks of tonsillitis and adenoiditis were closely related, sometimes occurring with less than a week of freedom between acute attacks Some remained in the subacute state, requiring continued administration of therapeutic doses of antimicrobials until the operation could be performed

In the control group, cases were selected solely on the basis that the history revealed they had received no antibiotics or chemotherapy for a minimum of two weeks or longer before operation

The majority of patients in both the study and control group gave a history of five or more attacks of clinically diagnosed tonsillitis and adenoiditis In one patient, age six years, the history revealed only one known attack of recognized tonsillitis at age three years before the second attack at age five and one-half years which became associated with acute nephritis The largest number of physician-recorded attacks in a child was 32 These occurred from age six weeks to 35 months when the operation was performed

TABLE I

Age Incidence

The patients' ages were distributed as follows

Age in Years	Study Group	Control Group
1 to 3 years	10	2
3 to 6 years	39	11
6 to 9 years	39	19
9 to 12 years	12	8
Total	100	40

The slightly higher age incidence in the control group six to 12 years is possibly explained on the basis of fewer or less severe attacks in the period from one to six years

INDICATION FOR THE OPERATION

A review of Table II shows that primary tonsil or adenoid disease leads the list as the dominant indication for the operation In this group are included those with systemic complications remote from the upper respiratory tract, such as kidney disease In most of these (73), the history of sore throat involving the tonsils preceded other manifestations of systemic disease or complications

DURATIONS OF ADMINISTRATION OF PREOPERATIVE ANTIMICROBIALS

The administration of the antimicrobials (see Table III) was directed or supervised by the referring physician, according to the indications In each instance it was felt by the physician in charge that the patient needed therapy to control or to reduce clinical evidence of active inflammation to a minimum so as to permit the patient to tolerate the operation more safely

TABLE II

	Study Group	Control Group
Tonsil adenoid, recurrent acute attacks	73	25
+ History of Otitis media, recurrent	17	6
+ History of Persistent Cough and Allergy	4	1
+ History of Repeated URT Infection	3	7
+ History of Impaired Hearing persistent	3	1
Total	100	40

TABLE III

Number of Patients Receiving Designated Antimicrobial Before Operation	
Chloramphenicol	4
Sulfonamides	7
Erythromycin	23
Penicillin	23
Tetracycline	34
Tetracycline + Novobiocin	9
Total	100

Eleven (see Table IV) of those who had been maintained on an antimicrobial for longer than seven days were patients who had been hospitalized for other conditions associated with acute tonsillitis and adenoiditis and had the operation performed during the hospital stay or recovery phase

GENERAL PLAN FOR THE STUDY

Study Group

In the study group of 100 patients, all received oral or intramuscular antibiotics preoperatively (see Tables III and IV) Blood was withdrawn aseptically from a suitable vein immediately before operation (patient fully anesthetized under general anesthesia) , the operation was performed, the tonsils and adenoids were placed in a sterile glass container and blood was again withdrawn while the patient was still asleep

Control Group

In the group of 40 patients as controls, preoperative antibiotics or chemotherapy had not been given for a minimum of two weeks to several months, otherwise, the technique was the same

TABLE IV

Preoperative Administration of Antimicrobials in Days

Days	1	2	3	4	5	6	7	Longer
Number	0	6	21	28	13	9	5	18
Total								100

Method

Bacteriological examination of the specimens was undertaken in the hospital diagnostic laboratories of the Medical College of Virginia Tonsil and adenoid tissues were received in sterile containers, within two hours of excision These tissues were macerated under sterile conditions, employing sand and a mortar and pestle Homogenized tissue was moistened with sterile physiological saline Tissue fluids and saline were then aspirated for employment as inoculum to trypticase-soy blood agar, eosin-methylene blue agar and thioglycollate broth, (B B L) for anaerobic and aerobic cultivation

Blood specimens were received pre-inoculated into the standard blood culture preparation of this laboratory These blood cultures are six-ounce prescription bottles with a perfoiable rubber closure, prepared as biphasic culture bottles containing both solid and liquid media. The liquid medium is 80 to 90 ml. of brain-heart infusion broth, with added para-amino benzoic acid, penicillinase and 0.1 per cent agar, (B.B.L., modified). The solid phase is provided by a slant of trypticase-soy agar, (B.B.L.). This preparation can be relied on to support growth of fastidious aerobic and anaerobic organisms, (*e.g.*, H. influenza, Bacteroides fundulifoimis, N. meningitidis, Cl. welchii, etc.). Blood cultures were examined daily until growth was observed, or discarded after two weeks' time.

TABLE V

Organisms Found in Tonsil and Adenoid Tissue Specimens

	Study Group	Control Group
Single Organisms		
Non hemolytic Staph.	4	0
Alpha Strep.	3	2
Hemolytic Staph. aureus	6	0
Beta Strep.	0	5
Combinations of Multiple Organisms		
Alpha Strep. + Neisseria	7	2
Alpha Strep. + hemolytic Staph. aureus	43	9
Alpha Strep. + hemolytic Staph. aureus + Beta Strep.	9	8
Alpha Strep. + Beta Strep.	5	0
Hemolytic Staph. aureus + Beta Strep.	14	3
Miscellaneous combinations	9	11
Total	100	40

(Organisms found in miscellaneous combinations: non-hemolytic staph., E. Coli, C. albicans, Pseudomonas, D. Pneumoniae, non-hemolytic strep., Vulgarius, Paracolon species.)

OBSERVATIONS

Tissues, as noted in Table V, produced predominantly gram-positive populations of organisms. Outstanding in frequency in these findings were staphylococci and Beta hemolytic streptococci as probable offending pathogens. In only ten instances in the study group and four instances in the control group, as can be seen in the Table, can the tissues have been considered free of pathogenic organisms (occurrence of alpha hemolytic streptococci singly or in combination only with Neisseria). Virus studies were not included.

The results obtained on blood culture study (see Table VI) were unexpected in that no positive cultures (excepting two contaminated) were obtained in the series. This finding is at variance with other reported studies[2] Interpretation of these results is speculative In transient bacteremia, a single sample of blood may not establish the condition Cultures taken in the immediate postoperative period, would be expected to reveal manipulation induced bacteremia if the organisms were circulating with any frequency The fact that these cultures were negative leads to the conclusion that there was no bacteremia of significant magnitude occurring in these patients This would suggest that the surgical removal of tonsils and adenoids, even in the presence of existing chronic infection, can be performed without exciting an active bacteremia of significant numbers of organisms It would also suggest that the administration of antimicrobials for the control of transient bacteremia before and after tonsil and adenoid operations should be on a selective basis or with indications and not as a routine procedure

TABLE VI

Results of Blood Culture Studies

	Number	Positive
Study Group		
Received antimicrobials preoperative—		
Blood culture before operation	100	0
Blood culture after operation also	35	0
Control Group		
Received no antimicrobial preoperative—		
Blood culture before operation	40	0
Blood culture after operation also	25	1 diphtheroid 1 mold

In an effort to make our study as complete as possible, we sent a copy of our results to Dr Paul S Rhoads[3] whose gracious reply follows

> In regard to your letter and interesting paper, which arrived yesterday, I can not explain the discrepancy between your results and the ones we obtained I think our technique was proper We have no one else's series to compare our tonsil work with, but when we did similar work with tooth roots snipped off after extraction of the teeth, our percentage of positive blood cultures was right in the middle of the range of positive blood cultures reported by other workers who had done similar studies

Our largest number of positive blood cultures was in the ones in which we used an anaerobic method[4] described in our paper and one which you did not use This was to put the melted agar containing the patient's blood into a sterile Petrie dish then to let it cool after which a layer of plain agar was put on top After this cooled, a layer of melted paraffin was put over the agar containing blood Then when colonies appeared in the agar, it was possible to take off a small piece of paraffin and go in and get the colony with a small sterile glass pipette and bring it out for identification

As to the fairly large numbers of Beta hemolytic streptococci found in your "study group" my guess is that these were not eliminated because the patients did not have the same routine which we used in our study For instance you say 23 patients received Penicillin but do not say whether it was given intramuscularly or orally and for how many days We soon learned that one could not count on the administration of any antibiotic for several days unless he administered it himself In the patients who received Penicillin intramuscularly preoperatively, in practically every instance we injected the material ourselves so we know those patients received the antibiotic As our paper indicated, antibiotics given orally and for only short intervals did not uniformly have the effect of eliminating Beta hemolytic streptococci You will also note that we used a quantitative method in our cultures of the tonsils and therefore shifts in the bacterial population of the tonsils which we described were much more meaningful (see the Table in both of our reprints)

Also I note in your figures on page 9 (Table VI) that you had a relatively small series of patients who had blood cultures postoperatively It is possible that had your series been as large as the series of blood cultures before operation you would have picked up more positive blood cultures In our report we were not trying to make the point that every patient who is to have a tonsillectomy should necessarily have five days of Penicillin administration by injection Transient bacteremia seldom is a serious threat Present views are that unless there are previously damaged heart valves subacute bacterial endocarditis is not apt to be set up when the bacteremia is of very transient duration and the valves are normal However I do think the precaution we suggested is important in patients who have had previous valvular damage We tried to point out in subsequent papers that there might be an even higher incidence of potentially dangerous Staphylococci in infected tonsils after the prolonged administration of antibiotics to which these organisms were resistant and we did point out that other organisms such as E coli Diplococcus pneumoniae, and so forth, were very apt to be on the increase in such persons Thus I do not think too much therapeutic implication should be read into our results We were giving them for what they were worth without making too many recommendations as to what doctors should do about it I do believe that our blood cultures are correct however

SUMMARY

The administration of various antimicrobials to patients with recurrent or persistent attacks of tonsillitis and adenoiditis does not eliminate the pathogenic organism in the tonsils or adenoids in most instances (90 per cent) The fact that our study did not show any demonstrable evidence of bacteremia in blood cultures should in no way minimize the importance of such a possibility in the event of clinical symptoms of bacteremia before or after tonsillo-adenoidectomy

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GILL MEMORIAL EYE, EAR AND THROAT HOSPITAL

The Gill Memorial Eye, Ear and Throat Hospital has just completed its Thirty-Third Annual Spring Congress in Ophthalmology and Otolaryngology and allied specialties The attendance was one of the largest in the history of the school with an attendance of 300 doctors and their wives There were 40 states, including Canada, represented There was a total of 18 guest speakers and there were 50 lectures during the five-and-a-half days of the Spring Congress In 1961, the Thirty-Fourth Annual Spring Congress will be held from April 9 through April 14

MANAGEMENT OF MAXILLO-FACIAL FRACTURES*

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The diagnosis and treatment of facial fractures form an integral part of the practice of many otolaryngologists The literature reveals varied methods of management. Some are simple, some are complex, but each endeavors to restore the contour and immobilize the fragments Most yield good results as exhibited by their respective proponents

A review of the anatomy of this region is important to re-acquaint us with some of the problems that are encountered and to understand better the principles and methods used to correct them

The skull is divided into the upper or cranial region, the middle or zygomatico, maxillary and nasal region, and lower or mandibular region

This paper will concern itself chiefly with the fractures of middle third and more especially the lateral portion (Nasal fractures will not be considered here) To gain a better perspective of the problem, a brief survey of the kinds and peculiarities of the fractures of the entire middle third will be discussed This will provide a better foundation for the further discussion of the fractures of zygomatic and maxillary bones

The orbital rim is formed above by the frontal bone, and laterally by the zygoma The zygoma and the maxilla form the inferior margin, the maxilla then curves with the frontal to form the medial portion At the inferior rim, both the zygoma and the maxilla continue inward as a thin plate of bone to help form the floor of the orbit or the roof of the maxillary sinus Viewed from below, the zygoma has an

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outward projection which, with the strong abutments of the arch, produces the most prominent feature of the cheek, the cheek bone This arch is formed by the buttress of temporal bone posteriorly and of the maxilla anteriorly Beneath this arch lies the converging fibers of the temporalis muscle The prominence of the zygoma blends and recedes into the anterior wall of the maxilla giving the face its bone for the cheek

CLASSIFICATION

A Fractures, Lateral Region A fracture involving the zygomatic bone and zygomatic arch without interference of mandibular movement, or a fracture of zygomatic bone with minimal displacement is classified as a first degree fracture When, in addition to the above, the lateral wall of the maxillary antrum is involved with alteration of facial contour and interference with mandibular movement, this becomes, then, a second degree fracture When, in addition to the above second degree, there occurs a comminution of orbital floor and/or gross separation of zygomatic-frontal suture and depression of orbital floor, this is considered a third degree fracture

B Fractures, Central Region

1 The simple alveolar fracture, it is usually bilateral There is avulsion of part of the bone

2 The low level subzygomatic fracture (Lefort I or Guerin)

Lefort I The low level subzygomatic involves the lower anterior wall or walls of the maxilla just below the zygoma The fracture line commences at point on the lateral margin of the anterior nasal aperture at the pyriform sinus, it passes laterally above the canine fossa and then inclines upward to the pterygo maxillary fissure to fracture the pterygoid lamina, likewise, it also passes from the anterior nasal aperture along the lateral wall of nose to join the fracture line behind This fracture may be unilateral or bilateral

3 Pyramindal subzygomatic fracture (Lefort II)

Lefort II has greater involvement than Lefort I The fracture line passes from upper end of the nasal bones at the frontal suture line down to across the frontal process of maxilla, down the lacrimal bone and laterally and downward over the inferior orbital margin at region of the zygomamaxillary suture and then laterally near the zygomatic buttress and upward to beyond the inferior orbital fissure This is higher than in Lefort I

C Combined Central and Lateral Fractures

a High level supra-zygomatic fracture (Lefort III)

Lefort III This is supra-zygomatic fracture It begins at a line above the nasal frontal suture and passes to the frontal processes of superior maxilla, it passes through upper limit of the lacrimal bone and then crosses the orbital plate of the ethmoid to down below the optic foramen and laterally to reach the inferior orbital fissure From here it passes from the lateral wall of orbit to reach the junction of zygomatic bone with greater wing of the sphenoid

b The above with addition of a midline split separating maxilla into two

c a and b with fracture of roof of orbit or frontal bone

From both a practical and clinical point of view it is obviously impossible to classify in every instance a fracture of the middle third of the skull in a certain specific category, however, a familiarity with each group does help to give a working knowledge of the pattern a fracture may assume

Applied Surgical Anatomy The immediate under side (middle third) of the cranium (upper third) is the architectual problem that is necessary to be understood

The maxilla, zygoma, nasal cavity, orbits, sphenoid bone, and palatine bones are the integral parts of the middle third of the skull

Maxilla By the arching of the palate and the abutments of the maxilla against the frontal, the zygomatic and ethmoidal sutures, any given force is greatly resisted and better distributed A further support is given by the sphenoid bone and its greater wing The zygomatic bone redistributes the load mostly to the frontal bone and some to the temporal bone The orbits and nasal cavity are contained within the framework in the middle part of the skull They diminish the area available for basal bone structure The sphenoid bone is the keystone of the skull and is situated between the facial skeleton and the cranial base

GENERAL CONSIDERATION OF NATURE OF INJURY

The nature of the structure of the facial skeleton permits it to resist much force from below A blow to the anterior, superior, or lateral aspects will tend to shear off the whole complex from the cranial base

With any maxillary bone injury there are three important associated injuries that should be considered and evaluated

1 A gross disruption of the maxilla will displace the elements of the nasal septum and nasal bones to expose the paranasal sinuses to infection The nasal respiratory tube may be obstructed by blood clot or displaced cartilage and bone

2 A gross disruption of the maxilla with severe comminution will not only affect the inner canthal ligaments of the eye, but will also involve the orbital floor which in turn affects the support of the eye Diplopia will be present immediately, and, possibly later, entropion

3 A gross disruption of the ethmoidal labyrinth will involve the cribiform plate and give direct access to the anterior fossa of the skull

With any zygomatic bone injury there are important associated injuries that should likewise be considered and evaluated

With slight disruption of the zygoma the protective function of the orbital rim is impaired, and the natural prominence of the cheek is reduced

With gross disruption of the zygoma with severe downward displacement, there is much greater loss of the rim of orbit and the cheek bone is now flattened In addition, the infra-orbital nerve may be crushed or torn with resultant anesthesia If still greater disruption of the zygoma occurs the external canthal ligament is displaced, the check ligaments are loosened, the extraocular muscles do not function properly, and the ocular level is lowered

Acute fractures of the facial bones are not, as a rule surgical emergencies however, if asphyxia shock or hemorrhage are present active and immediate intervention becomes necessary before any attempt at reduction and immobilization of the fracture can be done If these three exceptions do not occur, definitive treatment should be instituted as soon as possible when the general condition permits On the other hand, over enthusiastic and too early surgical intervention may add to the problem and may even cause death Good surgical judgment, then, is imperative

Management may consist of simple reduction with no immobilization and trusting that normal alignment and contour will be preserved This may suffice if no further force or violence is encountered by the patient, however, many fractures are so comminuted and depressed that some support either external or internal or both must be applied if immobilization is to be maintained

When comminution and depression do exist the normal cavity of the maxillary sinus is altered *i e* decreased in size and sequestered bone may protrude into it It is always desirable when the maxillary sinus is severely injured to use a Caldwell-Luc approach to inspect the sinus cavity for loose bone fragments Where possible, those fragments should be replaced in their normal relationship and position If the infraorbital rim is so shattered or disrupted, not only will it become necessary to use external skeletal fixation, but also an intra-antral packing may be essential to restore and maintain it

One or two wires passing underneath the zygomatic arch may be required to hold out the fractured pieces The zygoma may be so fractured that simple elevation is insufficient and tributed A further support is given by the sphenoid bone and its greater wing The zygomatic bone redistributes the load mostly to the frontal bone and some to the temporal bone The orbits and nasal cavity are contained within the framework in the middle part of the skull They diminish the area available for basal bone structure The sphenoid bone is the keystone of the skull and is situated between the facial skeleton and the cranial base

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A careful clinical examination, as well as X-ray examination then is most important before any decision should be made as to the best method to employ As a general rule, the fracture is more extensive than the X-ray picture usually indicates

As stated above, there are numerous methods which the surgeon may employ to reduce and immobilize the fracture I believe that the best method is that one which applies to that patient and his especial variety of fractures

The following methods from the simple to the more complex are enumerated

1 Simple elevation by hook or towel clamp, especially for maxilla

2 Elevation by incision and fulcrum action elevator, especially for zygoma

3 Open reduction and forceful elevation, either for the maxilla or zygoma or both

4 Open reduction and elevation by one of the first three methods and transosseous wiring

5 Open reduction and elevation by one of the first three methods and circumferential internal skeletal fixation

6 Open reduction and elevation by one of the first three methods and fixation by internal wire pin stabilization and, possibly in addition, by external skeletal fixation

7 Open reduction and elevation by one of the first three methods and intra-antral packing or balloon, or by mechanical device

The more severely comminuted and extensive the fracture, the greater the necessity for fixation and stabilization One patient may require two or a combination of the procedures to accomplish a satisfactory result

It is quite evident that one may not designate a certain method as superior and applicable to all fractures Personally, I would not like to be limited to any one procedure It is true certain surgeons will achieve much success by using one method in preference to others where another will employ a second form of treatment with equal success

Several examples have been selected of case histories and X-rays of the patients who have had varying degrees and types of fractures You will probably note I have none of internal wire pin fixation I have used that procedure earlier, but I have used other methods to a greater extent and success

Case 1 This patient received a sharp blow with a small hammer directly to his infraorbital ridge X-ray revealed a slight fracture of the infraorbital ridge with depression which was evident on palpation Traction by using a towel clamp was employed to elevate the bones A hook could have been used No operative incision was necessary In a fracture of this type, no further care is needed if no later force is inflicted Healing in good position resulted

Case 2 This patient gave the history of being accidentally hit with pipe while at work The clinical and X-ray findings were similar to Case 1 as was the management

Case 3 This patient received a blow by a fist X-rays revealed a fracture of maxilla and zygoma with internal distortion and depression There was too much ecchymoses with contusions to depend only on towel clamp or hook for elevation By a Caldwell-Luc approach there was found to be minimal comminution present An elevator was introduced into the sinus cavity and with manual manipulation the fragments were replaced into normal position No further fixation was used and the fragments remained in normal position and healed satisfactorily

Case 4 The patient received a blow to the face by fist probably with brass knuckles There were marked abrasions and contusions of the face and edema of conjunctiva with subconjunctival hemorrhage X rays revealed a markedly displaced and fractured maxilla and zygoma By using a Caldwell Luc approach several small pieces of loose bone fragment within the antral cavity were removed The maxilla revealed further extensive comminution of the remainder of the bone An elevator was inserted into antrum and the bones elevated into position Because of this comminution of the anterior wall and floor of orbit a rubber glove finger filled with half-inch gauze impregnated with Amertan and covered with Amertan was inserted into the antrum cavity after an antrum window had been made Further packing through the antrum window opening was then done to make the intra antral packing more firm and tight The Caldwell Luc incision was closed X-ray revealed the fragments in good alignment. Healing in good position resulted The intra antral packing was removed in two weeks

Case 5 A six year-old child was struck on the cheek by an auto bumper Her findings and management were essentially that as of Case 4

Case 6 The patient was in an auto accident and received severe contusions of the face on the left side Further examination revealed diplopia marked flattening and depression of cheek bone crepitation and crunching on slight pressure. X rays revealed severely comminuted fracture of zygomaticomaxillary bones. With a Caldwell Luc exposure of antrum there was complete comminution and depression of the entire infraorbital ridge. The zygomatic prominence was easily movable. After some free and unidentifiable bone pieces were removed from sinus cavity, an elevator was inserted and lateral wall pushed outward and upward. Simultaneously, a tenaculum was fastened to the zygoma and it was gently pulled upward and laterally. After an antrum window was made, wires were placed around infraorbital ridge and under zygomatic arch. Later these were attached to support from a plaster of Paris head cap. The Amertan covered rubber finger filled with half inch gauze was inserted and further firmly packed into the antrum cavity with its open end extending out through a previously made antrum window. This was left in three weeks and wires on the headcap for five weeks. Final result was satisfactory.

Case 7. A patient received a severe head injury in an auto accident. There was marked contusion and edema with ecchymoses. Diplopia was present. Palpation revealed emphysema and a little pressure gave a crunching sensation. By a Caldwell Luc approach the antrum cavity was found to be almost completely obliterated and what remained was filled with blood clot and pieces of bone. The larger fragments were pushed outward as much as possible and those fragments that could be replaced were positioned. Wires were looped around the zygoma and infraorbital ridge. The antrum cavity was packed with rubber glove finger containing gauze with half inch packing impregnated with Amertan, the open end protruding through an antrum window opening. External traction was instituted by attaching with rubber bands the wires to the metal supports from a plaster of Paris head cap. Gradually, over a period of three days, the zygoma and maxillary bones were drawn into position as the swelling subsided. The intra antral packing was removed in three weeks, and the wires were removed in six weeks.

Case 8. Patient was in a fight and was hit and kicked in the face. The right side was severely contused and abraded; diplopia was present. Palpation revealed crunching of the bones on moderate pressure, neither a cheek bone nor an infraorbital rim could be felt in normal position. A Caldwell Luc exposure revealed an antrum filled with fragments of bone and blood clot. By a tenaculum attached to the zygoma on the outside and elevator placed within the antral cavity the bones were gently pushed outward and upward into position. Some bone fragments could be replaced and others had to be removed. Wires were placed around inferior orbital ridge and around zygomatic arch. An intrasinal rubber finger with packing was inserted, the wires were fastened to suspension rods from a plaster head cap. At the end of three weeks the intra-antral packing was removed through antrum window and wires removed in five weeks. Result was satisfactory.

Case 9. Patient was kicked in the face by a horse and suffered multiple fractures of zygomatic and maxillary bones. There was marked contusion, abrasions, ecchymoses, crepitation and crunching, especially of anterior portion of cheek bone and all of anterior wall of cheek. By a Caldwell Luc approach it was found that all of the anterior wall of maxilla and zygoma were shattered and comminuted. Before an intrasinal rubber finger filled with half inch packing was placed within the antrum, circumferential wires were placed from the infraorbital ridge and anterior wall of the antrum to the zygoma and lateral process of the frontal bone. These were left in place for four weeks. The final result was satisfactory.

Case 10. This patient fell from a scaffold and suffered fractures of the nose and both maxilla as well as an inwardly displaced alveolar arch Marked contusions abrasions ecchymoses and subconjunctival hemorrhages were present The nose was flattened A Jelenko dental arch was fastened to the upper teeth, the alveolar process with metal arch was drawn forward Circumferential wiring was suspended from zygoma to each end of metal arch and tightened just enough to immobilize bone fragments and the alveolar process

The nose was not corrected at this time due to marked swelling of all intranasal and paranasal tissues

The wires were left attached for six weeks when they as well as the metal arch were removed Healing in good position occurred

SUMMARY

A review of the injuries occurring in the outer portion of the middle third of the skull revealed that a variety of operative methods were used The correction and immobilization of the fractures of the zygomatico-maxillary complex depended upon the extent of the trauma

A practical and working knowledge of all the types of facial fractures of the middle third of the skull, coupled with familiarity with the various corrective procedures, is pertinent to insure good results

After a careful appraisal of the character and extent of the injury, the surgeon may select that method or combination of procedures which in his hands proves most efficient

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CORDABRASION TREATMENT OF BENIGN VOCAL CORD LESIONS*

A Preliminary Report

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INTRODUCTION

Benign lesions of the vocal folds are not uncommon[1] and the technique for the removal of these tumors is well established[2] There is, however, considerable evidence that the results have been something less than satisfactory in many instances due to recurrent or residual voice abnormality, consequently, a technique is needed which will provide *1* a biopsy, *2* complete excision, and *3* a smooth vocal fold postoperatively which would produce a pleasing voice Unfortunately, with the present-day method of stripping the vocal cord, or excising small cordal lesions, the vocal fold is often distorted, so that the end result is chronic hoarseness or dysphonia It was with this in mind that the "Cordabrasion" technique was developed

MATERIALS AND METHODS

With the experience gained in the dermabrasion procedures, an attempt was made on 16 patients with benign vocal cord lesions to perform a biopsy with the cup bite forceps followed by a cordabrasion using the technique described hereafter The lesions were variously diagnosed as polyps, cysts, papillomas, laryngeal nodules, dyskeratoses and hyperkeratoses, chronic laryngitis, hemangiomas, and fibromas All of the patients experienced a recovery of normal phonation, and the vocal cord regained an entirely normal appearance

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TECHNIQUE

Anesthesia is especially important in cordabrasion It must produce relaxation of the vocal cords and provide sufficient time for the procedure to be completed The patient is first given Pentothal Sodium® intravenously and hyperventilated with oxygen This oxygenation allows a safe apneic period of three to five minutes, during which time the surgery is performed While the mask is being removed and the laryngoscope introduced, Anectine® is administered intravenously in a single injection to achieve temporary paralysis of the larynx The laryngoscope is passed down into the laryngeal vestibule so as to be wedged at its distal end between the false cords and almost directly on top of the true cords Biopsy-excision of bulky lesions is then done with the Cup-Bite Angular Jaw Forceps

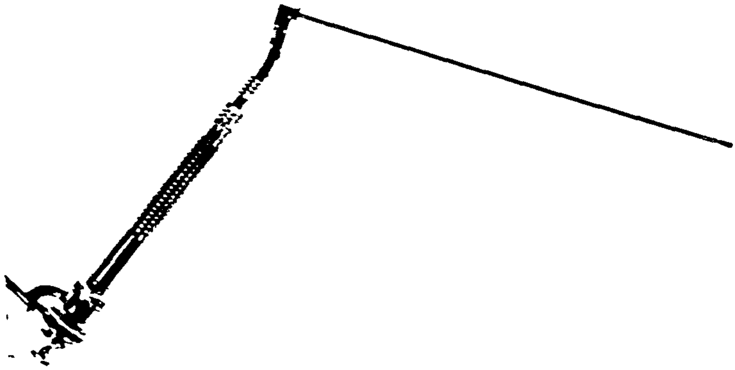

Fig 1 Specially-designed diamond burr attached to contra-angle handpiece

Thereafter, a specially-designed diamond burr* (see Fig 1), attached to the usual drill and handpiece used in temporal bone surgery, but with a contra-angle attachment to aid visualization through the endoscopic instrument (see Fig 2), is introduced into the glottis The abnormal portions of the vocal cords are abraded with this burr where indicated on the approximating and superior surfaces, smoothing out the biopsy defect at the same time Great care is taken to avoid the vocal processes and the anterior commissure, but both cords may be "sanded" at the same surgical procedure It was found to be more convenient to start posteriorly and produce the abrasion working forward or anteriorly

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*Manufactured by the O Rourke Diamond Company (Ordco) 11423 Vanowen North Hollywood Calif

In two patients, where the lesion was an extensive polyposis

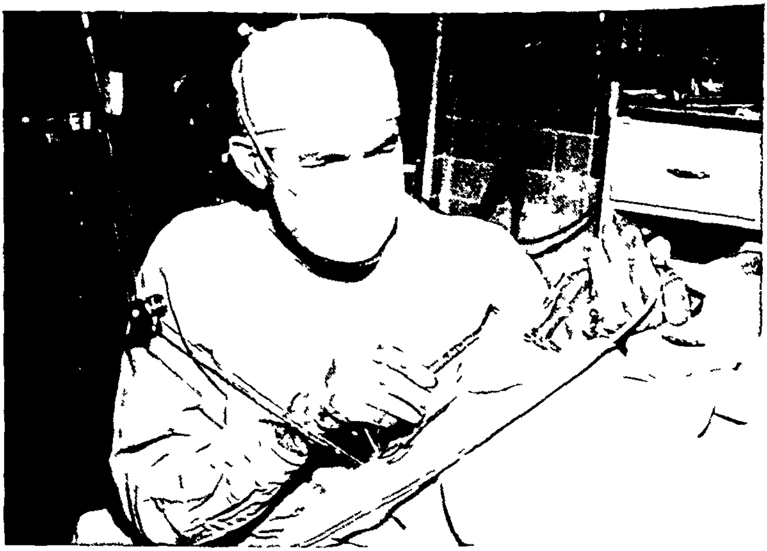

Fig 2 Cordabrasion through anterior-commissure laryngoscope under general anesthesia

of both cords requiring greater operating time, a small endotracheal tube was passed and left in the posterior commissure during the procedure This, surprisingly enough, did not interfere with the exposure of the cords and, furthermore, had the added advantage of holding the cords taut in order to facilitate the cordabrasion

Postoperatively, the patients were advised to not use their voices for two weeks, followed by a "whisper" period of another two weeks At the end of this month, phonation was only gradually permitted Harmful habits, such as smoking and vocal abuse, were discouraged from the beginning

RESULTS

The encouraging aspect of this preliminary report is that every one of the 16 patients regained a natural, clear voice, and the vocal folds uniformly returned to a normal appearance The technique was especially satisfactory in those cases of polyposis and dyskeratosis calling for a "stripping"

In my hands, stripping of the cords with laryngeal forceps has never been a satisfactory procedure The true folds are too often left ragged and irregular, whereas in this method, they remained smoothly denuded

The cordabrasion technique was found to be especially effective with early laryngeal nodules in professional singers and entertainers The smallest of incipient vocal cord nodule or fibroma may be trimmed without taking too much of the approximating edges A quick improvement in voice is thus obtained, and early lesions can be safely operated upon with this technique

The wisdom of denuding both cords at the same operation has been questioned, yet by avoiding the anterior commissure, no webbing, synechiae, or stenosis resulted By allowing only a regime of strict voice rest postoperatively there were no troublesome granulations or granulomas[3] This being, in essence a scarification technique, the complications of granulomata or anterior commissure webbing are considerably less likely

COMMENT

The primary difficulty of removing benign vocal cord tumors is that excision of a sufficient portion of the vocal fold to remove the mass may result in a dysphonia which is irreversible Cordabrasion appears to be a possible solution

Lowenthal[4] emphasizes the principle that removal of the lesion without damage to the underlying elastic membrane will produce a normal cord, however, herein lies the difficulty

I have recently seen a well-known singer whose voice was permanently damaged by the removal of a papilloma from the cord in such a fashion that a large niche was left permanently in the approximating edge

The Jacksons[5] recognized this when they warned against removing too much tissue and leaving a concavity They believed it better to "leave a convex edge of the lesion on the cord, even if this should allow a little of the basal tissue to remain If necessary, a secondary procedure can be done" This quotation would seem to underscore the difficulty of conventional techniques

Again, let me emphasize that this is a preliminary report and a more accurate evaluation of the method can be obtained only after further experience

CONCLUSION

1 The cordabrasion technique represents a possible solution to the oftentimes unsatisfactory results in voice quality which may be obtained by laryngoscopy and biopsy-excision

2 The technique herein described is effectively employed in the removal of the smallest of cordal lesions to the excision of extensive polyposis of the vocal cord

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VIRAL INFECTIONS OF THE RESPIRATORY TRACT*†

W Paul Havens, Jr, M D,

Philadelphia, Pa

The role of viral infections of the respiratory tract as a prime cause of morbidity throughout the world is recognized, and the impact of these diseases on civilian and military populations is rightfully regarded as an economic and medical threat of major importance Their frequent association with bacterial complications augments their dangers In clinical severity, they range through a broad spectrum from minor illnesses to swiftly fatal diseases, and epidemiologically they run the gamut between isolated sporadic cases and world-wide pandemics

During the past 25 years, a great deal of information has become available concerning viruses found in association with diseases of the human respiratory tract, although many of the problems concerned with these diseases remain unsolved[1] Unfortunately, the clinical significance of some of the more recently described viruses is not yet fully appreciated, so that classification on the basis of causative agents is inadequate In addition, the likelihood that numerous, as yet undiscovered, viruses exist further complicates the problem In view of these difficulties, the Expert Committee on Respiratory Virus Diseases of the World Health Organization has recently recommended a simple classification of respiratory syndromes caused by viruses[2] (see Table I) It is my intent to limit my discussion today to influenza viruses and adenoviruses and the diseases they cause, recognizing the fact that the common cold remains the most important unsolved problem in this field

INFLUENZA

Three antigenic types (A, B and C) of human influenza

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*Read at the meeting of the Eastern Section American Laryngological Rhinological and Otological Society Inc Philadelphia Pa Jan 8 1960

†From the Jefferson Medical College Philadelphia

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virus are recognized Types A and B have undergone several changes antigenically since they were first described, although the alterations in influenza B viruses have been much less striking than those observed in influenza A viruses No evident changes in antigenic constitution have been found in influenza C viruses Since the first recovery of influenza A virus in 1933, minor changes have occurred from year to year, with major alterations occurring infrequently and marked by the appearance of antigenically quite different strains of virus that remained predominant In 1946-47, A 1 influenza virus was recognized and represented the first important antigenic change of this kind The second and most striking change in antigenic character was observed in 1957 when the A 2, or Asian influenza virus, appeared

TABLE I

Respiratory Syndromes Caused by Viruses

1 Influenza	Types A, B, C
2 Common cold	Unknown
3 Virus pneumonia	Psittacosis Adenoviruses Atypical pneumonia (Eaton, et al)
4 Unclassified acute respiratory disease—	
Acute respiratory disease	Adenoviruses (ECHO, JH, 2060)
Nonbacterial exudative pharyng	Adenoviruses
Pharyngoconjunctival fever	Adenoviruses
Keratoconjunctivitis	Adenoviruses
Obst laryngotracheobronch	CA virus (Inf , HA 2)
Acute pneumonitis (infants)	Sendai (Adenoviruses, HA 1)
Herpangina	Coxsackie A
Pleurodynia	Coxsackie B

These data were assembled from the First Report Expert Committee on Respiratory Virus Diseases World Health Organization Technical Report Series No 170 1959

The pandemic caused by the Asian virus has been described repeatedly Following its appearance early in 1957 in China, the virus spread throughout the world during the next six months Four phases could be discerned in its progress The first phase was the introduction of the virus, spread by human contact, and apparently more frequently by means of shipping and rail traffic than by air travel

The second phase was that of virus seeding Although in some areas infection spread rapidly after introduction of virus, particularly in Europe and the United States, large outbreaks did not occur until six weeks or more following the first detection of virus

The third phase was that of the main epidemic Throughout the world, the morbidity was high, the mortality low, and outbreaks were often explosive Morbidity was greatest among young children, but mortality increased with age

The fourth, postepidemic, phase followed the main epidemic phase and was characterized by an apparent relative quiescence of infection, in an environment in which Asian virus was isolated for many months from sporadic cases

During the pandemic of 1957, death occurred in some patients with influenzal pneumonia without any evidence of coincident bacterial infection The histologic changes in the lungs of such patients closely resembled those seen in patients dying of influenza during the pandemic of 1918 When pneumonia occurred during the course of influenza in 1957, it was usually in patients with pre-existing chronic pulmonary congestion associated with disease of the heart and lungs or with late pregnancy In patients dying of Asian influenza, secondary bacterial infections were common and pneumococcal and staphylococcal pneumonias were most frequently encountered, the latter being more prominent

With the exception of atypical pneumonia associated with psittacosis, there is no specific treatment for any of the diseases of the respiratory tract due to viruses, including influenza When influenzal pneumonia is complicated by secondary bacterial infection, appropriate antibiotic therapy is indicated, however, the indiscriminate use of antibiotics in uncomplicated influenza in previously healthy persons is unwarranted There may be some justification for the use of antibiotics in patients with chronic pulmonary or cardiac disease who contract influenza, although this should be a matter of individual appraisal and not a routine procedure A similar attitude of mind should condition the approach to the care of patients with acute staphylococcal infections, diabetes, neurological disorders with respiratory involvement, and in late pregnancy patients who acquire uncomplicated influenza

The administration of 1 0 ml of inactivated virus vaccine containing representative strains of influenza A and B viruses is the most effective method for the prevention of influenza Immunization of children should be modified by reducing the amount of vaccine according to age and weight, and in all groups the injection of vaccine into egg-sensitive individuals must be avoided

ADENOVIRUS INFECTIONS

Among the recently described adenoviruses, there are at least 18 distinct antigenic types Some of these are important causes of acute infections in man, involving the respiratory tract from the nose to the lungs, the conjunctivae, and the intestinal tract The clinical pattern of disease depends on the extent and location of involvement Cellular necrosis, with inflammation and hypertrophy of the regional lymphoid tissue, occurs

The respiratory disease produced in adults by adenoviruses is characterized by headaches, malaise, and aching muscles Fever, pharyngitis, and cough are present, and they may be accompanied by varying degrees of evidence of involvement of other tissues, including conjunctivitis, coryza, exudate on the hypertrophic lymphoid tissue of the throat, lymphadenopathy, tracheobronchitis, bronchiolitis, and atypical pneumonia The syndromes of undifferentiated acute respiratory disease, nonbacterial exudative pharyngitis, atypical pneumonia, and pharyngoconjunctival fever fall in the group of conditions caused by these viruses In addition to respiratory illnesses, follicular conjunctivitis, epidemic keratoconjunctivitis, and, in children, gastrointestinal symptoms may be caused by certain of these viruses

Adenoviruses are widespread throughout the world They may be recovered from the nasopharynx and from feces, and may be transmitted by the respiratory or gastrointestinal route Outbreaks of acute respiratory illness caused by these agents are facilitated by crowding and poor hygiene, and favorite sites for epidemics are military camps, particularly among new recruits, and institutions for children These infections are especially prevalent during the Winter, and the morbidity in the Summer is usually low At present, they are far more common among the military, although Summer epidemics of Types 3 and 7 infections have been observed among civilians, especially children

Inactivated vaccines containing Types 4 and 7 have been proven to be very effective in preventing acute respiratory disease caused by these agents, however, the low incidence of disease in the civilian population and in permanent military personnel does not justify the use of the vaccine except among newly recruited personnel in the Armed Forces where the morbidity is high

SUMMARY

Infections of the respiratory tract constitute a problem of prime importance throughout the world In spite of the tremendous advances being made in discovering new respiratory viruses, most of these infections are still undefined as far as causative agents are concerned Much has been learned about influenza viruses and, more recently, about adenoviruses and the diseases they cause, however, the common cold still confronts investigators in this field as the most important unsolved problem, since the causative agent or agents are yet unknown

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ENZYME THERAPY IN OTOLARYNGOLOGY

A Report on the Use of Buccal Tablets of Streptokinase and Streptodornase (Varidase)*†‡

WATT W EAGLE, M D ,

Durham, N C

Enzymes of the hemolytic streptococcus bacteria have been used therapeutically since 1949 for various inflammations, with or without infections We shall trace this enzyme therapy through the era of topical application, the intravenous and the intramuscular methods of treatment This present report refers to our experience, using the buccal route of therapy, in which an enzyme is absorbed through the buccal or sublingual mucosa as the tablets dissolve

The historical background of the development by research of the enzymes that originate from hemolytic streptococcus cultures is a fascinating one and is excellently summarized in a paper by Sherry,[1] which I presume most of you from the St Louis area heard when it was presented in that city in March, 1958, and which the remainder of you read in the *Annals of Otology, Rhinology and Laryngology,* issue of March, 1959 This article is highly recommended reading, both from the historical and chronological viewpoints, but mainly for the rationale in the use of streptokinase and streptodornase, the enzymes we are discussing

In 1933, Tillett and Garner,[2] to their surprise, demonstrated that a culture of hemolytic streptococci added to normal human plasma had the capacity of causing immediate clotting, and that then the clotted fibrin immediately liquefied This was peculiar in that it differed from the usual digestion of solid proteins by a proteolytic enzyme They found the same

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*Read at the meeting of the Southern Section American Laryngological Rhinological and Otological Society Inc New Orleans La Jan 15 1960

†From Department of Surgery Division of Otolaryngology Duke University Medical Center

‡Streptokinase and Streptodornase (Varidase-Buccal Type) supplied by Lederle Laboratories Division of American Cyanamid Company

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reaction would take place from cultures obtained from patients, and there were 28 strains of hemolytic streptococci in the cultures used These infections included scarlet fever, erysipelas, cellulitis, abscesses, and numerous other infections In 1934 these same authors[3] demonstrated that blood sera from patients having acute bacterial infections are capable of agglutinating certain types of hemolytic streptococci, but this function was present only during the acute infections and disappeared soon after the patients recovered This function differed from antibody formation in combatting a disease They reaffirmed that plasma containing a culture of hemolytic streptococci caused immediate agglutination of the organisms in a very few minutes, even in dilute solutions The inability to produce agglutination in normal serum led to the testing with fibrinogen, which is the protein in plasma, but normally absent from serum They stated that fibrinogen increased in effectiveness during an acute infection and was the active principle in plasma for the agglutination

In 1945 Christensen[4] stated that the lysing factor in liquefying a clot represented a proteolytic enzyme normally present in human serum, but inactive On addition of the hemolytic streptococcus fibrinolysin, the lysing factor (fibrinogen) was activated, similar to the tryptogen, or the enterkinase in other physiological studies relative to the intestinal tract. He also stated that fibrinolysin had no proteolytic activity and that the lysing factor could stimulate the proteolytic activity spontaneously He postulated that the lysing factor was an inactive proteolytic enzyme, which is a zymogen, in serum activated by a kinase In this instance the kinase was hemolytic streptococcus fibrinolysin, which he termed streptokinase

In 1948 Tillett, Sherry and Christensen[5] were still investigating the effects of streptokinase on cultures and specimens of pus obtained from purulent infections, such as pleural exudates, and they discovered that there was a hemolytic streptococcal enzyme in the streptokinase preparation they were using which was capable of causing a complete dissolution of nuclear protein, which is the solid material and sediment in pus This enzyme was found to be desoxyribonuclease, which they termed streptodornase This new enzyme, strepto-

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the femoral and other large blood vessels in dogs By 1955 there were several reports[9] of intravenous therapy of streptokinase in humans, however, in the human, this type of therapy was not without complications, such as fever, systolic hypotension of temporary nature, and polymorphonuclear leukocytosis, but, there was no change in the clotting or bleeding time of any of the patients Extra-vascular bleeding was rare but also was a possible complication during intravenous therapy

Streptokinase and streptodornase were now recognized as most useful for the treatment of certain and restricted conditions, if used cautiously and a proper blood level maintained, not only in infections but also in injuries where there was extravasation of blood and fluid into the tissues with typical signs of inflammation and edema

Sherry[1] suspected that many diseases, such as arthritis gout, bacterial endocarditis, or any of the diseases which are manifested by a precipitation of abnormal material in blood vessels or body tissues may eventually be treated successfully by a specific enzyme

Soon after the complications were noted following intravenous therapy, attention was directed to the intramuscular method, however, due to localized pain and nodule formation at the site of the intramuscular injection, this method was used sparingly, and mainly in those cases where very few injections were required—and even then only for an acute inflammatory process of short duration The preparation had been used both intravenously and intramuscularly in animals for infections, inflammations, and prevention of abdominal adhesions, and even to the present date the preparations are still used in general surgery by these methods where there are thromboses and purulent infections

Since the intravenous and intramuscular methods of therapy had been followed by complications, studies were then begun to ascertain whether another method of administration was feasible or possible The therapeutic value of streptokinase and streptodornase had been established The problem was how best to administer it dornase, was found to liquefy completely purulent discharge by breaking up all of the nuclei of white blood cells and pus cells This then led to further studies by Tillett, Sherry and others,[6] reported in 1950, when they announced that streptokinase and streptodornase could be introduced locally and topically into the thoracic cavity to cause rapid enzymatic changes with debridement without any generalized reaction to the patient Streptodornase was found to have no effect on living cells The patients who were treated had empyema, pneumothorax, pneumonectomy, osteomyelitis, sinusitis, and degenerative ulcerative lesions

Of otolaryngological interest, this is the first instance of enzymatic therapy for a sinus infection The case treated was a 42-year-old male who had a chronic left maxillary sinus infection for three years, with an acute flare-up of ten days' duration One-half cc of pus was aspirated from the sinus by a needle, and 500 units of streptokinase and 1000 units of streptodornase were introduced through the needle A few minutes later 5 cc of pus had liquefied and, thus, could be aspirated Follow-up cultures were sterile, and the patient had immediate relief of the acute sinus infection

The same investigators observed that by topical application of streptokinase and streptodornase there was an acceleration of recovery of patients having any disease in which there was a fibrinous exudate They noted that extra-vascular clots or loculated hemorrhages quickly cleared up by lysis Most remarkable was the disappearance of all bacteria from wounds The removal of debris from these infected areas allowed natural and acquired immunity to function, and, together with antibiotics, which had made their appearance by this time, the recovery was dramatic and rapid In their studies they found that there was no injury to the mucosa or soft tissues during any local or topical therapy

In 1952 Johnson and Tillett[7] reported the injection of streptokinase and streptodornase intravenously in animals for dissolving venous thrombi in ears of rabbits, and Sherry and his co-workers[8] in 1954 demonstrated that intravenous injections of streptokinase caused dissolution of thrombi in thyroglossal duct cyst, tonsillectomy and adenoidectomy, and mastoidectomy We also have used the preparation on patients who have had third molar extractions by their dentists, abscesses of the cheek, hemorrhage and hematoma of the vocal cord, secretory otitis media, hematoma of the tongue (from biting the tongue), herpes labialis, and some of the dermatoses One case of poison oak cleared immediately, but two cases of trumpet vine (cow itch) did not respond to this therapy One case of erythema multiforme completely disappeared and did not recur for several months A case of chronic pharyngitis due to the local use of antibiotic lozenges did not respond, and neither did one case of secretory otitis media, but other cases of secretory otitis media did respond quite well Two cases of granulation tissue of the mastoid were successfully treated, but in one case granulation of the middle ear did not respond to a minimal amount of the therapy We really could not evaluate this latter case, however, because of the extreme sensitvity of this patient to every known drug, and treatment had to be discontinued because of stomatitis after using two tablets—our only case of such a complication

Our most remarkable results relative to rapidity of disappearance of inflammation and infection were observed following external frontal or Caldwell-Luc maxillary sinus surgery In most instances we were dealing with a purulent frontal sinus infection of osteomyelitic type, or a polypoid and purulent infection in the maxillary sinus In one case, surgery on the frontal sinus had to be delayed until the patient had buccal and tetracycline therapy because of intense acute infection due to bony perforation with edema, redness, and purulent infection In another instance there was an unusual frontal sinus disease found unexpectedly at the time of the original operation This was a case of a giant-cell type of fibrosing osteitis, whereby the entire nasofrontal duct had been sealed off A new nasofrontal duct was made, a rubber tube inserted, and some weeks later the tube was removed Then a few months later there was a sudden swelling and abscess formation over the frontal region, with elevation of the periosteum of the frontal bone and eventual perforation

After much study for many months it was found by many investigators[10] that there was good absorption of streptokinase and streptodornase by the buccal mucosa, that there was good penetration through the mucosa, and that the desired effect was produced as well by buccal absorption as by the intravenous and intramuscular methods, furthermore, there were no side ill-effects, which had occurred with the other methods of therapy Various workers experimented with various diseases, including bronchitis, bronchiectasis, retinal vein thromboses, chronic leg ulcers, phlebitis, post-dental extraction sockets, and oro-pharyngeal surgical wounds In addition, the preparation was used in acute coronary thrombosis, myocardial thrombosis and infarction, thrombosis of various arteries and veins, and other types of disease wherein there was a fibrin clot or exudate, with or without infection Bruises of muscles and other tissues, sprains of ligaments and tendons were found to respond well This is of extreme significance because of the economic factor of patients being able to recover quickly and return to work Athletes, who are continually subject to injuries, with inflammation and hemorrhage, have benefited markedly by this therapy, however, one must not prescribe the preparation for a patient having a collagen disease, a diminished liver function, or a blood-clotting disease The preparation also should not be administered after or during an acute hemolytic streptococcal disease because of a possible allergic manifestation

The Otolaryngological Service of Duke Hospital has used streptokinase and streptodornase, buccal type, for various types of cases, but this report is based entirely on my personal experience with my own private patients In June, 1958, a series of almost 100 cases was started in which we used the buccal method of therapy The greater portion of these cases reported herein was selected on account of postoperative edema, ecchymosis, hematoma, or infection prior to surgery We have used the preparation in conjunction with the following operations external frontal sinus, intranasal ethmoid, sphenoid, submucous resection of the nasal septum, Caldwell-Luc maxillary sinus, shortening of elongated styloid process, removal of pharyngeal and nasopharyngeal tumors, excision of

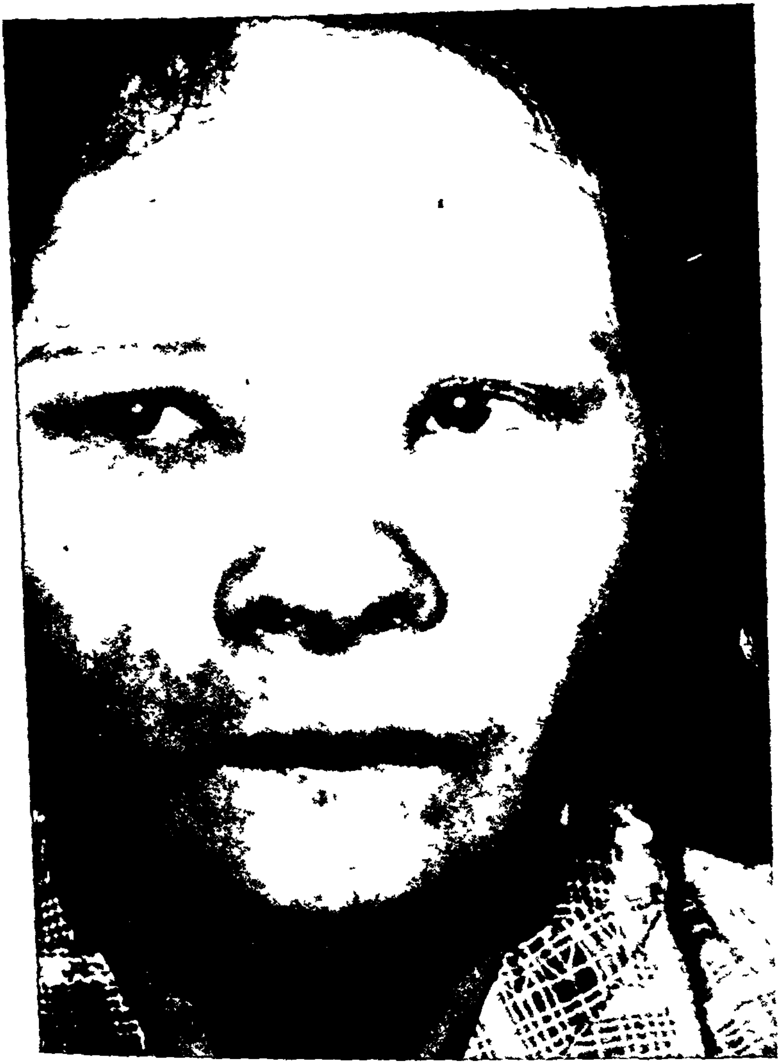

Fig 2 M S age 29 F-1 619 Submucous resection of nasal septum June 19 1959 Buccal streptokinase and streptodornase started June 1959 Photo made on June 20 1959

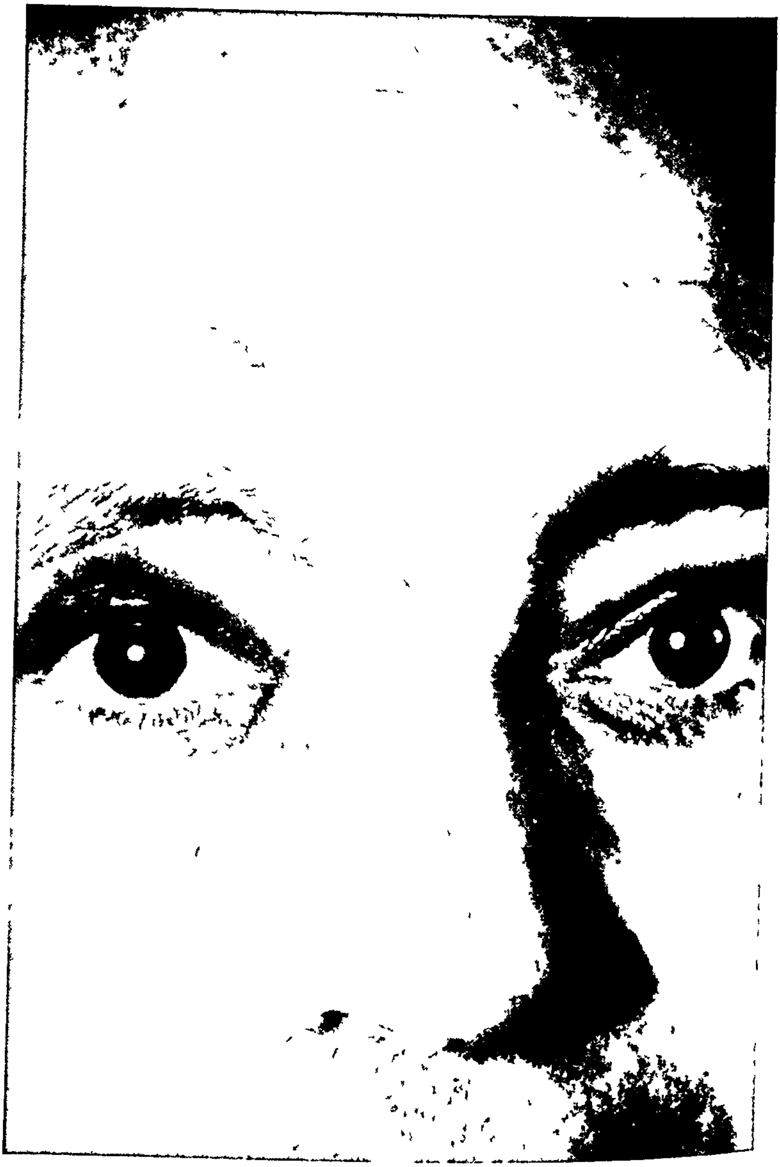

Fig 1 J L A age 50 D-55658 Had external frontal operation June 9 1959 at which time ossifying fibroma found—giant-cell type Nasofrontal duct obliterated Revised external frontal operation Sept 16 1959 following abscess with skin perforation This photo was made Oct 10 1959 and shows complete healing of incision and sinus tract by use of buccal streptokinase and streptodornase with tetracycline

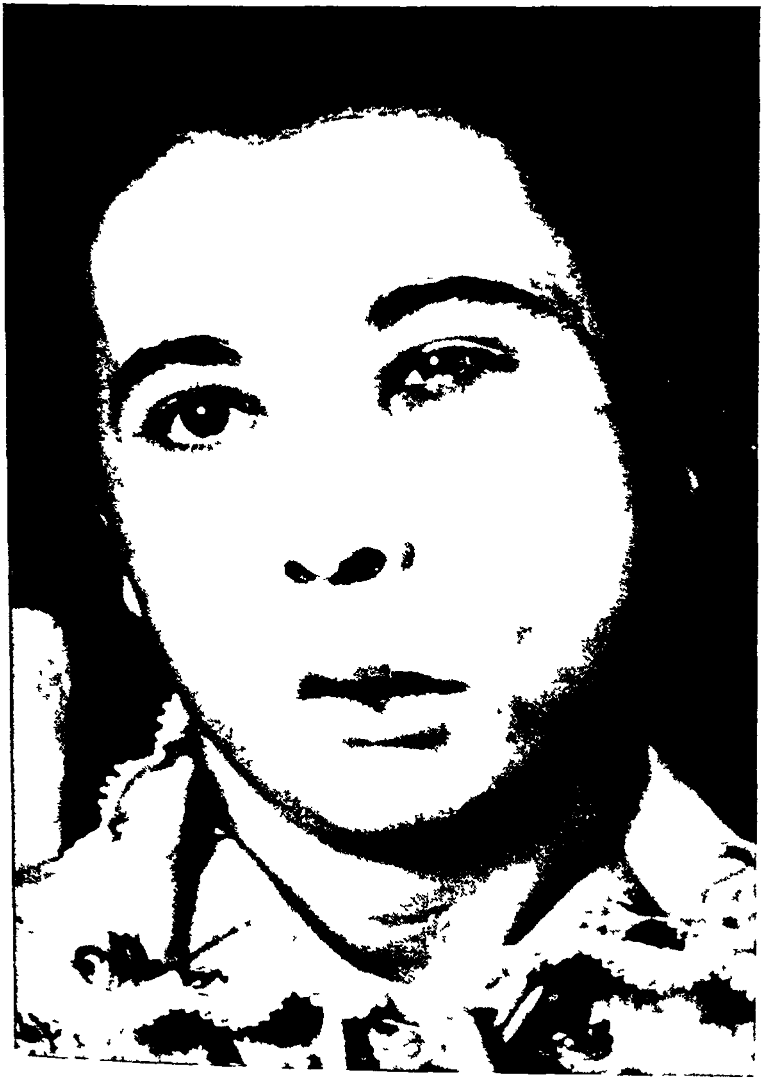

Fig 4 K H age 22 F-04782 Submucous resection of nasal septum Aug 10 1959 Caldwell-Luc left cysts and polyps found Aug 11 1959 Buccal streptokinase and streptodornase started Aug 12 1959 This photo made Aug 12 1959

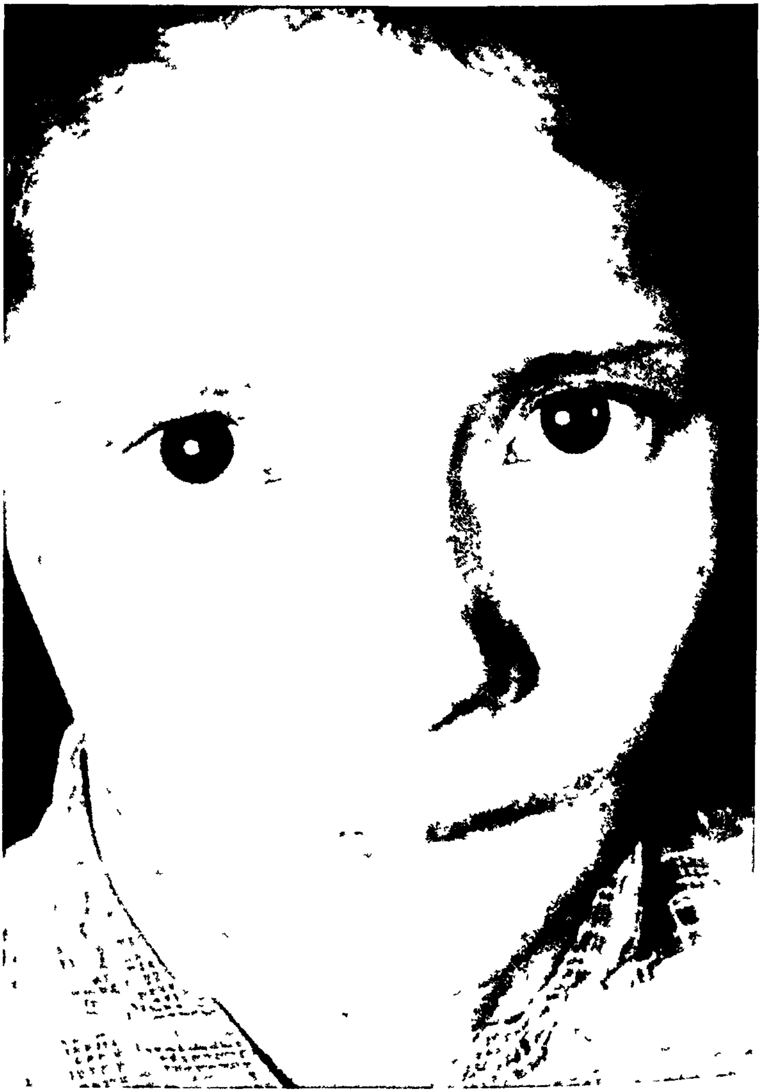

Fig 3 Same patient M S F-1 619 This photo made on June 22 1959 after use of ten buccal tablets Not an allergic individual unexplained postoperative edema

of the skin Again buccal and tetracycline therapy was used, with subsiding of the acute infection (see Fig 1) Then we were able to proceed with a revision of the external frontal sinus operation, at which time a polyethylene tube was inserted into the re-opened nasofrontal duct The patient is was very small or massive (see Figs 2, 3) Complete recovery was rapid in all cases except one, a Japanese student who seemed to have no response at all to the preparation All cases of postoperative maxillary sinus edema and ecchymosis following Caldwell-Luc operation responded with equal rapidity (see Figs 4, 5) The edema and ecchymosis that occurs, especially in allergic individuals due to stirring up of the infection by the surgery (an example of bacterial allergy), usually subsided in two or three days This method of therapy produces much more rapid reduction of edema than intravenous calcium therapy or any other means of reducing post-traumatic edema

I cite a case of herpes labialis, with the lesions extending over the cheek This patient, a nurse, had recurrent attacks of herpes since childhood, which came on quite often following exposure to sunshine Smallpox vaccinations and other means of treatment in the past had failed to provide relief She stated she would like a trial of the buccal therapy and did derive immediate benefit on one day's therapy but continued the medication for several days There was no recurrence for almost eight months, and then she was able to abort an attack during the recent holiday season by using one tablet

One patient accidentally bit her tongue early one morning, causing a hematoma about the size of a pencil eraser By late afternoon, after using only two buccal tablets, the hematoma had completely disappeared In another patient, a school teacher, a hematoma or submucosal hemorrhage of a vocal cord disappeared overnight following minimal buccal therapy Ordinarily such a hematoma would require two or three weeks to disappear spontaneously

We have not used streptokinase and streptodornase in as many post-tonsillectomy cases as we had hoped to but we can assert that the postoperative slough is less thick and disappears at least one day earlier, and it is also thought there is less pharyngeal angina We plan to use the buccal therapy on a larger series of adult cases, because we are satisfied that it is beneficial

It has been established that the optimum dosage of strepto-

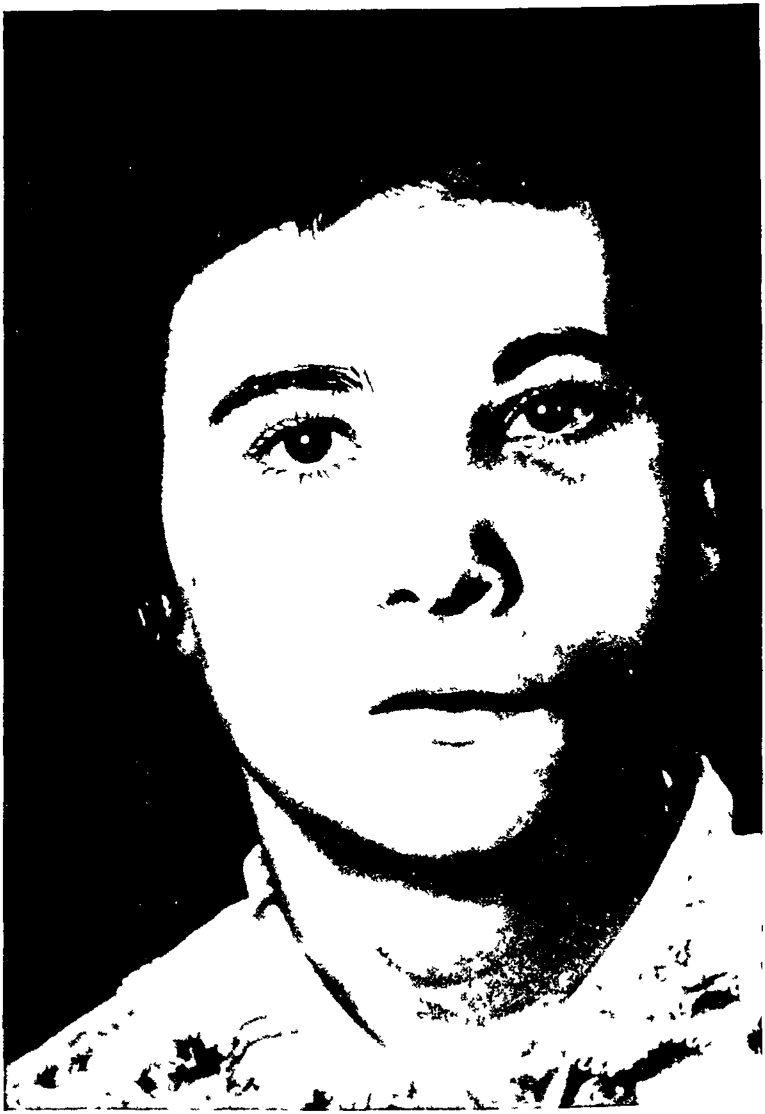

Fig 5 K H F-04782 This photo made on Aug 15 1959, after use of 14 buccal tablets

still wearing the tube *in situ,* and he has had no recurrence of any trouble up to the present time—seven months

The buccal streptokinase and streptodornase were used in many cases of postoperative hematomata of the nasal septum, after the hematoma had been removed, whether the hematoma cases in which buccal type of therapy was used for various otolaryngological conditions

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Duke University Medical Center

HUMBOLDT UNIVERSITY OF BERLIN (CHARITE)

The celebration of the 250th Anniversary of the Charité will be held in Berlin from November 6-19, 1960, in connection with the 150th Anniversary of the Humboldt University

Applications for participation are to be directed to the Committee for the Preparation of the 250th Anniversary of the Charité, Berlin N 4, Schumannstrabe 20-21, c/o Dozent Dr med habil Dagobert Muller, secretary of the committee kinase and streptodornase for an average-sized adult is four tablets per day Each tablet contains 10,000 units of streptokinase and at least 2,500 units of streptodornase The tablets are placed between the cheek and the upper alveolar process If the patient is edentulous, the tablets are dissolved beneath the tongue The patient must swallow as little as possible in order to permit complete absorption of the tablets through the buccal mucosa to obtain the full benefits One, of course, must not drink any fluids as long as the tablet has not dissolved, which generally required 20 to 40 minutes The tablet must not be chewed or swallowed whole, since there is no benefit to be derived from it after it reaches the stomach The duration of treatment depends on the type and severity of the condition being treated In all cases where hematoma or ecchymosis without infection is involved, treatment for two or three days may be long enough When infection is present, one must continue the treatment for a minimum of five days, and also when infection is present, it is most important that tetracycline drug therapy be used along with the buccal therapy The tetracycline is administered in full dosage for a minimum of five days also There are some instances, however, with or without infection, when the therapy must be continued for periods longer than stated above

SUMMARY

We have traced the production of a fibrinolytic enzyme, from the hemolytic streptococcus organism, which causes dissolution of blood clots and the melting of exudates and fibrin collections This enzyme was named streptokinase It was later discovered that this material also had another enzymatic property which caused liquefaction of pus and other dead cells in any type of discharge, the nuclei of these cells being dissolved by this enzyme, which was named streptodornase

We have traced the therapy with these enzymes through the topical application method, then the intravenous and the intramuscular methods, and in this paper we have described the buccal method of therapy of streptokinase and streptodornase We are reporting our experiences with almost 100 founders and trustees of the Pennsylvania Hospital, the first hospital to be established in the colonies His father was variously a cotton broker, textile manufacturer, magazine writer and novelist

From the University of Pennsylvania, he received a B A degree in 1894 and an M D degree in 1897 During his college years he was active in sports, serving as captain and manager of the track team and manager of the football team As a champion bicycle racer, he received numerous trophies and awards

He engaged in the study of otolaryngology with Dr Francis R Packard at the Pennsylvania Hospital and with Dr Walter Roberts at the Philadelphia Polyclinic and College for Graduates in Medicine from 1901 to 1905 At the latter institution, he became associate professor of otology and, with its merger with the Graduate School of Medicine of the University of Pennsylvania, he was appointed professor and chief of service of otolaryngology in the Graduate School He was also professor and chairman of the department of otolaryngology in the School of Medicine of the University of Pennsylvania and chief of service at the University Hospital from 1933 until he became emeritus professor in 1939 For many years he was chief of service at the Presbyterian, Abington Memorial, Germantown and Chester County Hospitals

Dr Coates became a director of the American Board of Otolaryngology in 1930 and a senior consultant in 1956 He was a past president of the American Laryngological Association, the American Otological Society, the American Laryngological, Rhinological and Otological Society, the American Academy of Ophthalmology and Otolaryngology and the Philadelphia Laryngological Society He was the recipient of the Newcomb Award of the American Laryngological Association in 1943, and of the de Roaldes Gold Medal in 1949, the Award of Merit of the American Academy of Ophthalmology and Otolaryngology in 1946, and the Gold Medal Award of the American Otological Society in 1949

Dr Coates was chairman of the section of Laryngology, Otology and Rhinology of the American Medical Association in 1927, chairman of the ear, nose and throat section of the

In Memoriam

GEORGE MORRISON COATES, M.D.,

1874-1960

Dr. George Morrison Coates died suddenly of coronary occlusion at his home in West Whiteland, Pa., on February 7, 1960, within a month of his 86th birthday. He continued the private practice of his specialty in Philadelphia and the performance of his editorial duties up to the time of his death.

He was born in Philadelphia on March 24, 1874. His Quaker ancestors settled in the area prior to the arrival of William Penn, and members of the family were among the

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COLBY COLLEGE EIGHTH CONSECUTIVE INSTITUTE ON OCCUPATIONAL HEARING LOSS

Colby College, in Waterville, Maine, announces its Eighth Consecutive Institute on Occupational Hearing Loss, August 8-13 The course is designed to train physicians, plant nurses, plant engineers and others in initiating and conducting hearing conservation programs in noisy industries The course is comprehensive and includes all phases of the problem The fee of $200 includes tuition, board and room Requests for further information should be made to William A Macomber, Colby College, Waterville, Maine Dr Frederick T Hill and Dr Joseph Sataloff are directors of the program

Pennsylvania Medical Society in 1921, chairman of the ear, nose and throat section of the Clinical Congress of Surgeons of North America from 1921 to 1936, and chairman of the ear, nose and throat section of the College of Physicians of Philadelphia from 1928 to 1930

He was a corresponding fellow of the Societé de Laryngologie des Hôpitaux de Paris and a fellow of the American Bronchoesophagological Association, the American Society of Immunologists, the American College of Surgeons and the Pacific Coast Oto-Ophthalmological Society

Dr Coates was editor-in-chief of the *A M A Archives of Otolaryngology* from 1937 to the time of his death, co-editor with Dr Chevalier Jackson of "The Nose, Throat and Ear and Their Diseases" (1929), co-editor with Dr I S Ravdin of the American Edition of "Kirschner's Operative Surgery" (1937), and editor of the five-volume "Otolaryngology" (1955)

As Assistant Surgeon in the U S Navy, he participated in the Battle of Santiago Bay during the Spanish American War, and later served as Lieutenant to Lieutenant Colonel in the Pennsylvania National Guard from 1900 to 1916 During World War I, he served as Major to Colonel, MC, U S Army, and was chief of the section of head surgery at the base hospitals in Camp Sevier, S C, and Camp Hancock, Ga, commanding officer of Base Hospital No 56, Allerey, France, and president of the Reclassification Board, Bordeaux, France

Dr Coates played a major role in the development of graduate teaching in otolaryngology and contributed more than a hundred scientific papers to the literature His primary interest was teaching and counselling young men in the specialty and his inspiration shaped the careers of many otolaryngologists both in this country and abroad

Dr and Mrs Coates were active in the Episcopal Church and their home in Philadelphia was always a center of hospitality for colleagues and students Mrs Coates died in 1954 and his son met a tragic end in 1955, leaving a widow and two sons

He is survived by his daughter and two grandsons

H P S

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DYSRHYTHMIA IN THE POST-CALORIC NYSTAGMUS ITS CLINICAL SIGNIFICANCE*

J S RIESCO-MACCLURE, M D
(By Invitation),
and
MALCOLM H STROUD, M D,
(By Invitation),
St Louis, Mo

The functional examination of the vestibular system is based primarily on the observation and study of one sign, the nystagmus The study of nystagmus, however, is not easy because of its great variety of form and presentation

First, there is spontaneous nystagmus which may be due to an inner ear disturbance, to a lesion in the central vestibular system, it may be of cerebellar origin, or it may even be present in supratentorial tumors

Second, there is positional nystagmus which according to Nylén's[1] classification might be divided into three different types Aschan's[2] modification of Nylén's classification is in perfect agreement with our clinical experience; therefore, we have adopted it Type I is a direction-changing nystagmus non-paroxysmal, with no latency period, persistent (which means nystagmus of unlimited duration in a given position), and very seldom accompanied by postural vertigo It is found chiefly in posterior fossa lesions[3]

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*Read at the Sixty-Third Annual Meeting of the American Laryngological Rhinological and Otological Society Inc Miami Beach Fla March 15 1960

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may be seen in streptomycin intoxication) *2* Vestibular hyperexcitability This means a large and long duration nystagmus, above the normal values We believe it is due to an increased excitatory state of the central vestibular system rather than to an end-organ hyperexcitability[6] *3* Cochlea-vestibular dissociation[6] This means normal hearing and complete vestibular paralysis (we have found it in tumors destroying the vestibular nuclei in the floor of the fourth ventricle) *4* Nystagmo-vertiginous dissociation (large post-caloric nystagmus without sensation of vertigo) We believe this is due to a cortical inhibition of the sensation; it also might be present in lesions in various sites of the brain *5* Directional preponderance of the nystagmus[9] This means the elicited nystagmus is larger in amplitude and longer in duration in a certain direction, regardless of the ear stimulated (a condition sometimes seen in brain stem lesions as well as in cases of Ménière's disease)[6] *6* Perverted nystagmus This means the nystagmus induced by caloric stimulation in the usual position is vertical instead of horizontal We have verified it in partially destructive lesions of the vestibular nuclei[8] *7* The absence of the quick component This is a rare otoneurological symptom According to Nylén[10] and Aubry[11] it is found when the medial longitudinal fasciculus has been involved by lesions in the pons or brain stem Experimental work, however, has shown that lesions in the reticular formation without sectioning the medial longitudinal fasciculus produce a monophasic nystagmus consisting of a steady slow component (Lorente de No[12]) *8* Inhibition of the nystagmus either with the eyes open due to fixation of the visual axis, or occasionally only, with the eyes closed due to a higher (perhaps cortical) inhibiting influence There is no question that in the majority of the cases the nystagmogram shows a larger nystagmus with the eyes closed,[2] due to the fact that fixation of the gaze inhibits the nystagmus when the eyes are open, but not too infrequently there are exceptions in which the nystagmogram shows no nystagmus when the eyes are closed and a clear nystagmus as soon as the eyes are opened (see Fig 1) We believe that some individuals are capable of inhibiting the nystagmus without voluntary effort by some unknown mechanism Because this

Type II is a direction-fixed nystagmus Whatever the position of the head, the nystagmus always beats in the same direction

Type III is again a direction-changing nystagmus, but paroxysmal in character, with a short latency period preceding the onset of the nystagmus It is transitory, since it lasts for a short period of ten to 30 seconds, and is also transitional, since it may change its direction with any given posture, and is usually accompanied by severe postural vertigo This type has been called "paroxysmal positional nystagmus of the benign type"[4] because as a rule it is a sign of a benign disturbance, nevertheless, we[3] have been able to verify it in two cases of astrocytoma in the cerebellar vermis, and recently Fernández, et al ,[5] have demonstrated it in cats, after the flocculo-nodular lobe in the cerebellar vermis was surgically damaged

The clinical significance of Type II depends on its similarity with the other two types If it is direction-fixed, non-paroxysmal, persistent and without postural vertigo it should be grouped with Type I In 27 patients with encephalic lesions[6] we found Type I in 13 and Type II in 14 On the contrary, if it is paroxysmal, transitory and accompanied by severe postural vertigo, we group it with Type III which as we said, is usually a benign condition

Fernández, et al ,[7] have found that ablation of the nodulus in the cat was followed by a Type II vertical nystagmus toward the lower eyelids, either persistent or transitory In some animals, however, a paroxysmal nystagmus was superimposed upon the persistent one Both were in the same direction, but the paroxysmal feature appeared after a short latency period and lasted for a few seconds We believe this unusual disorder should be looked for in patients with suspected lesions in the cerebellar vermis

Finally, there are *abnormalities in the nystagmus induced by the caloric tests* We shall mention them briefly *1* Dissociation between the cold and hot responses[8] This means the nystagmus may be induced only by irrigation with water at 44° and not with water at 30° or at 18° (a condition which

The purpose of this communication is to study the clinical significance of this otoneurological sign (dysrhythmia of the post-caloric nystagmus) and to emphasize its value in the diagnosis and localization of central vestibular lesions.

In order to present this otoneurological sign and its clinical significance we have adopted the following procedure for this publication: *1* a description of that part of the central vestibular system in which a lesion might produce the sign; *2* a report of our early cases in which the dysrhythmic nystagmus was verified by direct observation, *3* a report of two cases examined with electronystagmography and *4* a discussion of the subject.

THE CEREBELLO-VESTIBULAR INTERRELATIONSHIP

The cell bodies of the first vestibular neuron are in the vestibular ganglion or Scarpa's ganglion which appears as an enlargement of the vestibular nerve in the internal auditory meatus. Their dendrites are the nerve fibers which originate at the specialized epithelium of the cristae and maculae and form the three ampullary nerves, the utricular and the saccular nerves. Their axons form the vestibular nerve. When this enters the medulla oblongata (bulb) it divides into two fascicles: one is the "vestibulo-bulbar" root which conveys impulses to the vestibular nuclei of the brain stem, the other is the "vestibulo-cerebellar" root which conveys impulses directly from the vestibular receptors to the flocculo-nodular lobe of the cerebellum, to the fastigial nuclei and in a lesser degree to some parts of the cerebellar vermis, such as the lingula in the anterior lobe and the uvula in the posterior lobe (see Fig. 3).

The flocculo-nodular lobe is the vestibular lobe of the cerebellum, and its afferent and efferent connections are almost entirely vestibular. The afferent fibers are *predominantly* of vestibular origin. Some of them are primary neurons which come directly from the vestibular nerve (see Fig. 3) and others are secondary neurons from the vestibular nuclei in the floor of the fourth ventricle (see Fig. 4). These fibers which are estimated to be five times as numerous as inhibition may be overcome by distracting the patient's attention we think that it could be of cortical origin It does not depend upon the presence of a sensation of vertigo (see Fig 2)

There is another abnormality in the post-caloric nystagmus which one of us (J S R-McC) has observed during the last

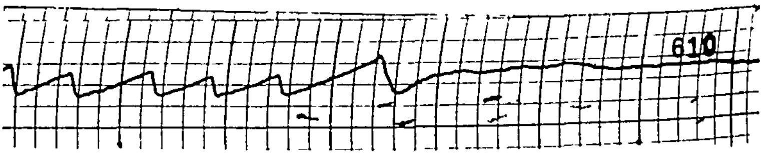


Fig 1 Post-caloric nystagmus present only with the eyes open It disappears with the eyes closed due to inhibition

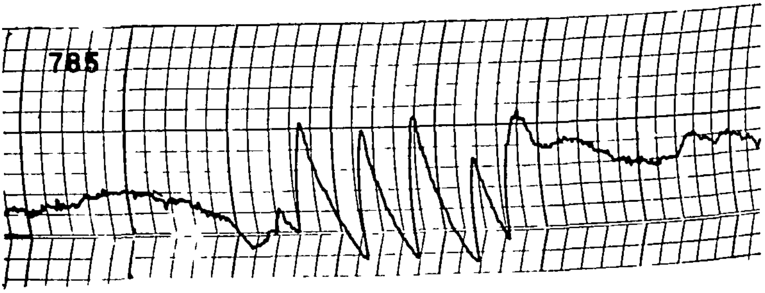


Fig 2 Post-caloric nystagmus with the eyes closed It appears only when the patient is distracted by conversation such as 'Have you ever been in jail?'

three years and termed it “irregularity” This is because the eye oscillations were not regular in their amplitude and frequency, and sometimes the nystagmus appeared in periods of a few seconds with pauses of a few seconds during which no nystagmus could be seen Aschan[2] refers to it as a “dysrhythmia in nystagmus induced by calorization” He states, “We have quite often seen such dysrhythmia, in cases of suspected cerebral lesions, but we have encountered it only extremely rarely in connection with peripheral lesions ” formation and medial longitudinal fasciculus All these connections are ipsilateral (see Fig 5). The efferent fibers of the flocculus also are ipsilateral long Purkinje axons According to Jansen and Brodal[15] "the efferent fibers from the flocculus pass the floccular peduncle and then follow two routes One group of fibers arches above the fibers of the restiform body to terminate in the dorsolateral part of the vestibular nucleus of Deiters The remainder of the fibers forms the 'angular bundle' of Lowy which within the medial part of the juxtarestiform body turns sharply in a rostral direction to terminate in the vestibular nucleus of Bechterew."

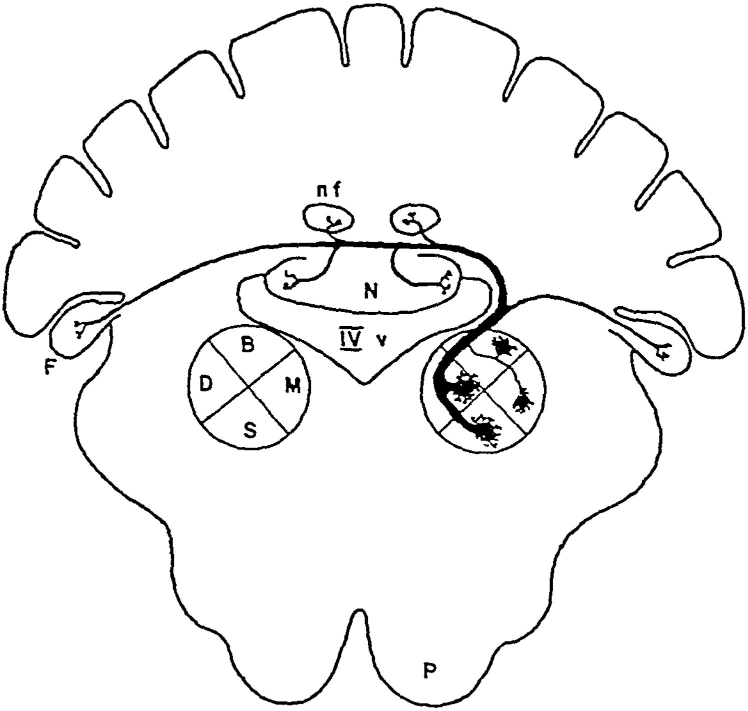


Fig 4 Afferent secondary vestibular neurons to the cerebellum. See text for the description.

The flocculonodular lobe and its afferent and efferent connections are phylogenetically the most ancient and the name of archicerebellum has, therefore, been given to this lobe

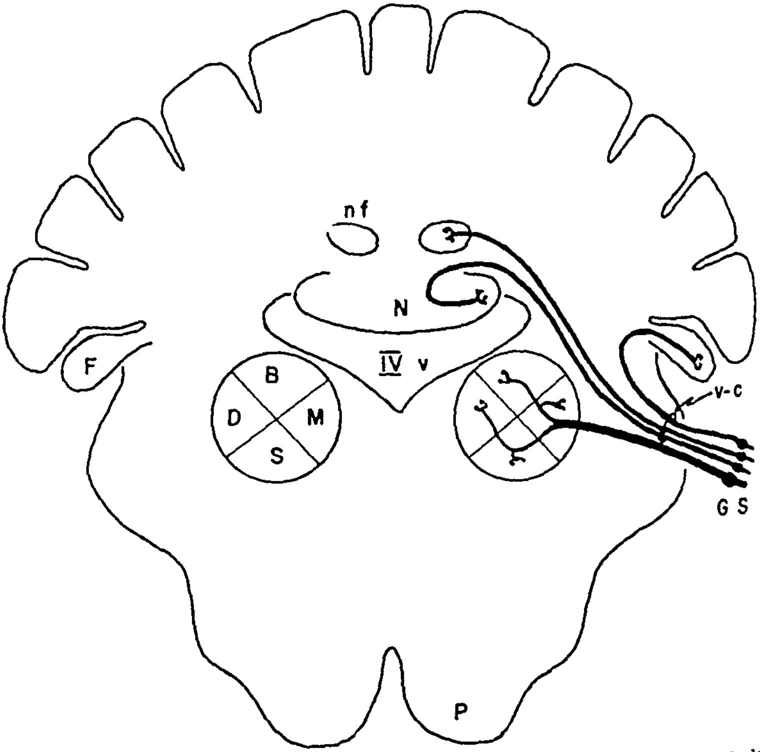


Fig 3 Afferent primary vestibular neurons This oversimplified diagram was composed after the description of Dow[13 14] and Jansen and Brodal[15] In this and following figures the structures are identified by the symbols F flocculus N nodulus, IV v fourth ventricle n f nucleus fastigii B nucleus of Bechterew, D nucleus of Deiters, M medial nucleus S descending nucleus v-c vestibulo-cerebellar root, G S, Scarpa's ganglion P, pyramid For the description of the pathways see text In order to simplify this figure the lingula and uvula were omitted

the direct fibers[13] originate mainly in the medial and descending vestibular nuclei They run dorsally into the cerebellum on the medial aspect of the restiform body (juxtarestiform body) between the latter and the fourth ventricle Some of these bend laterally over the restiform body to reach the flocculus of the same side, others turn medially on the cerebellar wall of the ventricle (roof), ending in the nodulus, uvula, and fastigial nuclei of both sides, and some pass to the flocculus of the opposite side[13 14 15]

The efferent fibers of the nodulus may be divided into short and long Purkinje axons The former end in the nucleus fastigius and the latter in the vestibular nuclei, reticular vestibular fibers from the vestibular nerve and vestibular nuclei (see Figs 1 and 2) but unlike the flocculo-nodular lobe it is not, however, a predominantly vestibular nucleus Actually, the main bulk of its afferent fibers comes from all of the cortex of the cerebellar vermis, and through these fibers the nucleus fastigius is strongly influenced by proprioceptive impulses that have reached the Pudkinje cells of the cerebellar vermis through the spino-cerebellar tracts

The efferent fibers from the fastigial nuclei project to the vestibular nuclei via two bundles, the *direct fastigiobulbar and the crossed uncinate fasciculus or hook bundle* (see Fig 5) The former passes via the juxtarestiform body and terminates in the ipsilateral vestibular nuclei and reticular formation The " hook bundle" sweeps across the midline, loops around the superior cerebellar peduncle and nucleus fastigius of the opposite side, and ends in the vestibular nuclei, especially in the lateral nucleus In normal vestibular reflexes, impulses from the nucleus fastigius decrease or inhibit the discharge from the vestibular nuclei of the opposite side [17] The efferent fibers from the lingula and uvula project into the fastigial nuclei

The ascending vestibulo-cerebellar neurons (afferent) and descending cerebello-vestibular neurons (efferent), which run mainly on the lateral walls of the fourth ventricle (medial aspect of the restiform body or juxtarestiform fibers) and also on its roof, have been named by Crosby[18] the "cerebello-vestibular interrelationship" Through it the cerebellum exerts its influence on the central vestibular system We believe the lack of this influence, due to injury to the connections between the cerebellum and vestibular system, to be responsible for the dysrhythmia in the post-caloric nystagmus

EARLY CLINICAL OBSERVATIONS

Occasionally we face a problem such as the following

A five-year old girl was sent to one of us with the suspicion of a cerebellar tumor The neurosurgeon wanted to know the degree of involvement of the midline structures because such an involvement would make the operation more difficult and also impair the prognosis Although he knew that the VIIIth nerve was intact he wanted to know whether there was dysfunction of the central vestibular neurons (central vestibular

Apparently the cerebellar stamp has to be placed on some of the vestibular data before they can be properly utilized[16]

The *nucleus fastigius* belongs to the group of cerebellar nuclei situated in the depths of the corpus cerebelli immediately dorsal to the roof of the fourth ventricle and extending laterally somewhat beyond it In each side, this group consists

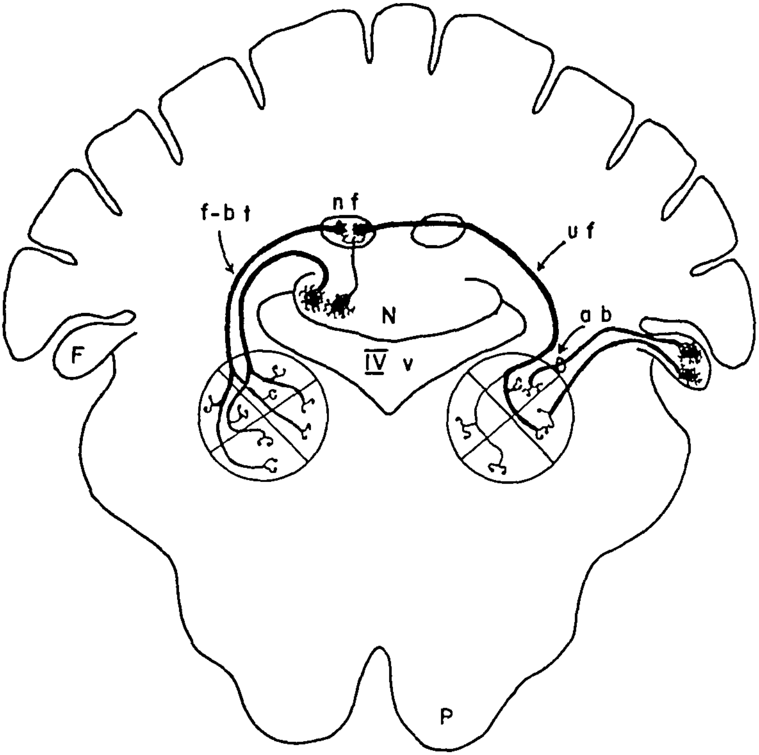


Fig 5 Efferent cerebellar neurons to the vestibular system See text for the description The connections to the reticular formation and medial longitudinal fasciculus were omitted a b angular bundle" of Lowy f-b t fastigiobulbar tract u f uncinate fasciculus

of the nucleus fastigius, nucleus globosus, nucleus emboliformis, and nucleus dentatus The most medial are the fastigial nuclei, sometimes called the "roof or tectal nuclei" because of their position close to the midline in that part of the cerebellum overlying the roof of the fourth ventricle Like the flocculo-nodular lobe, the nucleus fastigius receives afferent line structures of the posterior fossa which involved the cerebello-vestibular pathways

The following case, in which one of us missed the diagnosis, is good support for the diagnostic value of the irregularity or dysrhythmia in the post-caloric nystagmus

A four and a half-year-old boy was sent to the Otoneurological Department of the Neurosurgical Institute, Santiago, Chile in very poor condition The main symptoms were severe headache, vomiting papilledema loss of balance and very marked generalized asthenia He was a very restless child and complained and cried most of the time Functional examination of the VIIIth nerve was somewhat difficult, nevertheless, the hearing was considered normal in both ears The Romberg test was positive he walked on a wide base and was constantly wobbling He practically had a complete loss of balance

He showed bilateral cerebellar signs

No spontaneous or positional nystagmus was found but the nystagmus induced by cold and hot irrigation of both ears was dysrhythmic For a moment it was large and fast, then it almost stopped, and when it reappeared it was small and slow It seemed to be a very abnormal post-caloric nystagmus As the patient was restless moving about and crying we did not know whether the irregularity in the oscillations of the eyes was actually an abnormality or a product of the child's uneasiness In order to be certain we decided to use a stronger stimulus irrigating with 18° water for 60 seconds in each ear We thought that if the abnormalities that we had just seen were true and not due to faulty observation, they should become obvious using a stronger stimulus A perfectly normal nystagmus was elicited on either side, therefore we reported that "there was no involvement in the cerebellar vermis or in the brain stem The central vestibular neurons were intact"

On the next day one of us was called to the operating room to see a large medulloblastoma which not only invaded the cerebellar vermis but also expanded into the fourth ventricle This mistake occurred because we had deliberately disregarded the dysrhythmia in the nystagmus elicited with water at 30° and 44° respectively and considered as valid only the responses at 18° There is no doubt that irregularity in the post-caloric nystagmus was present when the horizontal semicircular canals were stimulated with water at 30° and 44°, but it disappeared when a stronger stimulus was used This was due perhaps to a sort of recruitment phenomenon in the cerebello vestibular interrelationship This unknown possibility was responsible for our mistake Since the post-caloric dysrhythmia was present in a patient with a tumor which involved the midline structures in the posterior fossa the clinical value of this otoneurological sign is supported

DYSRHYTHMIA IN THE POST-CALORIC NYSTAGMUS VERIFIED WITH ELECTRONYSTAGMOGRAPHY

A *Apparatus and Method*

Following the work of Aschan, et al ,[2] it may be said that electronystagmography is an established clinical aid in vestibnuclei and central vestibular pathways), the cerebellar vermis and the important connection between the two (cerebello vestibular interrelationship) The dysfunction of the central vestibular neurons would indicate that the tumor had involved the midline structures The main symptoms and signs in this child were headache, occasional vomiting, slight papilledema, slight unsteadiness and slight impairment of voluntary movements on the right side

The functional examination of the VIIIth nerve showed the following

1 Bilateral normal hearing

2 Slight disequilibrium The Romberg test was normal, and she did not walk on a wide base, but staggered slightly to the right

3 The cerebellar tests showed a slight asthenia and dysmetria on the right side, otherwise the coordination was fairly good There was no intention tremor or dysdiadochocinesia

4 She had no spontaneous nystagmus or positional nystagmus, and the caloric tests were completely normal

Comment The lack of spontaneous and positional nystagmus, and the absence of abnormalities in the caloric responses proved that the central vestibular neurons in the brain stem and cerebellar vermis had not become involved by any tumor In other words, there was no evidence of involvement of the cerebello-vestibular interrelation

Operation An astrocytoma in the right cerebellar hemisphere was found It was close to the midline but did not involve it

As we said before, about three years ago one of us began to notice a very peculiar abnormality in the post-caloric nystagmus in patients with tumors in the midline of the posterior fossa This induced nystagmus beat in the correct direction with normal or longer than normal duration, but the oscillations of the eyes were not regular in amplitude or frequency In addition, sometimes the induced nystagmus appeared in periods of five to ten seconds, stopped for a few seconds and then reappeared, so we could see large or very large oscillations at a rather high frequency alternating with small and low frequency jerks We formerly called this abnormality, "irregular post-caloric nystagmus" As we found it only in lesions in the midline of the posterior fossa, we interpreted it as the lack of cerebellar influence over the vestibular nuclei, due to the damage of the cerebello-vestibular interrelationship Based on our clinical experience and the correlative neurosurgical findings we were able to diagnose lesions in the mid-

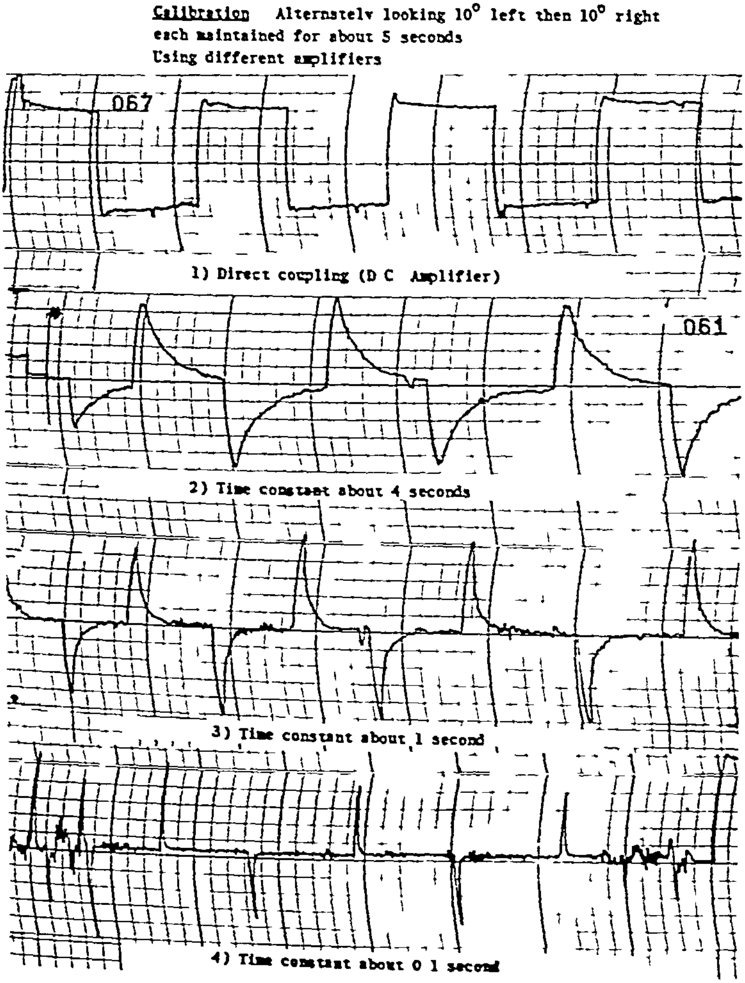


Fig 6 Calibration using different types of amplifiers The patient is asked to look to the left for about five seconds and then look to the right for a similar period The cycle is then repeated The points for visual fixation to the left and right are placed so as to subtend an angle of 20° with the patient

of the slow phase has been reduced to zero, by having the patient fix the gaze to the left for about five seconds and then fix the gaze to the right for about five seconds This brings out the effect of the time constant of the apparatus, uncomplicated by the slow phase of the normal nystagmus The error in each part of the graph will be added to the recording ular investigation, especially in the evaluation of certain difficult diagnostic problems The technique has the advantage of supplying a graphic record of the nystagmus which is available for immediate, delayed and repeated examination It also allows the important comparison and contrasting of the nystagmus with the eyes open and with the eyes closed

The method utilizes the difference in potential between the retina and the cornea of the eye In man the retina has a negative charge whereas the cornea has a positive one When the patient looks to the right the cornea (+ve) of each eye rotates to the right and the retina (–ve) moves to the left Consequently the outer canthus of the right eye will have a positive charge, and the outer canthus of the left eye will have a negative charge

This charge persists as long as the eyes are deviated but is cancelled when the eyes return to the resting position The polarity is reversed by a deviation to the left Movements of the eyes, in the horizontal plane of the outer canthi, produce a variable direct current potential If we wish to amplify and record this occurrence we should, in theory, use a D C amplifier Many difficulties arise so that the use of other types of amplifiers may be considered, but their use represents a compromise

An alternating current amplifier will amplify a change in potential but does not record the duration of the changed state A capacity coupled amplifier will record a rapid change in potential, but a slow change (as in the slow phase of the nystagmus) is not accurately amplified and recorded because of the effect of the time constant of the apparatus The shorter the time constant, the greater the inaccuracy of the recording of the slow phase, the longer the time constant, the more accurate the representation of the slow phase[19] In other words, if the apparatus has a short time constant then the recording will approximate more closely to an A C recording, whereas if the T C is longer then the recording will approximate more closely to that of a D C recording but may be attended by some of the difficulties of a true D C recording Fig 6 is an example of recordings in which the speed or ocular dysmetria will become evident by a wavering of the plateau

In some patients a good D C recording cannot be made This may be due to involuntary movements of the face or profuse sweating during the caloric test In these cases a capacity coupling may be used, but it is realized that the cause of the inaccuracy of the recording is still present but is hidden by the added effect of the time constant of the apparatus used

B Clinical Cases

Case 1 On January 9, 1959, a six-year old boy was admitted to St Louis Children's Hospital with the chief complaint of headache and vomiting of about one and one-half months' duration

In December 1958 he began to have spells of vomiting in the morning and complained of headaches These episodes did not seem to be related to meals and the headaches might occur during any part of the day but particularly at night These symptoms gradually became more frequent, and because his mother noticed that something appeared to be wrong with his eyes, they appeared somewhat sunken and dark he was seen by an ophthalmologist who noted bilateral papilledema and referred him to the hospital On physical examination he was a very pleasant, co operative little boy, who did not appear acutely ill

Examination of his cranial nerves revealed no abnormalities except bilateral papilledema with hemorrhages The functional examination of the VIIIth nerve was as follows

1 Hearing Normal in both ears

2 Equilibrium

a Romberg Unsteady but not positive

b Gait Slight unsteadiness This was noticed primarily when he turned quickly He slightly staggered to both sides and only scarcely widened his base while walking

3 Cerebellum Slight bilateral asthenia, dysmetria and intention tremor Dysdiadochocinesis was more marked on the right side

4 Nystagmus No spontaneous or positional nystagmus was observed The caloric tests showed normal vestibular excitability however, the post caloric nystagmus showed a definite dysrhythmia The oscillations of the eye globes were irregular in amplitude and frequency but apparently not in the speed of the slow component

Fig 7 shows a normal nystagmus after the conventional caloric stimulation (Hallpike's method) The nystagmus is perfectly regular in amplitude and frequency Usually this is best seen in the recording between 70" and 90" after the start of the stimulation

Fig 8 shows the dysrhythmia in the nystagmus induced by caloric testing in this patient There is a gross irregularity in the amplitude and in the frequency of the eye movements but the speed of the slow component is about the same of the nystagmus when an apparatus having each appropriate time constant is used

A D C recording is the ideal method if the difficulties can be overcome We have used this method and will discuss the difficulties encountered

1 Base line drift due to the amplifier This difficulty has virtually been eliminated by the use of Grass polygraph 5 P1 and 5 P3

2 Base line drift due to the variation of skin potential and resistance developed at the skin electrode coupling

This has been considerably reduced by *a* Carrying out the test in a chilled room to minimize the apparent and insensible sweating The history is obtained and other examinations are carried out in the same room, to allow for the cooling to take effect

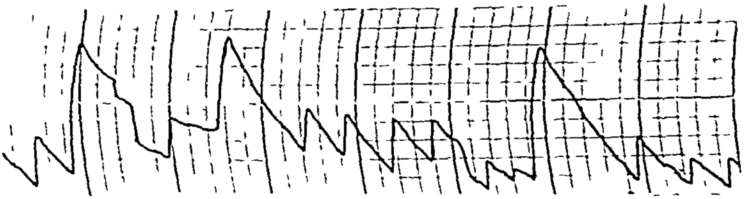
b Thorough cleansing of the skin with a grease solvent such as alcohol or ether

c Rubbing of the skin at the site of electrode contact with fine sandpaper to remove the keratinized layer and reduce skin resistance

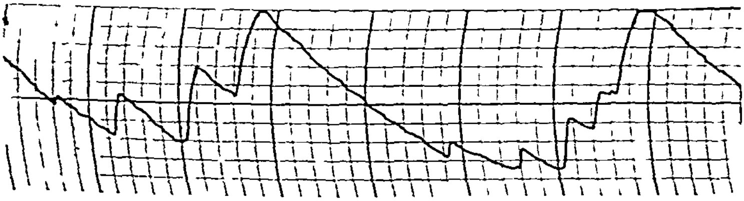
d Using calomel electrodes with 1 per cent saline on cotton as the actual contact The calomel electrodes are very small, they are easily attached by tape and are quite durable, providing a little care is exercised in their storage They consist of a small tube with a platinum wire fused into one end The inner end of the platinum wire is immersed in mercury which is then sealed in with a layer of calomel, the final layer being cotton soaked in 1 per cent saline More cotton soaked in saline is the actual contact with the skin

Calibration is carried out by asking the patient to alternate his gaze between two vertical stripes set at about ten feet distance and far enough apart to subtend an angle of 20° If each new position of the gaze during the alterations is maintained for a few seconds before reverting to the previous position, then any drift can be detected by a lack of squareness in the waves Any large defect in fixation of the gaze cystic cerebellar tumor was encountered which proved to be an astrocytoma

In his operative notes the neurosurgeon wrote "The tumor lay essentially in the midline—and at the end of the procedure when (he) tried to visualize how much of the tissue there lay between the inferior surface of the tumor and the



Nystagmus Dysrhythmia Due to a Tumor in the
Cerebellar Vermis Before Operation



Nystagmus Dysrhythmia Due to a Tumor in the
Cerebellar Vermis After Operation

Fig 8 Case 1 Dysrhythmia in nystagmus induced by calorization There is an irregularity in the amplitude and frequency of the eye movements but the speed of the slow component remains about the same in every jerk Sometimes the nystagmus stops for a few seconds and then reappears If the cause is a lesion in the cerebellar vermis the dysrhythmia increases when the patient opens his eyes

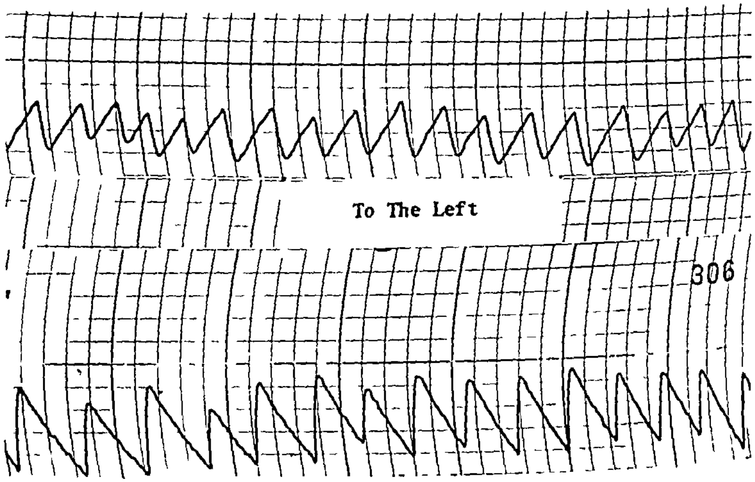
After the operation in spite of the improvement of the patient s condition the dysrhythmia became more apparent due to the added surgical damage of the cerebello-vestibular interrelation

fourth ventricle it was realized that there seemed to be only the ependymal layer left over the ventricle"

Two weeks after the operation, the functional examination of the VIIIth nerve was repeated The disequilibrium had improved and there was no asthenia or dysmetria, only a slight dysdiadochocinesis on the right side No spontaneous nystagmus was seen The positional nystagmus and postural vertigo were not tested because of the soreness of the wound

We considered this type of *abnormality* in the caloric responses to be due to a lack of cerebellar influence over the vestibular nuclei because of damage to the cerebello-vestibular interrelationship.

Our interpretation and comment on this case was the following: "From the otoneurological point of view there is no involvement of the VIIIth nerve, but a marked involvement of the cerebello-vestibular interrelationship shown by a remarkable dysrhythmia in the nystagmus induced by calorization. This sign plus the disequilibrium and the bilateral cerebellar signs indicate a lesion involving the cerebellar vermis, and the roof and lateral walls of the fourth ventricle but not its floor." Skull X-rays revealed diastasis of the coronal and sagittal sutures. The E E G revealed a slow dysrhythmia with slight left occipital predominance. On January 14, a ventriculography was performed which revealed a mass protruding into the fourth ventricle, and following this a posterior fossa craniotomy was performed. A midline



Post-Caloric Nystagmus To The Right.
Normal Response With 30° For 40"

Fig 7 Irrigation with water at 30° for 40 seconds in the ear elicits nystagmus towards the opposite side. The quick component which designates the direction of the nystagmus is recorded as an almost vertical line. In a normal subject the post-caloric nystagmus shows a good rhythm.

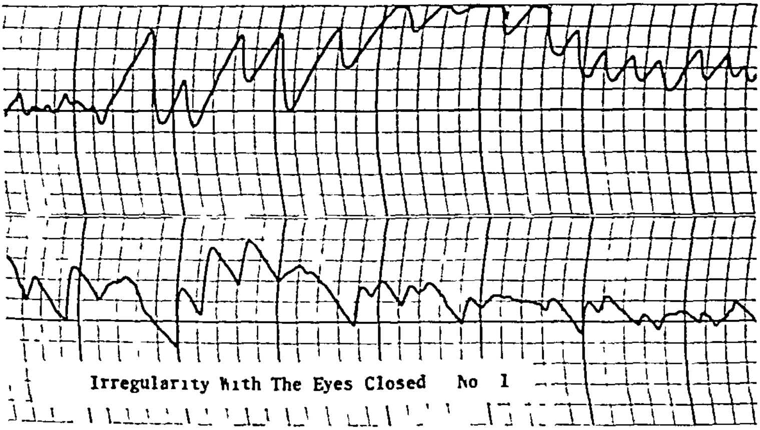


Fig 9 Case 2 October 1959 Slight dysrhythmia still present though patient had improved after therapy

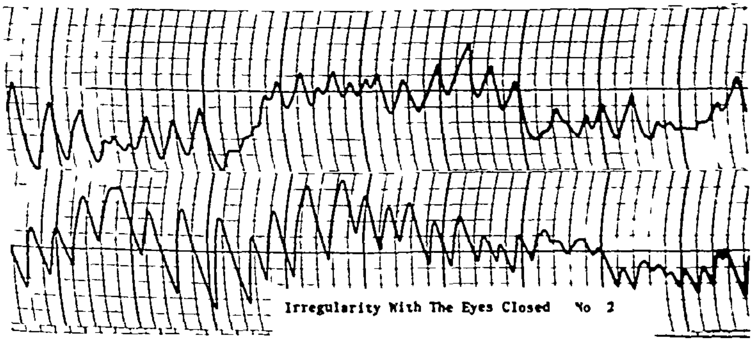


Fig 10 Case 2 February, 1960 Eyes closed Dysrhythmia more marked than previously with eyes closed

We saw the patient on February 11 1960, and found her much worse than before She had a spontaneous nystagmus in all directions of the gaze which was altered in quality by changes of head posture Type I Very slight bilateral cerebellar signs were verified, and the dysrhythmia in the post caloric nystagmus was clearly present (see Figs 10 and 11) The hearing was still normal In view of these findings the diagnosis of a central vestibular syndrome due to a lesion in the cerebello vestibular interrelation was confirmed

Later that day neuro surgical air studies were performed and a craniotomy was done The surgeon found an ependymoma and wrote " I cleaned out the tumor which had replaced the vermis and removed a large nodule which had dilated the upper part of the fourth ventricle" The tumor extended along the lateral margin of the fourth ventricle on both sides more on the right than on the left

Actually the patient had done extremely well after the operation, and the funduscopic examination revealed the discs to be flat

The post-caloric nystagmus, however, showed a greater dysrhythmia (see Fig 8) which is easy to understand if we realize that the neurosurgeon had dissected to the roof of the fourth ventricle in order to extirpate the tumor

Case 2 In October, 1959, a 38 year old woman was admitted to the medical department of Barnes Hospital, St Louis Six weeks prior to admission she had been well but developed a severe occipital headache which lasted an hour, leaving her "light headed" She became dizzy and nauseated on lying down, especially on the right side This improved after a few days and she was well for the next four weeks, then the nausea returned in certain positions of the head, resulting in vomiting She had had this for about one week when she was admitted There were no hearing problems

Functional Examination of the VIIIth Nerve

1 Hearing tests Normal thresholds for pure tones and speech with normal discrimination

2 Equilibrium

a Romberg Slightly unsteady but not positive

b Gait A little unsteady becoming a little worse with the eyes closed

3 Cerebellum No lateral cerebellar signs

4 Positional nystagmus and postural vertigo

There was no spontaneous nystagmus There was slight vertical (downward) nystagmus with the head extended (hanging) and also with the head over the right shoulder There was marked vertigo and the nystagmus persisted until the patient vomited Type 2

5 Caloric tests

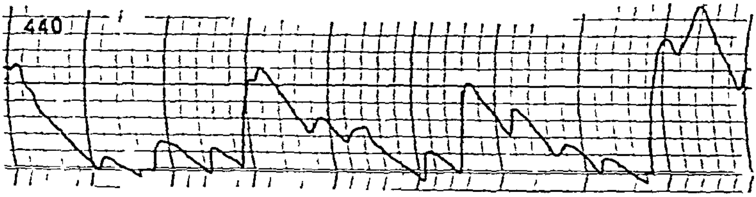
The nystagmus was within normal limits as regards amplitude and duration, however, there was a marked dysrhythmia

Comment From the otoneurological point of view there is no involvement of the VIIIth nerve The cochlear and vestibular excitability is normal There is a central vestibular syndrome characterized by *1* Postural vertigo with vomiting and positional nystagmus Type II *2* Dysrhythmia in the post caloric nystagmus and *3* disequilibrium

In view of this one of us (M H S) reported that "the findings suggest a midline cerebellar lesion" The patient was treated by intravenous fluids to correct an ionic imbalance The patient improved very quickly Three days later there was no postural nystagmus, but there was still a little postural vertigo The dysrhythmia was much less but still present (see Fig 9) There were no other neurological signs to suggest a posterior fossa lesion and the patient was discharged home

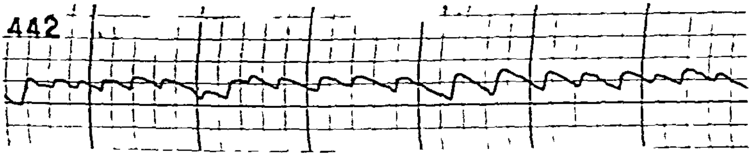
She improved for several weeks but was still a little unsteady She then developed increasing headache with weakness and was readmitted to hospital with bilateral papilledema equilibrium normal cerebellar function, no spontaneous or positional nystagmus and normal vestibular excitability The caloric tests however, showed a marked dysrhythmia in the nystagmus (see Fig 12) There was a marked irregularity in the amplitude and frequency of the eye oscillations only when they were closed, but as soon as they were opened the dysrhythmia disappeared, and the nystagmus became perfectly rhythmic (see Fig 13)

Our previous experience with electronystagmography had taught us that the spontaneous slow swinging movement of the eyes could simulate a dysrhythmia in nystagmus induced by calorization and we also knew



Dysrhythmia present when the eyes are closed

Fig 12 Dysrhythmia due to the superposition of the nystagmus induced by caloric stimulation on spontaneous eye movements



Dysrhythmia disappears when the eyes are open

Fig 13 The dysrhythmia not caused by a lesion in the cerebello-vestibular connections disappears when the patient opens her eyes

that this disappeared when the patient opened the eyes because of fixation of the gaze

The perfect equilibrium and the absence of neurological symptoms in our patient did not support the possibility of a lesion in the cerebello-vestibular interrelation We thought therefore that the only explanation of the dysrhythmia would be the existence of spontaneous slow swinging of the eyes when closed Next we ran the electronystagmograph without caloric stimulation in order to detect eye movements Spontaneous large and slow swinging movements were recorded (see Fig 14) which vanished when the patient opened her eyes (see Fig 15)

We attributed the eye movements to the daily intake of phenobarbital

We consider the dysrhythmia in nystagmus induced by the

DISCUSSION

The purpose of this paper is to emphasize dysrhythmia in the post-caloric nystagmus as a possible clinical sign of damage to the cerebello-vestibular interrelation by a tumor or degenerative lesion of the midline of the cerebellum

Dysrhythmia may also be found in normal subjects who exhibit spontaneous large and slow swinging movements of the eyes when closed Aschan[2] has described two types of normal eye movements One consists of small irregular eye movements which do not interfere with post-caloric nystagmus, and the other consists of slow swinging movements, in which each swing lasts several seconds The super-position of the induced nystagmus over the swinging movement can simulate a dysrhythmia We believe that the following case clearly illustrates this point

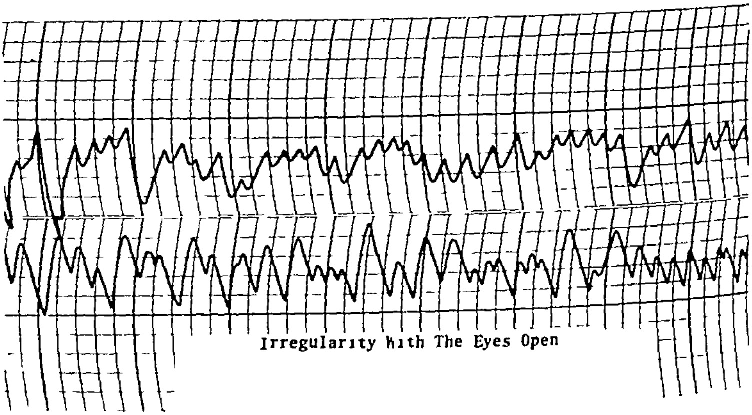


Fig 11 Case 2 February 1960 Eyes open Dysrhythmia more marked than with eyes closed

A 36 year old woman was sent to us on May 1, 1959, from the Neurological Department Washington University, St Louis for functional evaluation of the VIIIth nerve because she was complaining of hearing loss and dizziness She had recently had a few mild epileptic seizures The E E G showed a focus in the left temporal lobe Some of her relatives were epileptic For five months she had been under treatment with Dilantin® (0 1 gm 3 times a day) and phenobarbital (100 mg at bedtime daily) and during this time she had been free of attacks

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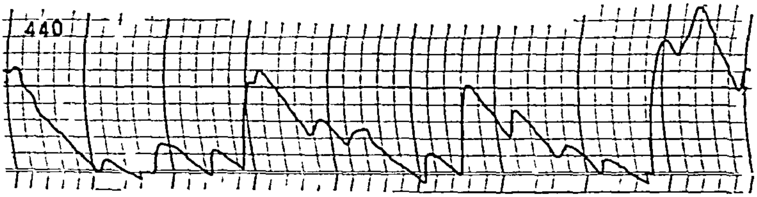

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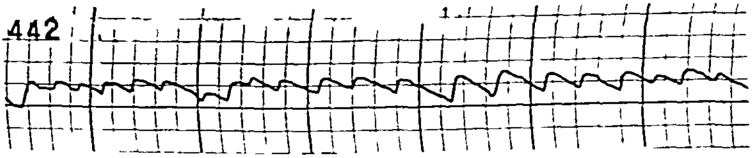
Fig 12 Dysrhythmia due to the superposition of the nystagmus induced by caloric stimulation on spontaneous eye movements



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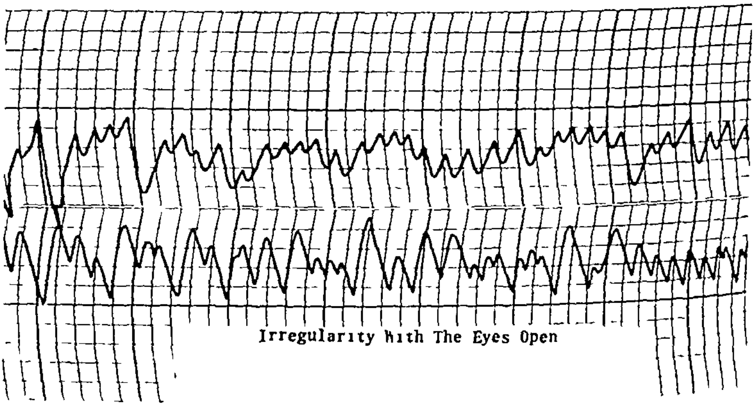

Fig. 11. Case 2. February 1960. Eyes open. Dysrhythmia more marked than with eyes closed.

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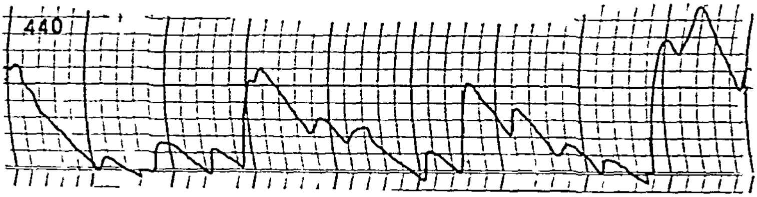
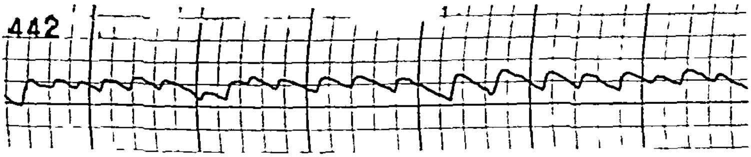


Dysrhythmia present when the eyes are closed

Fig 12 Dysrhythmia due to the superposition of the nystagmus induced by caloric stimulation on spontaneous eye movements



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We attributed the eye movements to the daily intake of phenobarbital

We consider the dysrhythmia in nystagmus induced by the

caloric tests as an otoneurological sign of a destructive lesion in the cerebello-vestibular interrelationship We have verified it in medulloblastomas, astrocytomas, ependymomas, and recently in an advanced case of multiple sclerosis (see Fig 16) which, therefore, must be considered in the differential diagnosis Of course, it also may be present in other lesions

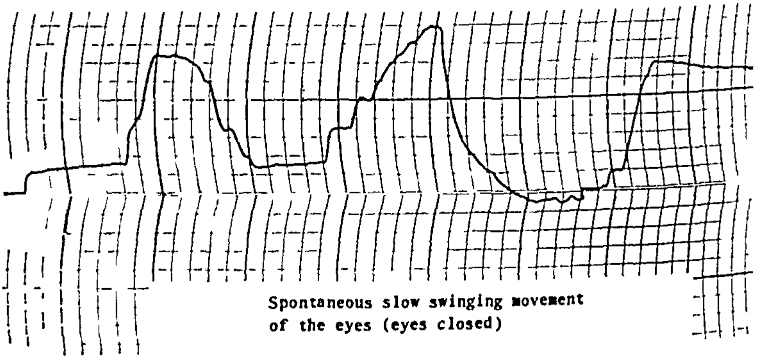


Fig 14 Eyes closed Large slow swinging of the eyes attributed to daily intake of phenobarbital

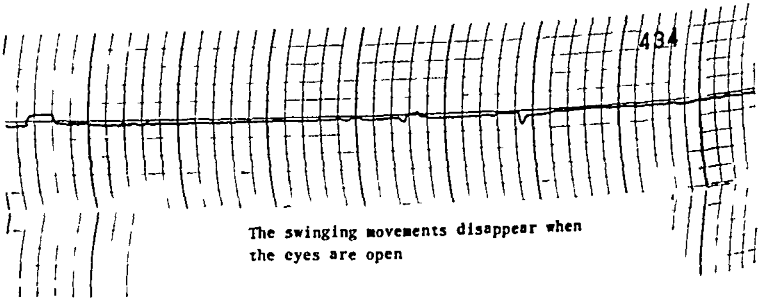


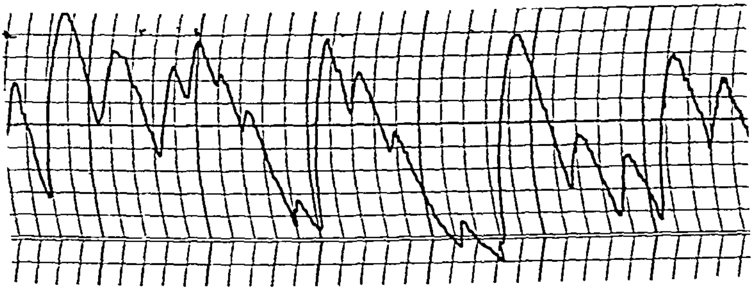
Fig 15 Eyes open Swinging movements disappeared due to eye fixation

involving this structure Dysrhythmia resulting from the superposition of the induced nystagmus over spontaneous movements of the eyes must be excluded

If the cause is a lesion in the midline structures in the posterior fossa, the dysrhythmia will be present with the eyes closed and open In addition, electronystagmography has shown us that the dysrhythmia may be accentuated with the eyes open

If the cause is the slow swinging of the eyes which is occasionally encountered in normal individuals, the dysrhythmia will be present only with the eyes closed, and it will disappear with the eyes open, because the swinging movement stops with fixation of the gaze

Another source of error in the evaluation of this sign is the inhibition of the nystagmus with the eyes open due to voluntary fixation of the visual axis in order to avoid the unpleasant sensation of objective displacement Any dysrhythmia due to variation of the patient's effort at fixating the visual axis must be excluded, but this variation would occur only with the eyes open Irregular movements due to local lesions in the orbit also must be differentiated



Nystagmus Disrhythmia Due To Multiple Sclerosis

Fig 16 This patient had a very marked disequilibrium she could scarcely walk she had bilateral celebellar symptoms had spontaneous nystagmus in several directions of the gaze and Type I positional nystagmus (non-paroxysmal) The vestibular excitability was normal but the post-caloric dysrhythmia evidenced a lesion in the cerebello-vestibular connections

SUMMARY

1 The clinical evaluation of the vestibular system is based specifically on the examination of the nystagmus

2 A brief account of spontaneous nystagmus positional

nystagmus, and abnormalities in the post-caloric nystagmus is given

3 One of these abnormalities is the dysrhythmia in nystagmus induced by calorization which has been consistently verified in patients with tumors in the midline structures in the posterior fossa

4 We believe that the post-caloric dysrhythmia is produced by damage to the cerebello-vestibular interrelationship This consists of the afferent and efferent connections between the cerebellum and the vestibular system which mainly course on the lateral and superior walls of the fourth ventricle (medial aspect of the restiform body and roof of the ventricle) Through these ascending and descending fibers the cerebellum exerts its influence (inhibition, tonus, coordination) on the vestibular responses Its destruction by tumors or disease diminishes or suppresses this influence and results in dysrhythmia in the induced vestibular nystagmus

5 Electronystagmography occasionally may show dysrythmia in normal subjects with spontaneous swinging eye movements when the eyes are closed The superposition of the induced nystagmus over the swinging movement might simulate a dysrhythmia The differential diagnosis, however, is easily made The spontaneous swinging movements seen with the eyes closed disappear as soon as the patient opens his eyes, due to fixation of the gaze, therefore, if the dysrhythmia is due to such movements, it will disappear as soon as the eyes are opened On the other hand, if it is caused by a lesion in the midline structures in the posterior fossa, the dysrhythmia will be present with the eyes closed and open Apparently with the eyes open it is even larger

ACKNOWLEDGMENTS

We are greatly indebted to Dr Theo E Walsh, in whose department this work was largely carried out, to Dr Cesar Fernández of the University of Chicago for the drawings of the vestibulo-cerebellar pathways as well as for his help in the neuroanatomical description of the cerebello-vestibular interrelation, to Dr Leonard Furlow and Dr William Cove

of the Neurosurgical Department of Washington University, St Louis, Mo , and to Dr Reinaldo Poblete of the Neurosurgical Institute, Santiago, Chile, who allowed us to study some of the cases presented here

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PSEUDO-OTOSCLEROSIS[*][†]

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Los Angeles, Calif

The patient with an intact and mobile tympanic membrane, patent Eustachian tube, negative Rinné, and excellent air-bone gap is usually correctly called an otosclerotic, but he may not have otosclerosis at all

A number of conditions can occur which will mimic otosclerosis Thus "Pseudo-otosclerosis" is not a clinical entity but a concept which must constantly be in the mind of the otologist who plans a tympanotomy for a case of supposed otosclerosis

Under the heading of "Pseudo-otosclerosis," I have obviously not included many other conductive lesions involving tympanic disease in which there may occur tympanic perforations, otorrhea, polyposis, and other signs of active tympanic disease In order to qualify as a "pseudo-otosclerotic" an ear must have an intact mobile tympanic membrane, a significant conductive hypacusis, and all of the other criteria described above The vast majority of these cases are unilateral While unilateral true otosclerosis certainly occurs, the differential diagnostic study should be especially searching in unilateral cases

In presenting this concept for consideration it is really the intention of the author to clarify the differential diagnosis of so-called clinical otosclerosis The following groups of cases and their illustrative examples are by no means intended to constitute a complete compendium of all such diagnostic possibilities There are other examples which could be added to this list, but this group consists only of those significant types of cases which have come to the attention of the writer

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[*] Read at the meeting of the Western Section American Laryngological, Rhinological and Otological Society Inc Denver Colo Jan 22 1960

[†] From the Department of Otolaryngology Cedars of Lebanon Hospital and The Western Foundation for Hearing Research

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and his colleagues In this presentation, each clinical group will be illustrated by only one clinical case record in most instances, although in some of the categories multiple examples are present in our files

In classifying the various conditions which can mimic clinical otosclerosis, it was decided to group them in an essentially anatomical manner since all of these conditions produce similar physiological sequellae

I Total Ossicular Chain Fixation

- *A* Chronic glue ear with adhesive fibrosis
- *B* Granulomatous otitis
- *C* Diffuse fibrosis
- *D* Tympano-sclerosis
- *E* Pan-osteo-arthritis of ossicular chain

II Diseases of the Incus

- *A* Traumatic dislocation of the incus
- *B* Fixed incus
  - *1* Incus annulus fusion
  - *2* Incudo-malleolar fusion with cholesteatoma
- *C* Incus atrophy
- *D* Lenticular process necrosis

III Non-Otosclerotic Diseases of the Stapes

- *A* Paget's disease
- *B* Osteogenesis imperfecta
- *C* Tympano-sclerosis
- *D* Footplate arthritis
- *E* Peristapedial tent
- *F* Congenital fixation

IV Anomalies of Ossicles and Windows

I TOTAL OSSICULAR CHAIN ANKYLOSIS

Among the sequellae of chronic otitis media there is universal awareness of the dramatic problems of perforations, suppurations, cholesteatomata, polypi, etc The apparently innocuous sequel of diffuse fibrosis rarely produces symptoms unless its severity immobilizes the ossicular chain In such instances a conductive hypacusis ensues Such an involvement may cause hearing losses from 10 db to 60 db Obviously, when the loss reaches 35-40 db it becomes significant otologically in the differential diagnosis of otosclerosis

A number of forms of diffuse fibrosis may occur of which three different examples are presented

In addition, other lesions can cause total ossicular chain ankylosis Tympano-sclerosis, possibly a special type of fibrosis involving the tympanic muco-periosteum is exemplified by a case Pan-osteo-arthritis of the ossicular chain can also completely immobilize it and simulate far advanced otosclerosis This is illustrated by a case

A Chronic Glue Ear with Adhesive Fibrosis

Serous effusion of the middle ear has become a very serious cause of conductive hearing loss and the frequency of this condition is undoubtedly increasing One of the final sequellae of an apparently innocent serous effusion is the so-called glue ear in which very thick exudate of almost solid consistency fills the middle ear This is another variant of chronic low grade otitis media

In some cases the glue ear may persist for many, many years, and may be accompanied by co-existent tympanic fibrosis In many cases it undoubtedly is a precursor of total tympanic fibrosis, however, in some cases it is possible for both glue-like fluid and the fibrotic tissue to co-exist and to contribute in a parallel way by ossicular fixation and window blockade to a major conductive hypacusis

Case 1 L K, a 30 year old white female, gave the history of a hearing loss in the left ear since early childhood She denied any attacks of otitis media and denied any myringotomies or any type of therapy to the left ear Examination revealed that the left tympanic membrane was intact but somewhat dull in appearance and slightly retracted It was, however, mobile and there was evidence of good tubal function on politzerization No definite fluid could be seen although a suggestion of fluid appeared on several examinations X rays of the mastoids revealed a definitely sclerotic and infantile left mastoid There was a questionable history of family deafness There were no other significant findings except for low grade chronic tonsillitis

Audiometric examination revealed a marked left conductive hypacusis

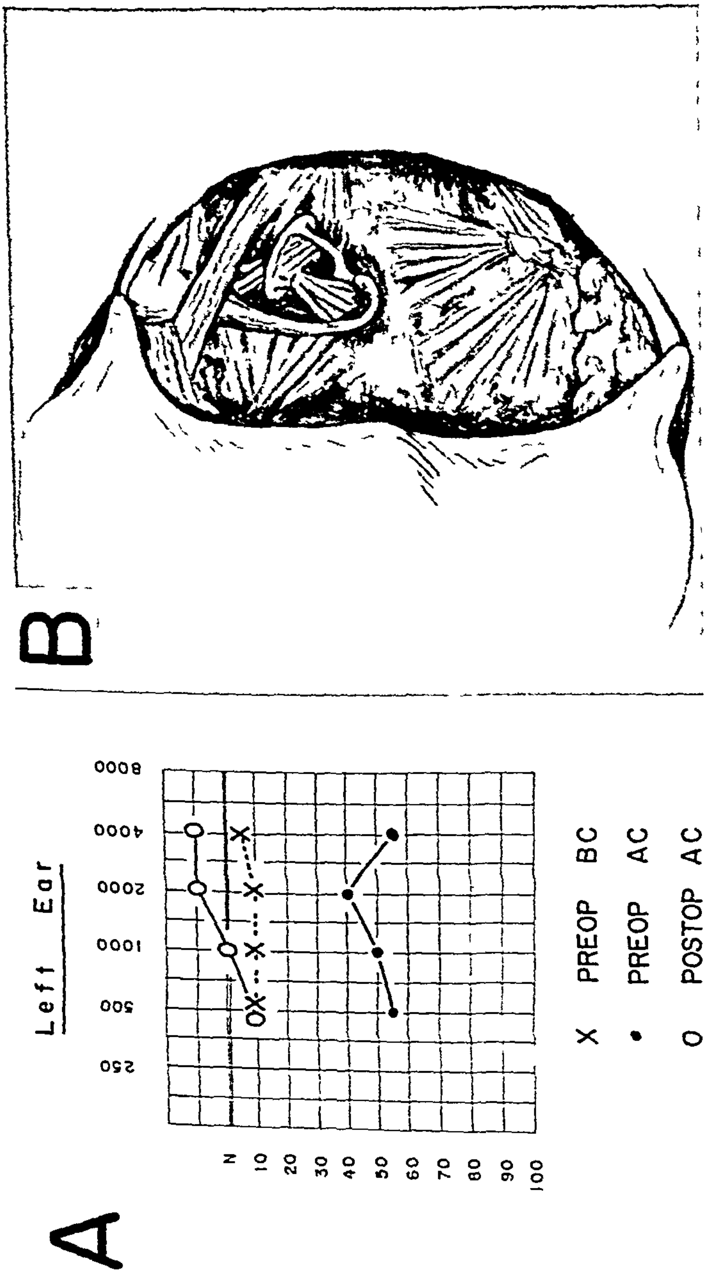

Fig 1 Chronic glue ear with adhesive fibrosis A—Audiogram B—Tympanotomy findings

Tuning fork tests showed a negative Rinné on the left with the Weber referred to the left

A left exploratory tympanotomy revealed a moderate amount of thick glue like secretion in the hypotympanum, accompanied by large bands of scar tissue with one very dense fan shaped band between the incus and the annulus A thick fibrous band filled the round window niche The stapes was quite firmly fixed by bands of scar tissue between the neck, crura, and the promontory The footplate was mobile Following removal of the glue secretion and dissection of all of the bands of scar tissue her hearing returned completely to normal on the operating table There was no evidence of otosclerosis and an excellent round window reflex was obtained on palpation of the stapes (see Fig 1)

B *Granulomatous Otitis*

The formation of granulomata and granulomatous polypi is not a rare aspect of the pathologic course of chronic otitis media In most cases such granulomatous otitis is accompanied by similar changes in the mastoid antrum and periantral cells It is possible for such changes to represent only a transitional stage in the progression toward ultimate irreversible fibrosis, however, this granulomatous phase may continue for a long time, undoubtedly for years and years before fibrosis takes place When the granulomatous process is sufficiently extensive, it may create a mechanical impedance involving the entire ossicular chain and produce a conductive hypacusis as is illustrated in the case below

Case 2 G R This 42 year old white male gave the history of progressive hearing loss in the right ear of six years' duration There was also a history of deafness in his paternal relatives

The middle ear inflated well on politzerization The Rinné response was negative, and the Weber was referred to the right side Audiometric studies showed a right conductive hypacusis There were no significant abnormalities in the left ear There was a moderate deflection of the septum to the right and there was evidence of a low grade chronic tonsillitis Roentgen studies showed a normal well pneumatized left mastoid The right mastoid was mixed, pneumatic and diploic, and there was diffuse haziness in the pneumatic areas

On exploration of the right middle ear via tympanotomy under local anesthesia the middle ear was found to be completely filled with thick granulomatous polypi in which the ossicular chain was completely embedded and immobilized Following meticulous removal of the granulomatous tissue the ossicular chain returned to normal mobility The stapes was found to be completely normal and mobile following stripping of the granulomata from the crura

In spite of visible return of mobility to the ossicular chain there was no demonstrable improvement in hearing audiometrically, however within two weeks he obtained marked improvement of hearing which has persisted (see Fig 2)

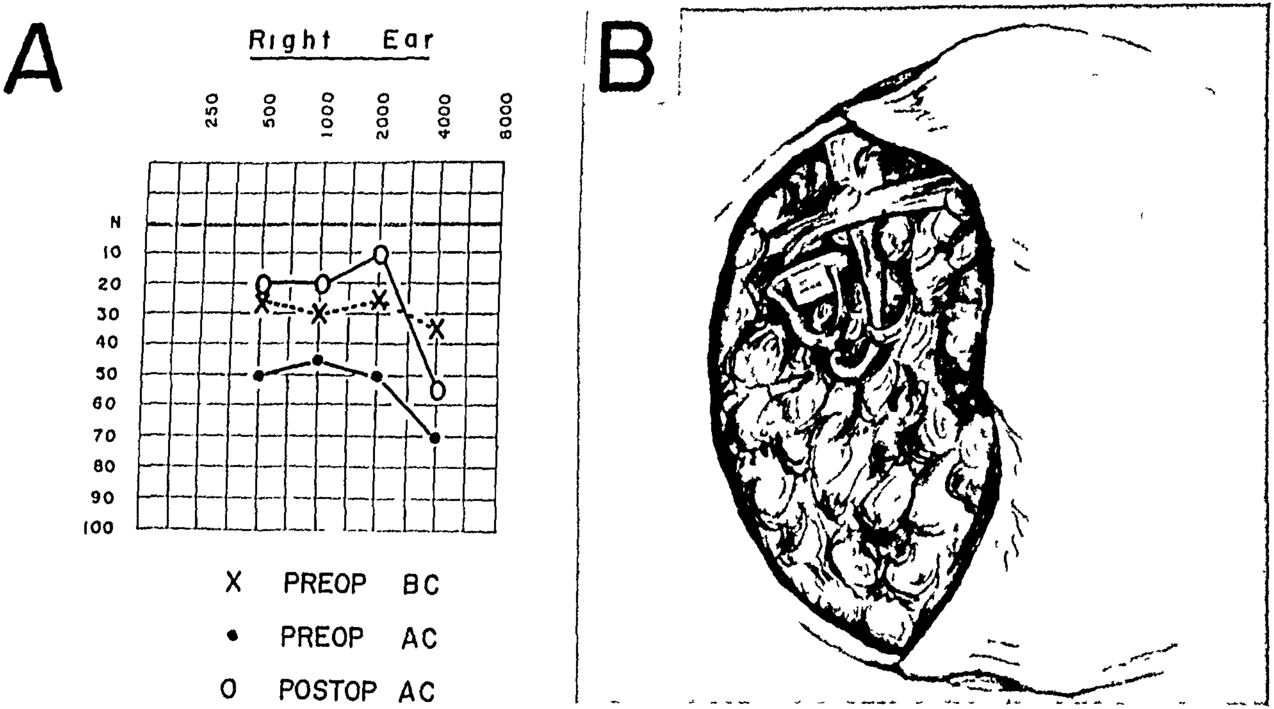

Fig 2 Granulomatous otitis A—Audiogram B—Tympanotomy findings

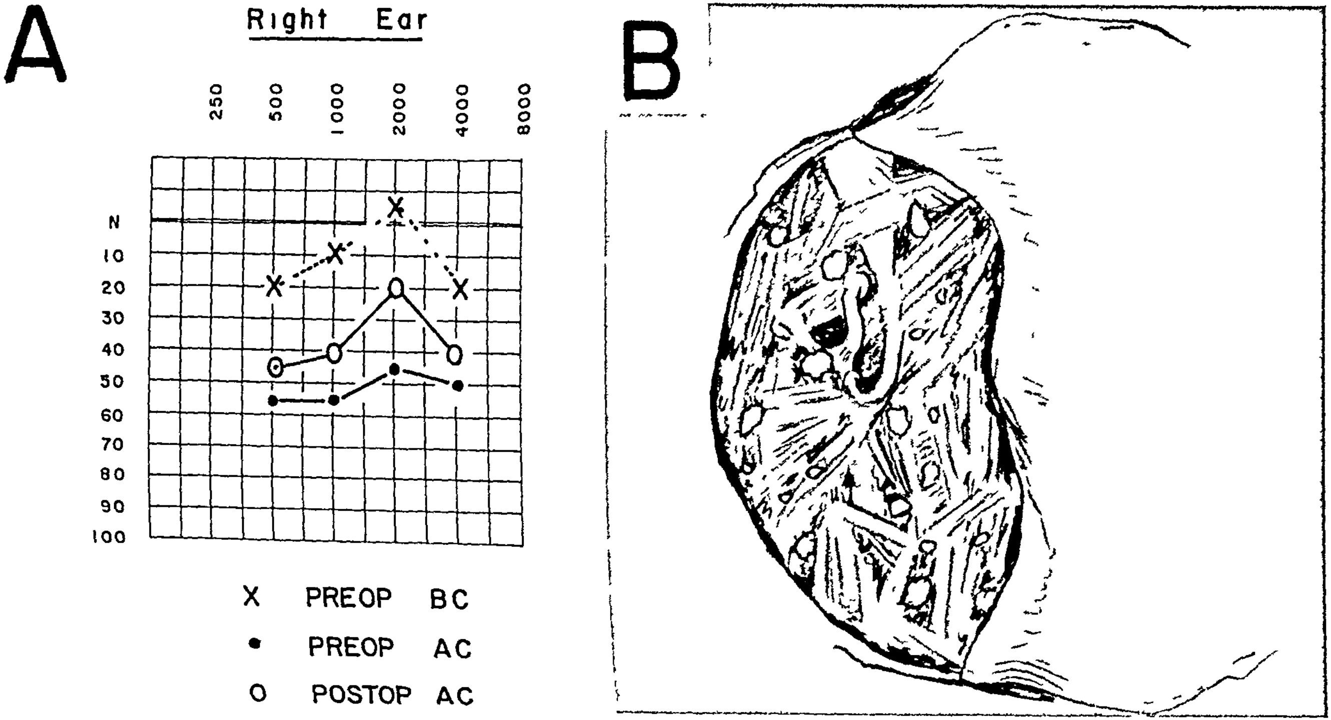

Fig 3 Diffuse fibrosis A—Audiogram B—Tympanotomy findings

C Diffuse Fibrosis

The most common version of chronic otitis media in the presence of an intact tympanic membrane is the slow development of a diffuse pan-fibrosis Such fibrotic changes can effectively fix the entire ossicular chain as is demonstrated in the following case

Case 3 J O a 29 year old white male gave the history of recurrent ear infections in infancy and was aware of a major bilateral hearing loss since early childhood There was no familial history of deafness

Examination revealed both tympanic membranes to be intact and moderately scarred but mobile on pneumatic massage Both Eustachian tubes were patent on politzerization Both Rinné responses were negative There were no significant nasal or pharyngeal findings X rays revealed infantile non pneumatized mastoids bilaterally

A right exploratory tympanotomy revealed the middle ear completely filled with diffuse fibrotic scars The incus was bound anteriorly and posteriorly by dense scars and the same types of scars were present in the peri footplate regions With great difficulty all of the visible bands of scar tissue were dissected free following which palpation revealed a perfectly mobile stapes and following which there was a marked improvement in hearing This improvement, however did not persist and an audiogram six weeks postoperatively showed only a moderate improvement in hearing (see Fig 3)

D Tympano-sclerosis

Tympano-sclerosis, a recently described type of chronic otitis media, is not a rare finding in tympanoplastic surgery Onion-like scales of keratin seem to grow throughout the tympanic cavity and a keratinization and hyalinization process may involve both mucosa and periosteum It may further involve the bone of the otic capsule and may destroy any or all of the ossicles It is most commonly diffuse in the tympanic cavity and thus produces a total ossicular fixation as is described in the following case

Case 4 J L a 52 year-old white female, had a history of recurrent bilateral otorrhea since childhood following scarlet fever Examination revealed intact but scarred tympanic membranes bilaterally The left tympanic membrane was moderately mobile and showed a calcified plaque anteriorly The Eustachian tube function was normal on politzerization The Rinné response was negative and audiometry showed a typical conductive hypacusis X rays showed acellular undeveloped infantile mastoids bilaterally

A left exploratory tympanotomy revealed a middle ear filled with scar tissue bands which were attached to onion like lamellae of sclerotic tissue covering the manubrium of the malleus the long process of the incus and the stapedial crura Other lesions were present over the facial nerve and seemed to involve the otic capsule throughout the promontory region and throughout the region of the niche of the round window There was total fixation of the ossicular chain and in view of these findings no definitive surgical procedure was carried out There was no improvement in hearing (see Fig 4)

E Pan-Osteo-Arthritis of Ossicular Chain

Among the lesions responsible for stapes ankylosis is a true osteo-arthritis, however, it is possible for such an arthritis to involve the entire chain and to create not only a fixed stapes but a fixed incudo-malleolar joint and a fixed incudo stapedial joint as is exemplified in the following case

Case 5 P G, a white male, aged 63, gave the history of a bilateral hearing loss since childhood which continued to become progressively worse, requiring the use of a hearing aid during the past 38 years There is a very strong family history of otosclerosis

Examination revealed bilateral intact tympanic membranes Both were mobile and both Eustachian tubes were patent on politzerization Both Rinné responses were negative There were no significant nasal or nasopharyngeal findings with the exception of moderate low grade chronic tonsillitis X rays revealed well pneumatized mastoids bilaterally, but both sides showed equal periantral sclerosis

Audiometric studies revealed evidence of a very profound bilateral mixed deafness with a major neural component There was, however, a persistent negative Rinné response to the lower tuning forks bilaterally A diagnosis of far advanced bilateral otosclerosis with cochlear degeneration was made and a right exploratory tympanotomy was advised, with a very poor prognosis

A right tympanotomy exploration revealed that the incus was completely fixed by incudo malleolar fusion The incudo stapedial joint was ossified Both crura were attached to the promontory by bony union and there was a marked anterior peribasal ankylosis involving a good deal of the anterior crus and footplate The long process of the incus was amputated and the stapes crura were removed A polyethylene tube was placed between the posterior aspect of the footplate and the medial aspect of the tympanic membrane following which the posterior one-third of the footplate was mobilized by chisel cuts There has been no significant gain in hearing (see Fig 5)

II DISEASES OF THE INCUS

A number of conditions involving the incus exclusive of the stapes can produce major conductive hypacuses simulating otosclerosis These may include traumatic dislocation of the incus, fixed incus, incus atrophy, and lenticular process necrosis Each of these may occur in a number of forms of which only major examples will be cited

A Traumatic Dislocation of the Incus

While it is certainly possible for otosclerosis to develop in

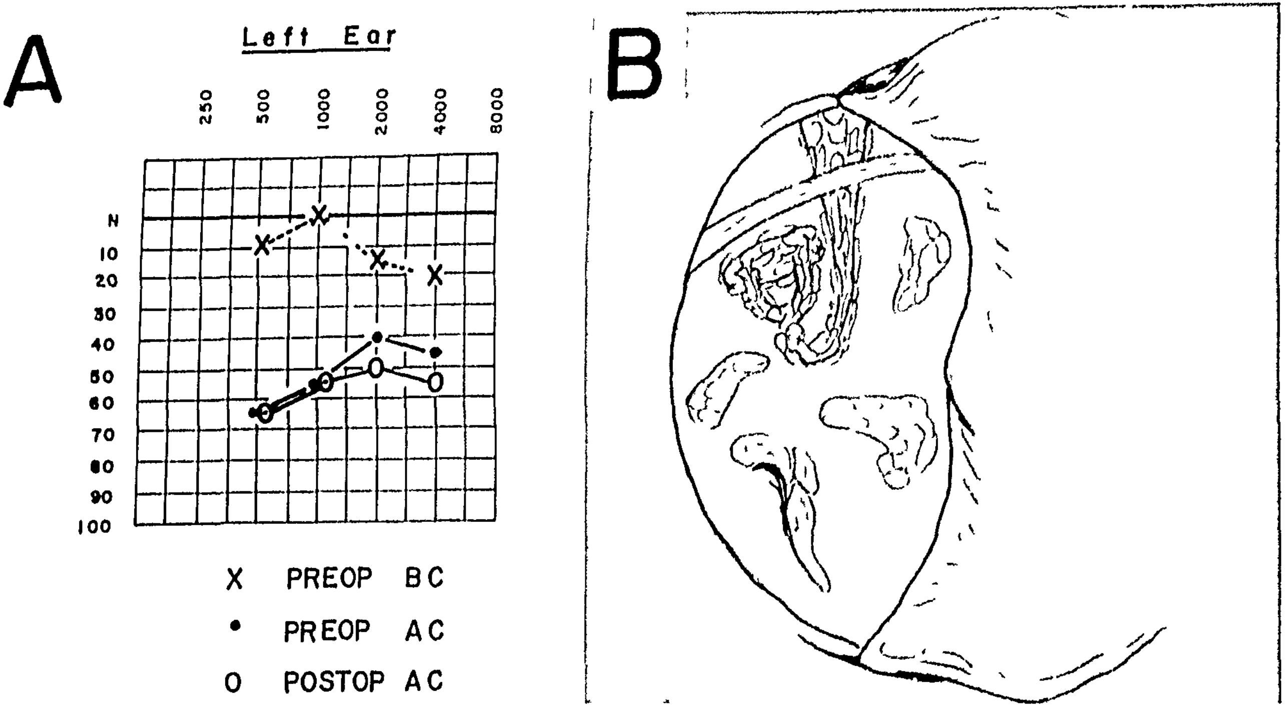

Fig 1 Tympanosclerosis diffuse A—Audiogram B—Tympanotomy findings (Note tympanosclerotic lesions on the incus stapes and on diffuse areas of the otic capsule)

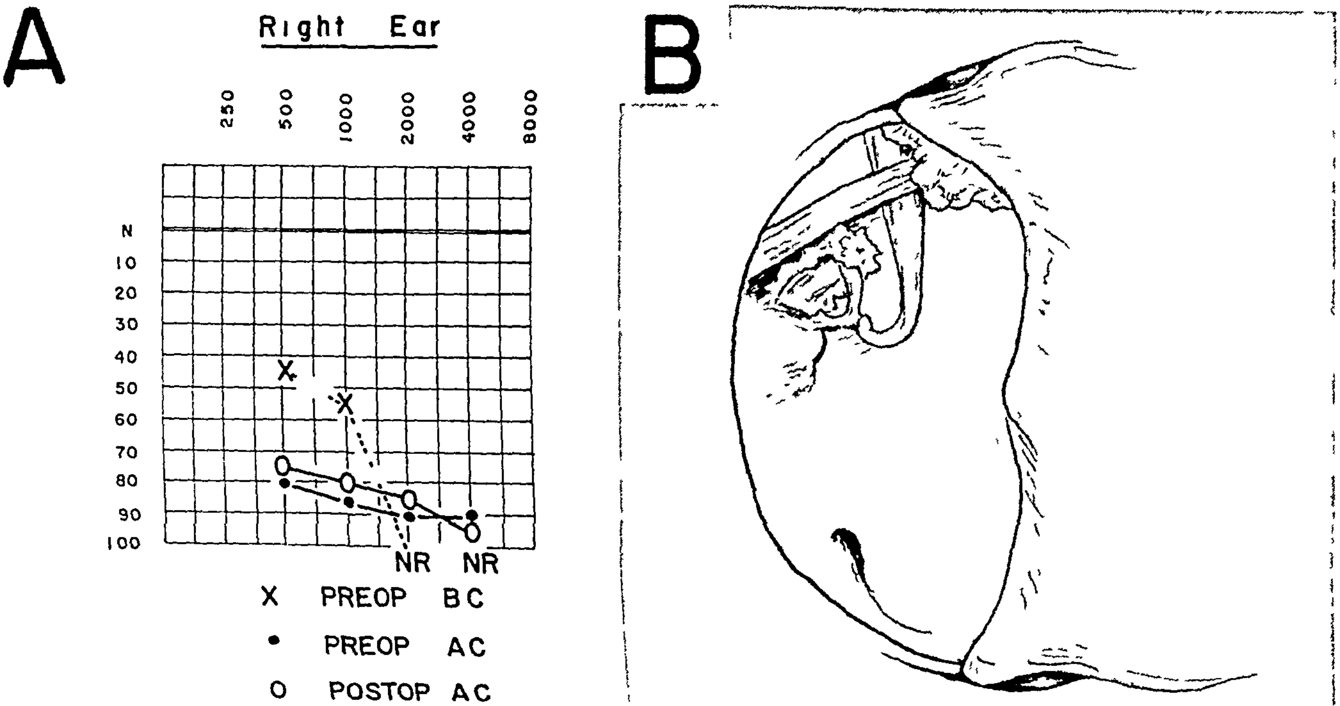

Fig. [illegible] I[illegible]n-osteo-arthritis of ossicular chain A—Audiogram B—Tympanotomy findings an ear which had been the seat of acute otitis media and acute mastoiditis in infancy, the otologist should be wary of the presumptive otosclerotic who gives a history of an infantile mastoidectomy and who shows a small postauricular scar Not infrequently such a supposed otosclerotic has a perfectly mobile stapes and a traumatically dislocated incus It is not unusual for this to have occurred bilaterally

Case 6 I L This 28-year-old white male gave a history of a bilateral mastoidectomy at the age of nine months following otitis media secondary to scarlet fever He has been aware of a hearing loss since childhood and has worn a hearing aid for many years

Examination revealed bilaterally intact tympanic membranes Each tympanic membrane showed moderate scarring and small calcified plaques but both were mobile and both ears showed evidence of good tubal function There was no fluid in either ear Small healed postauricular incisions were present bilaterally Both Rinné responses were negative and audiometric examination revealed evidence of bilateral conductive hypacusis X rays surprisingly revealed bilaterally normal and extensively pneumatized mastoids

Right exploratory tympanotomy revealed a somewhat atrophic incus in the epitympanum with no contact with either malleus or stapes It was removed The stapes was perfectly mobile and normal in every respect A polyethylene strut was placed on the capitulum of the stapes and allowed to contact the posterior half of the tympanic membrane There was an immediate improvement in hearing which has persisted (see Fig 6)

B *Fixed Incus*

The incus can become fixed in a number of ways, and in each situation it will result in immobilization of the ossicular chain and a conductive hypacusis regardless of a mobile stapes and a normal round window membrane Two examples of fixed incus conditions will be presented

1 *Incus Annulus Fusion*

In tympanic fibrosis of long duration eventual osteogenetic activity supercedes the fibrosis in many cases

Case 7 I L a 65-year-old white female with diabetes under control presented herself with a bilateral conductive hypacusis The right tympanic membrane was intact translucent and mobile but showed several small calcified plaques The right Eustachian tube was patent on politzerization X rays revealed bilaterally infantile non pneumatized mastoids

Exploratory tympanotomy revealed a middle ear with tremendously thickened mucosa and large strands of fibrous tissue between the three ossicles the tympanic membrane and the promontory The incus was completely immobilized by a firm bony bridge between it and the postero superior aspects of the annulus The stapes appeared to be perfectly

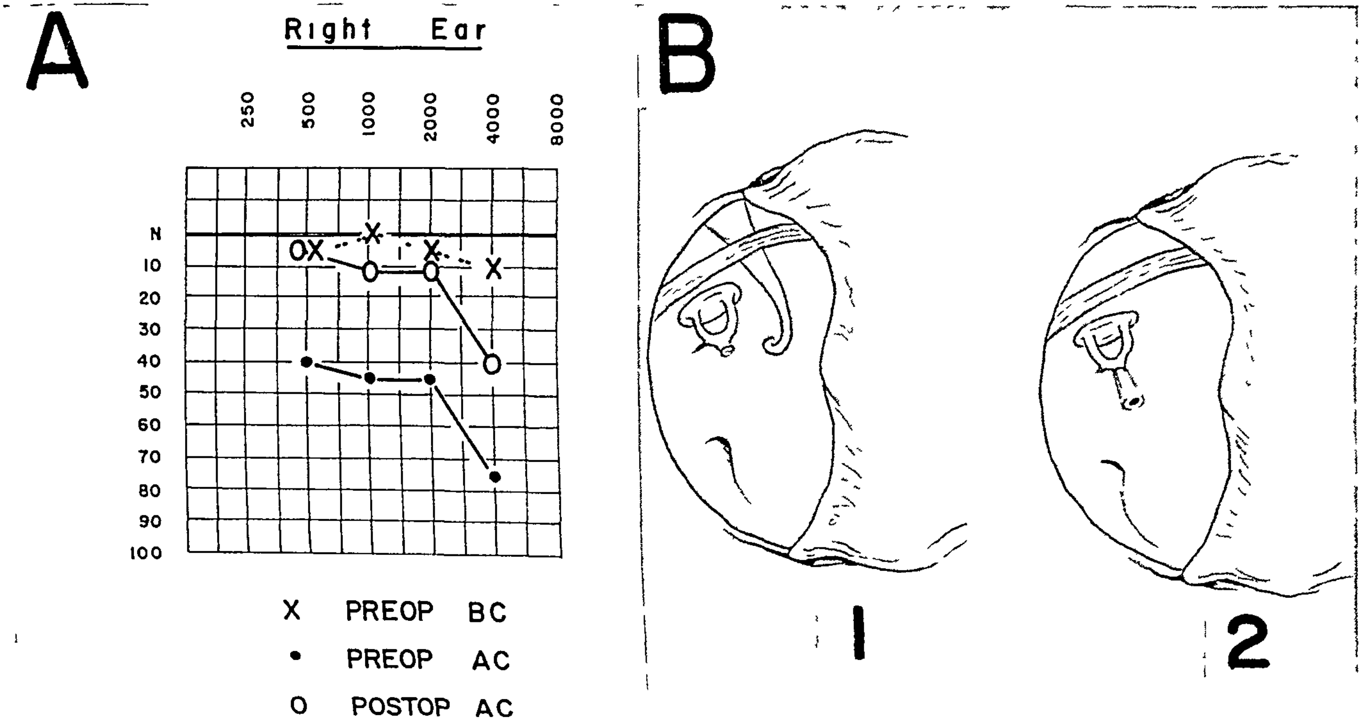

Fig 6 Traumatic dislocation of the incus A—Audiogram B—Tympanotomy findings 1 Note discontinuity between dislocated incus and stapes 2 Polyethylene prosthesis on stapedial capitulum

mobile An attempt was made to resect the bony bridge between the incus and the annulus but this attempt failed even after extensive annulus removal Accordingly, the lenticular process and a portion of the long process of the incus were removed and a myringostapediopexy was performed by placement of invaginated tympanic membrane onto the stapedial capitulum This was followed by marked improvement in hearing (see Fig 7)

2 *Incudo-Malleolar Fusion with Cholesteatoma*

It is an old laryngological cliche that a patient with hoarseness due to carcinoma of the larynx can also have tuberculosis and syphilis of the larynx It is similarly true that a patient who has otosclerosis can have other otologic diseases such as illustrated in the case below

Here we are dealing with a case of bilateral progressive deafness in which there was a marked otosclerotic lesion on the left side, however, the left ear also contained a silent hidden cholesteatoma which had produced incudo-malleolar fusion The fixed incus on this side added to the impedance problem, and the solution will be described below

Case 8 A L a 36 year old white male gave a history of progressive bilateral hearing loss of 20 years duration He had no history of otitis media and gave no family history of deafness

Examination revealed that both tympanic membranes were intact, moderately scarred and somewhat fibrotic with slightly impaired mobility Both Rinné responses were negative and the audiometic studies revealed a bilateral conductive hypacusis and an approximate 55 db loss in each ear with excellent bone conduction There were no significant nasal or nasopharyngeal abnormalities X rays revealed bilaterally diploic mastoids with moderate bilateral periantral sclerosis

Left exploratory tympanotomy revealed a completely fixed incus and a solitary epitympanic monolocular cholesteatoma with extension into the attic The incus was fixed because of incudo malleolar fusion The stapes was also fixed by a fairly large anterior otosclerotic tumor Because of the multiple problems no definitive surgical procedure was performed on this ear at this stage At a subsequent date an endaural left radical mastoidectomy was carried out with removal of the cholesteatoma and with the creation of a classical fenestra in the horizontal semicircular canal The fenestra was closed by a split thickness graft A satisfactory gain in hearing was obtained

At a subsequent stage a right exploratory tympanotomy was performed An enormous anterior otosclerotic tumor was visualized It almost completely filled the fossa ovalis leaving only a small posterior remnant of the footplate which could be easily mobilized by chisel fracture A polyethylene tube was placed in contact with this mobilized area and articulated with the lenticular process of the incus following a preliminary stapedectomy (see Fig 8)

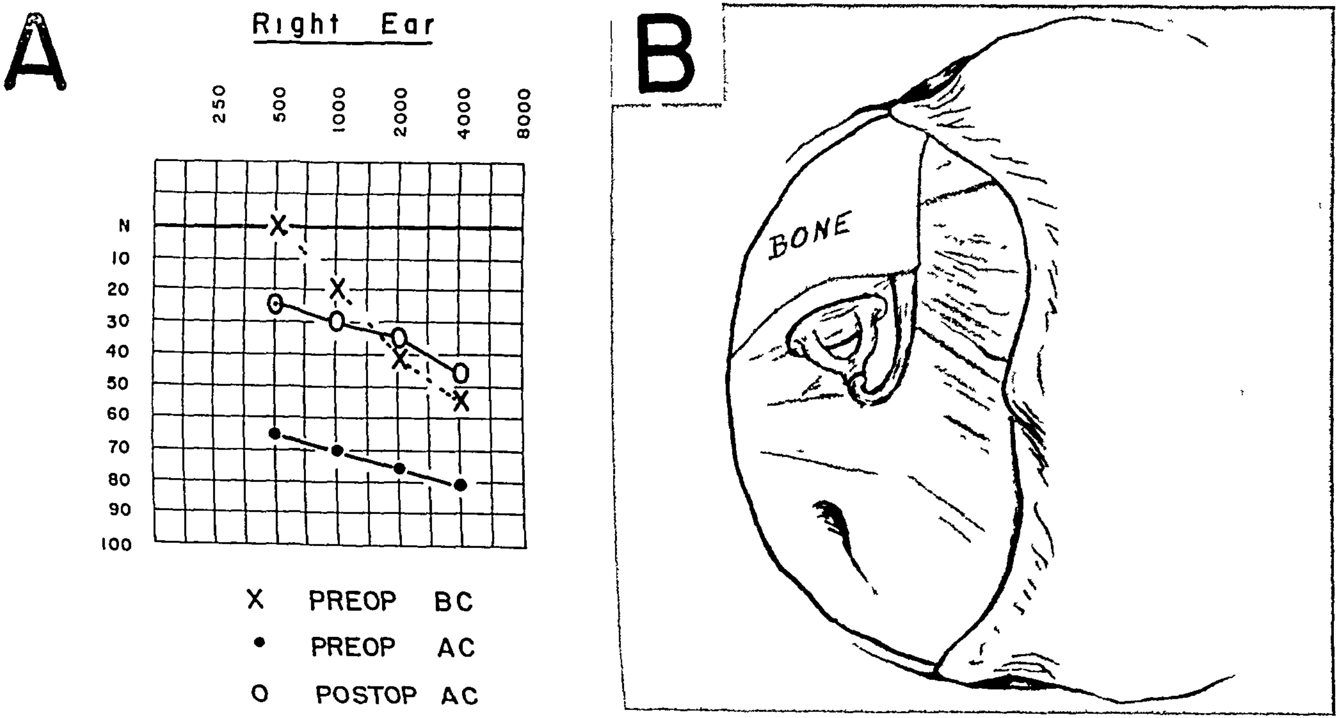

Fig 7. Incus annulus fusion A—Audiogram B—Tympanotomy findings Note bony union between long process of incus and annulus

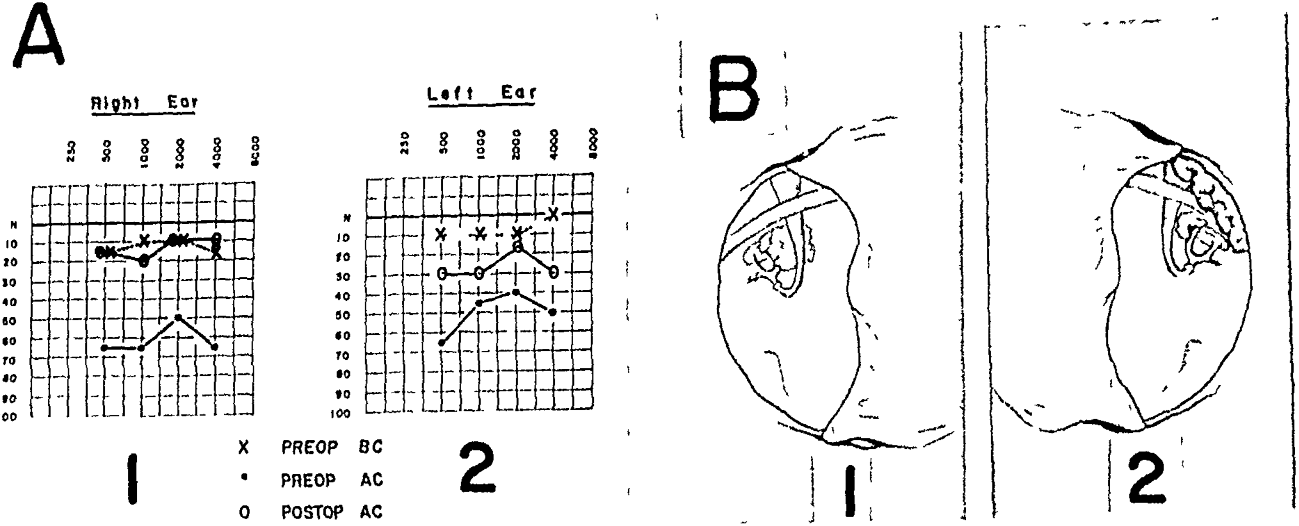

Fig 8 Co-existent otosclerosis and incudo-malleolar fusion by cholesteatoma A—Audiograms 1 Audiometric findings on otosclerotic right ear 2 Audiometric findings in ear with combined otosclerosis and incudo-malleolar fusion due to cholesteatoma B—Tympanotomy findings 1 Right tympanotomy findings showing large otosclerotic anterior tumor, 2 Left tympanotomy findings showing anterior otosclerotic tumor and epitympanic cholesteatoma with incudo-malleolar fusion

D *Incus Atrophy*

In addition to the problem of dislocated incudes and fixed incudes it is possible for a major conduction hypacusis to result from a fibrous atrophic incus which no longer is capable of transmitting acoustic energy because of loss of stiffness The following is an example of this problem

Case 9 J B This white male, age 30, gave a history of hearing loss in the left ear of 20 years' duration which he dated to a series of mastoidectomies done when he was a child He stated that he had four mastoidectomies on each side during a period of several years He has had excellent hearing in the right ear but a very marked loss in the left since that time

Examination revealed a postauricular healed scar on the right with an intact but slightly scarred right tympanic membrane with a positive Rinné response On the left, however, in addition to a well healed post auricular scar there was also a perfectly intact and mobile tympanic membrane The Rinné response was definitely negative There was evidence of good tubal function bilaterally and there were no significant nasal or nasopharyngeal findings X-rays revealed operative mastoid defects bilaterally

The left conductive deficit was accompanied by a lateralization of the Weber to the left side and audiometric findings showed a very definite left conductive hypacusis

A left exploratory tympanotomy revealed a fibrous incus attached to the capitulum The stapes was perfectly mobile with evidence of an excellent round window reflex The fibrous incus was sectioned and the lower remnant removed from the capitulum region A polyethylene prosthesis was placed between the capitulum of the stapes and the posterior aspect of the tympanic membrane There was a marked improvement in hearing which has persisted (see Fig 9)

D *Lenticular Process Necrosis*

One of the most common causes of conductive hypacusis other than otosclerosis is necrosis of the lenticular process of the incus This is very commonly seen in the course of mastoidectomy and tympanoplasty surgery, but it may also occur behind an intact non-suppurating drum and in such cases may definitely mimic otosclerosis

Case 10 D B, a 56 year old white female, gave a history of recurrent right otorrhea since childhood, and she has been progressively hard of hearing for many years, having worn a hearing aid on the left ear for 15 years There is a history of deafness in a son, but in no other members of the family

Examination revealed a suppurating right ear with evidence of chronic right mastoiditis* The left tympanic membrane, however, was intact

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*Incidentally the right ear was operated on separately via a postauricular radical mastoidectomy and a Type III Tympanoplasty with marked improvement in hearing

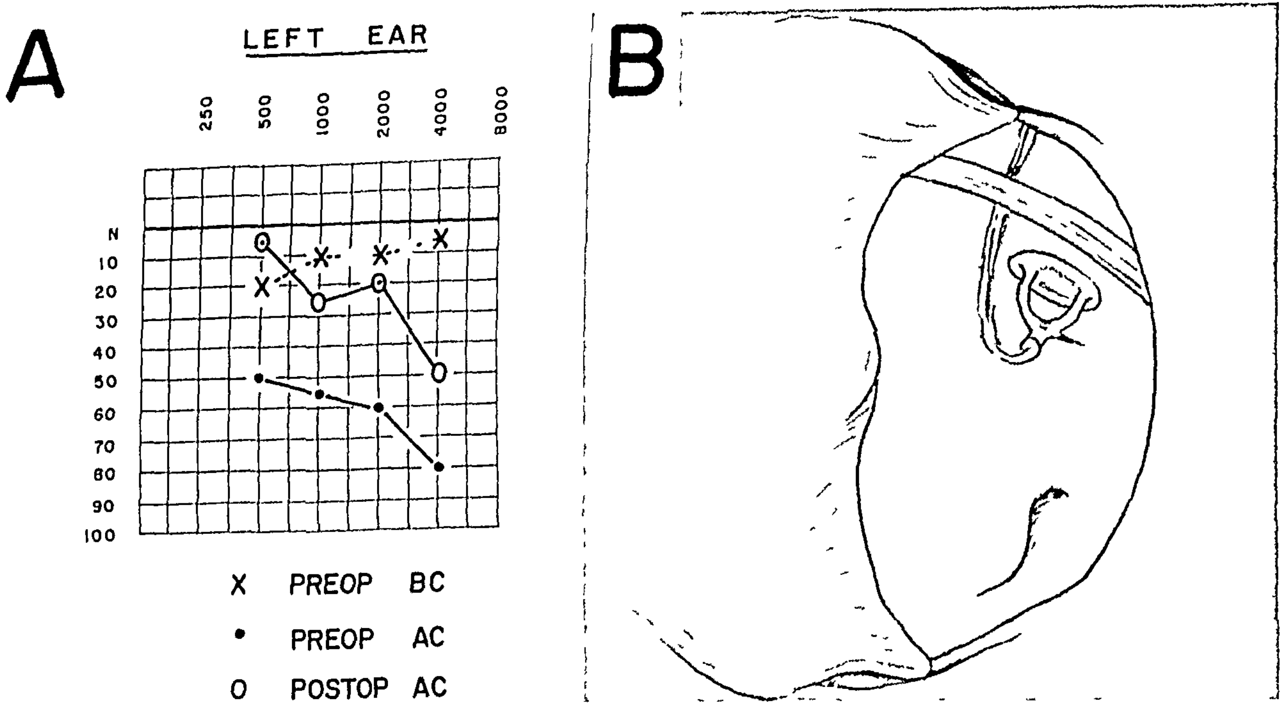


Fig 9 Incus atrophy A—Audiogram B—Tympanotomy findings Note the atrophic non-conductive incus

and only slightly scarred and mobile There was excellent left Eustachian tube function The left Rinne response was negative and there were no significant nasal or nasopharyngeal findings A bilateral conductive hypacusis was present on audiometry X rays revealed a pneumatic right mastoid with attic erosion, the left mastoid was diploic with periantral sclerosis

A left exploratory tympanotomy was done The stapes was perfectly mobile, the incus showed evidence of necrosis of the lenticular process Incudectomy was done by rotation and version A myringostapediopexy was performed which resulted in a fair improvement in hearing (see Fig 10)

III NON-OTOSCLEROTIC DISEASES OF THE STAPES

The histopathology of otosclerosis strangely received a great deal of attention during the period when the surgery of otosclerosis was at its lowest ebb The slight flurry of stapes mobilization surgery during the days of Miot,[1] Jack,[2], Blake[3] and their contemporaries died out completely by the second decade of this century It was during the second and third decades when there was virtually no surgery for otosclerosis (except for the pioneer fenestration research of Gunnar Holmgren[4] and Sourdille[5]), that a great deal was written on otosclerosis pathology Great controversies were waged in Europe by Otto Mayer,[6] Wittmaack,[7] and Nager,[8] but more heat was generated than light We still have no clear picture of the pathogenesis of otosclerosis

During the present renaissance of surgery for otosclerosis, relatively little research has added to the work of the twenties and thirties We enter 1960 with the comfortable term, "clinical otosclerosis" but with no real knowledge about this serious and disabling disease

There are, it must be admitted, certain gross pathologic observations which are accepted quite universally as the manifestations of otosclerosis, however, ankylosis of the stapes footplate can also be produced by certain well defined bone diseases which are not part of the concept of "clinical otosclerosis" These include "Paget's" disease, "brittle bones," tympano-sclerosis, osteo-arthritis, and other diseases A few examples will be presented

A *Paget's Disease (Osteitis Deformans)*

Paget's disease has been known for many years to be a

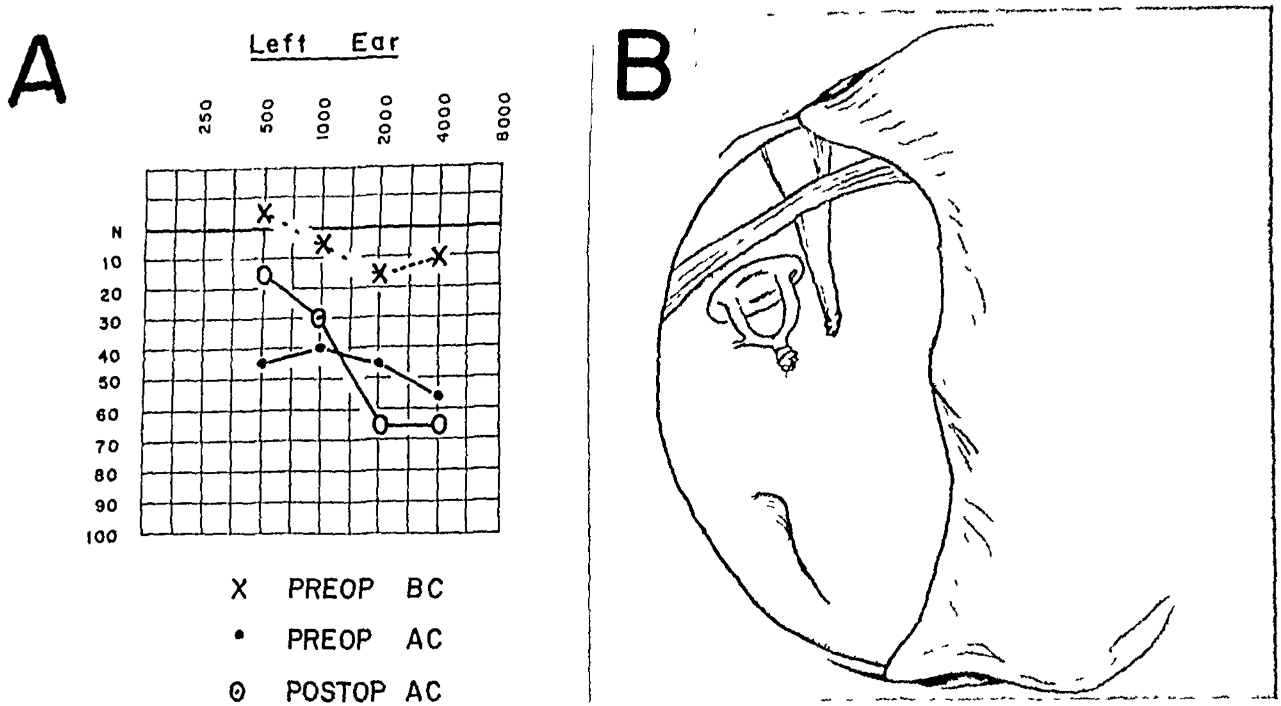


Fig. 10 Lenticular process necrosis A—Audiogram B—Tympanotomy findings Note discontinuity between incus and stapes due to lenticular process necrosis

progressive bone lesion with primary involvement of the skull Occasional reports have appeared in the literature describing not only ankylosis of the stapes but the occurrence of the large osteomata in ear canals This disease, also known as osteitis fibrosa, can mimic otosclerosis very closely, as illustrated in this case

Case 11 J G, a white male, aged 67, gave a history of a hearing loss in the right ear of only two years' duration He had no loss in the left ear There were no other otologic complaints or symptoms He told us that he had "marble bone disease" with a very strong family history since both his brother and sister have been diagnosed as definite cases of Paget's disease

Examination revealed non obstructive osteomata in both external auditory canals The right tympanic membrane was intact, translucent and mobile The right Rinné response was negative and the Weber was referred to the right ear There was good tubal function on politzerization The left tympanic membrane was intact, translucent and mobile with a positive Rinné response There were no significant nasal or nasopharyngeal findings Audiometric examination revealed evidence of a bilateral high frequency neural hypacusis with a superimposed right conductive hypacusis X-rays of the skull were reported as showing very definite evidence of Paget's disease of the skull

An exploratory tympanotomy was done with difficulty because of a very narrow and tortuous canal and the presence of osteomata A good deal of annulus was removed and several bone chips were sent to the laboratory for histologic examination The footplate was found to be elevated with a very large chalky lesion in the anterior peribasal region This lesion did not resemble grossly the usual otosclerotic process but was somewhat more brittle and much whiter in color A peribasal chisel procedure resulted in complete mobilization with a moderate improvement in hearing which has persisted

Histologic examination of the bone chips from the annulus region showed irregular marble like mosaic formations characteristic of Paget's disease (see Fig 11)

B Osteogenesis Imperfecta (Fragilitas Ossium, Osteopathyrosis, Periosteal Dysplasia, Lobstein's Disease, Eddowes Disease)

Brittle bone disease, known by many synonyms as shown above, is characterized by the occurrence of multiple fractures with poor healing not only in the long bones but in the calvarium There is a rather high incidence of so-called otosclerosis with this disease In most instances, the so-called otosclerosis is a pseudo-otosclerosis, namely a conductive hypacusis caused by some other middle ear lesion Rarely does one see true otosclerosis in conjunction with brittle bone disease The following case is an example of this type of problem

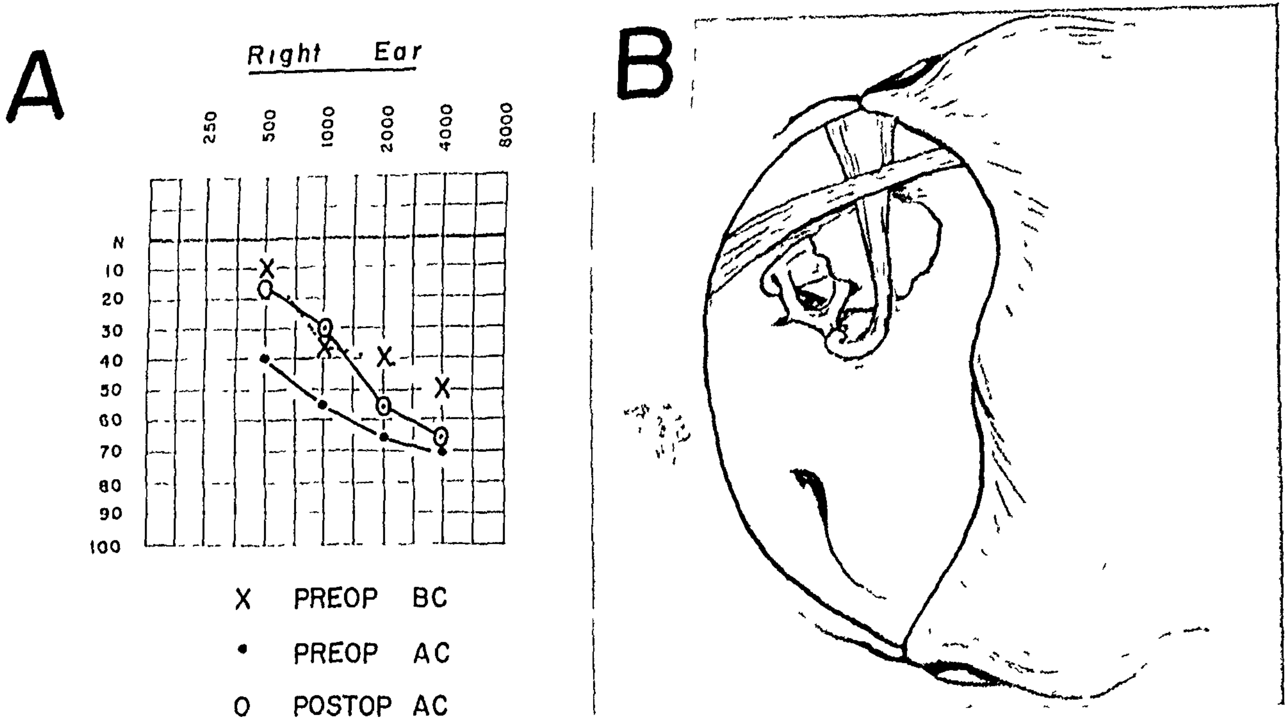


Fig. 11 Paget's disease (osteitis deformans) A—Audiogram B—Tympanotomy findings, showing large flat anterior calcified lesion due to Paget's disease

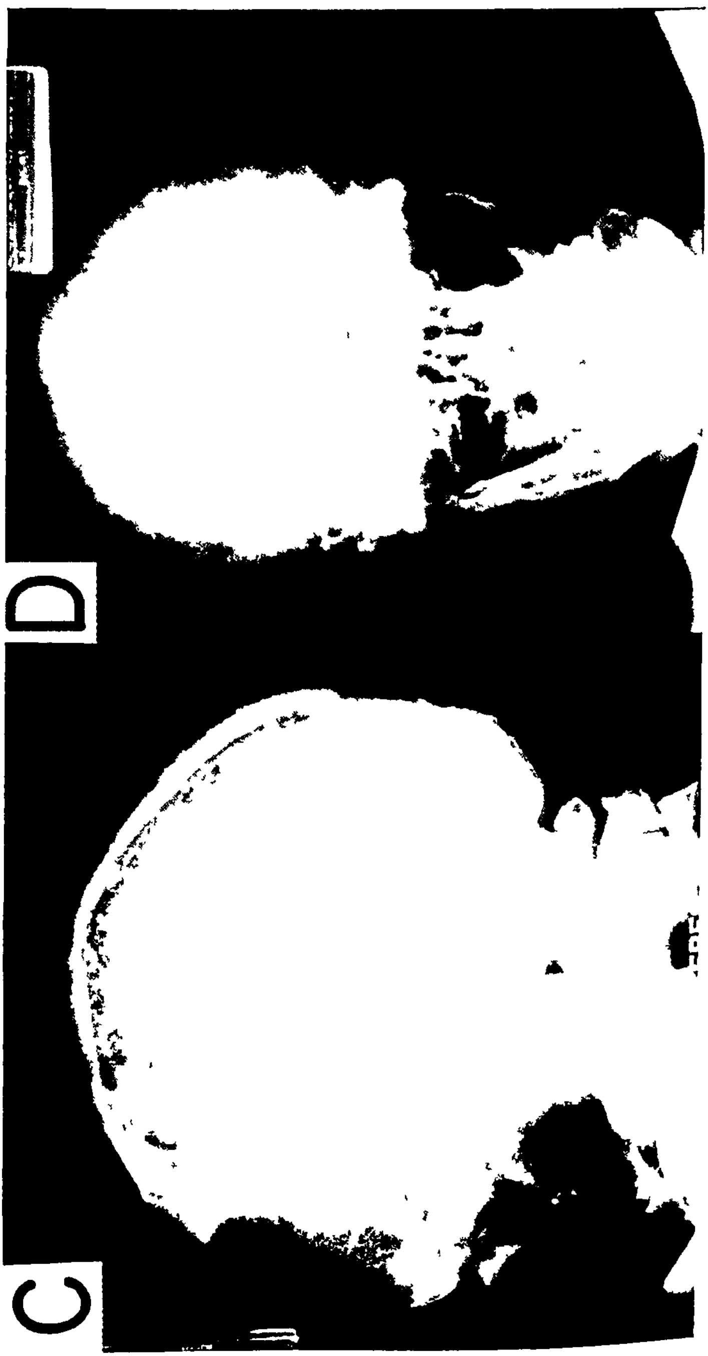


Fig. 11. C.—Lateral view of skull showing typical marble bone deformation. D.—Antero-posterior view of skull showing typical findings of Paget's disease.

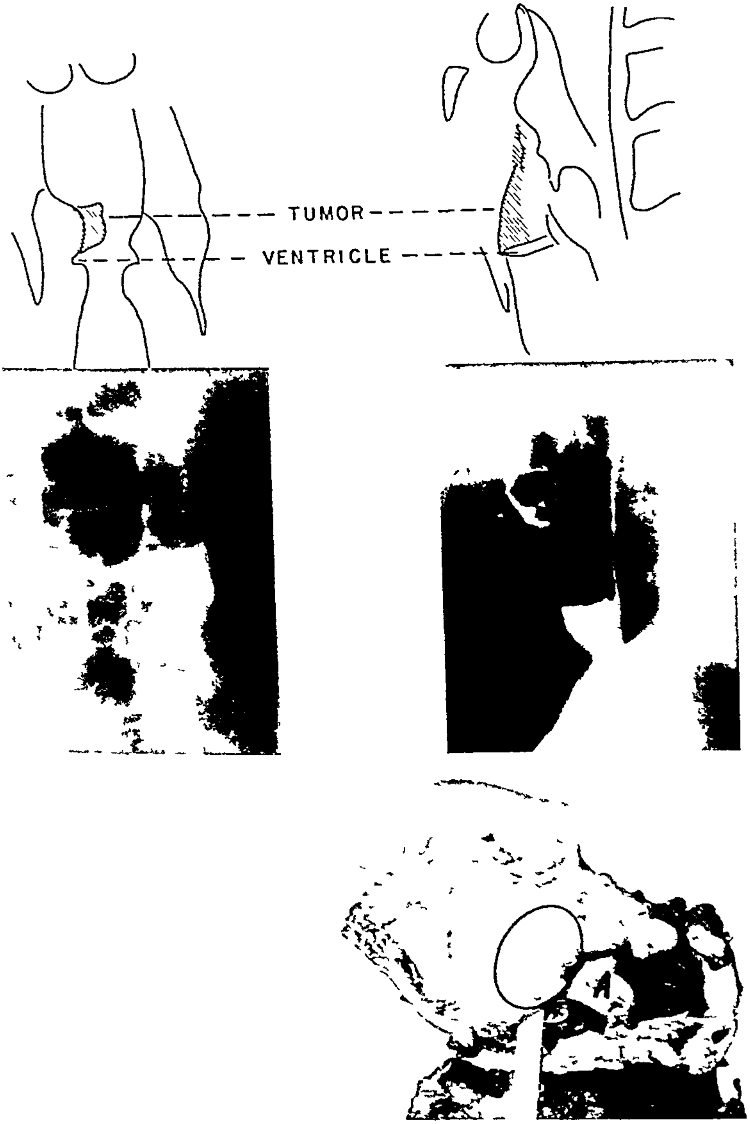


Fig 6 Supraglottic Frontal view—A tumor mass is present on the right false cord The ventricle and true cord are free of tumor There is an adequate margin of safety for a conservation operation

Lateral view—Note the tumor extension on the false cord

Specimen—The wooden applicator is inserted into the ventricle A—arytenoid errors) An adequate margin of safety to the true vocal cords was present in those subjected to total laryngectomy (see Fig 7) , however, these operations were performed before the significance of laryngogram findings was apparent In the remaining six instances of total laryngectomy, the surgical margin to the true cord area was too narrow to justify this conservation operation (see Fig 8)

In 23 instances the tumor was in the midline In six instances, the unilateral position of the tumor was demonstrated both by laryngograms and in the specimen

Transglottic (See Table I)

In the group of 16 patients, clinical accuracy was assessed correctly in ten, but it was missed in six instances Subglottic involvement was not suspected when the true cord was fixed in three cases False cord and ventricle involvement was missed in two instances, and base of epiglottis invasion was missed three times In two cases, two adjacent area involvements were simultaneously present and not suspected In two additional instances only "slight subglottic extension" was suspected when actually extensive infraglottic involvement was demonstrated by laryngograms These two were not listed as additional errors to the six listed

Laryngogram accuracy was highest in this group There were no errors in classification (see Fig 9) The four errors listed under the infraglottic group were actually called transglottic because of suspected false cord invasion

The majority of these tumors were unilateral In four instances, the tumor crossed the midline on laryngograms and this was confirmed on the specimen

Pyriform Sinus (See Table I)

There were 27 pyriform sinus cancers in this group While these tumors are hypopharyngeal and are not strictly laryngeal in origin, these have been included in this study since surgical treatment employed is most frequently laryngectomy and neck dissection (see Fig 10), use of partial or complete skin grafts for complete reconstruction of the pharynx, or

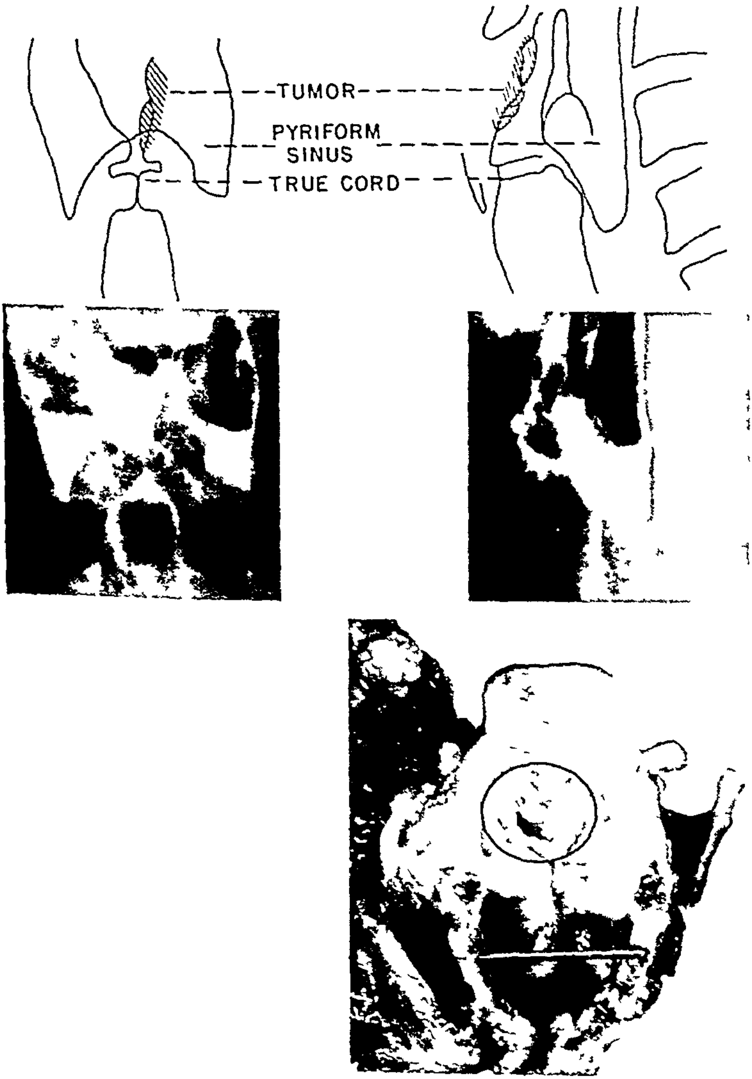


Fig 7 Supraglottic Frontal view—On the superior portion of the false cords there is narrowing of the vestibule and irregularity of the mucosal surfaces indicating presence of tumor The true cords subglottic area and ventricles are free of tumor There is a good margin of safety for a conservation operation

Lateral view—The laryngeal surface of the epiglottis is lobulated and irregular The tumor extends below the petiole but well above the ventricles

Specimen—Note that it was possible to perform a supraglottic operation

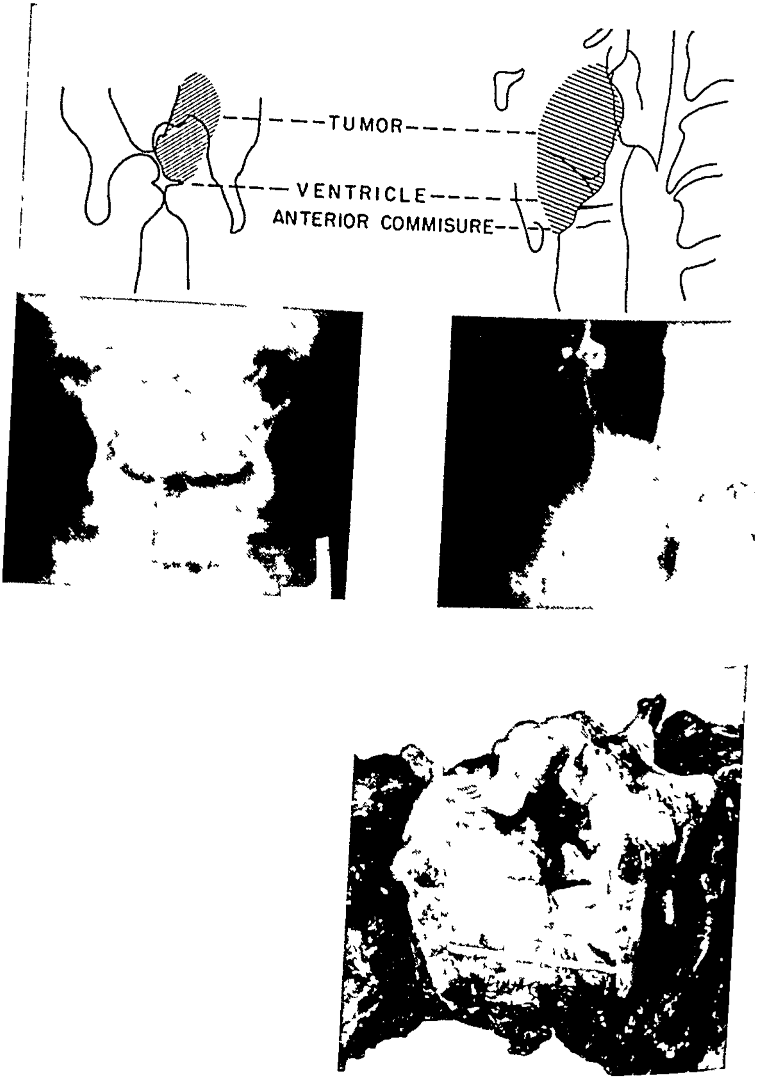


Fig 8 Supraglottic Frontal view—A tumor mass distorts the laryngeal vestibule bilaterally but mainly on the right The true cords are free of tumor

Lateral view—A bulky ulcerated tumor involves nearly the entire laryngeal surface of the epiglottis with invasion of the pre-epiglottic space The inferior margin of the tumor extends into the anterior commissure There is no margin of safety for a conservation operation

Specimen—Note extensiveness of the tumor with marginal invasion of the anterior commissure

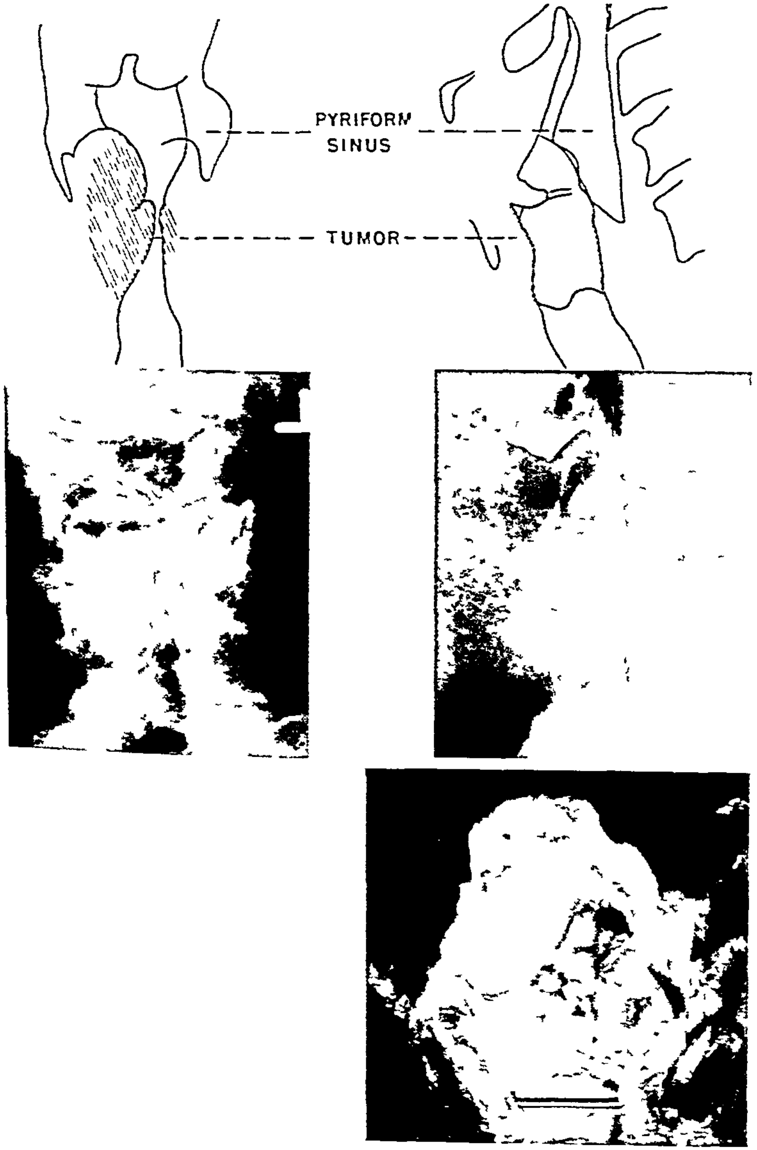


Fig 9 Transglottic Frontal view—The right laryngeal ventricle is obliterated by tumor which extends from above the false cord to a point two centimeters into the subglottis The cord is fixed

Lateral view—The tumor mass begins above the false cord The inferior margin is near the lower border of the cricoid cartilage Note that the anterior subglottic area is irregular The thyroid cartilage is destroyed

Specimen—Note the extensive tumor crossing the ventricle and cords

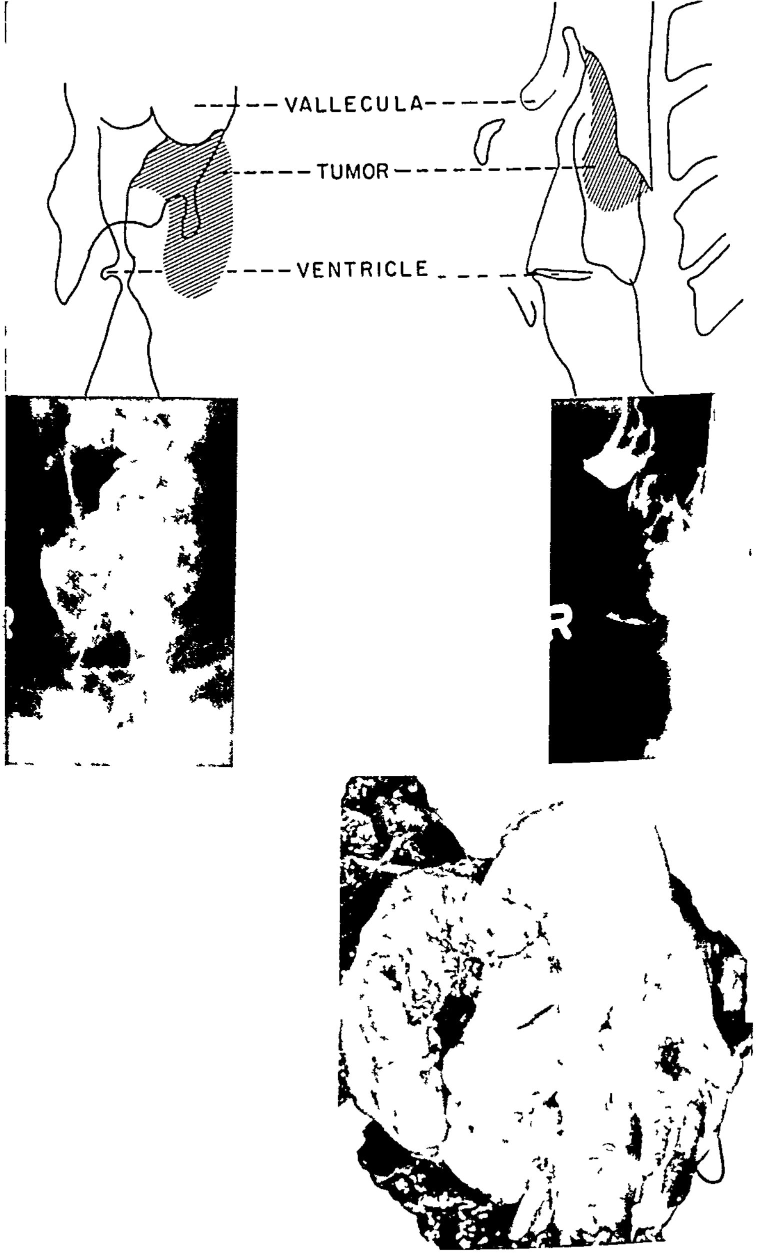


Fig 10 Pyriform Sinus Frontal view—The left pyriform sinus is almost completely replaced by a circumferential tumor that extends from the level of the vallecula to the apex of the pyriform sinus There is tumor involvement of the ary-epiglottic fold The vestibule false cords and true cords are displaced and edematous

Lateral view—The free edge of the epiglottis and the ary-epiglottic fold are irregular due to tumor The anterior wall of the pyriform sinus is involved

Specimen—Note the large size of the pyriform sinus carcinoma conservation of function, *i e*, transhyoid partial laryngopharyngectomy

Accuracy of diagnosis in this group has been high Clinically and radiologically in 26 of 27 cases the lesions were accurately placed In six of the 27, conservation of function operation was performed These were correctly described in five of the six cases by the radiologist

The following criteria were used for conservation of function operation These tumors should be small (under 2 1/2 cms), be free of thyroid cartilage invasion, demonstrate mobility of the arytenoid, and have a tumor-free apex in the pyriform sinus (see Fig 11) Preferably they should be placed on the lateral hypopharyngeal wall Direct examination of the posterior cricoid area should show no evidence of tumor

Clinically one error was found A small 1 cm lateral wall tumor was missed completely and was picked up by the radiologist (see Fig 12) The lone laryngogram error was an edematous ary-epiglottic fold together with non-filling of the pyriform sinus The tumor was interpreted primarily on the ary-epiglottic fold, actually it was on the medial wall of the pyriform sinus

SIZE OF PRIMARY LESION FROM LARYNGOGRAMS

The correction factor for magnification of the laryngeal structures on roentgenograms was determined by using a radio-opaque measuring ruler or a metal object of known size This factor for distortion was 20 per cent for the frontal views and 33 1/3 per cent for the lateral views All laryngogram measurements of the tumor were made of the superior-inferior extent, and no attempt was made to measure the transverse or anterior-posterior dimension

All laryngographic measurements were recorded first and final accuracy compared with the pathology records and gross photographs for orientation

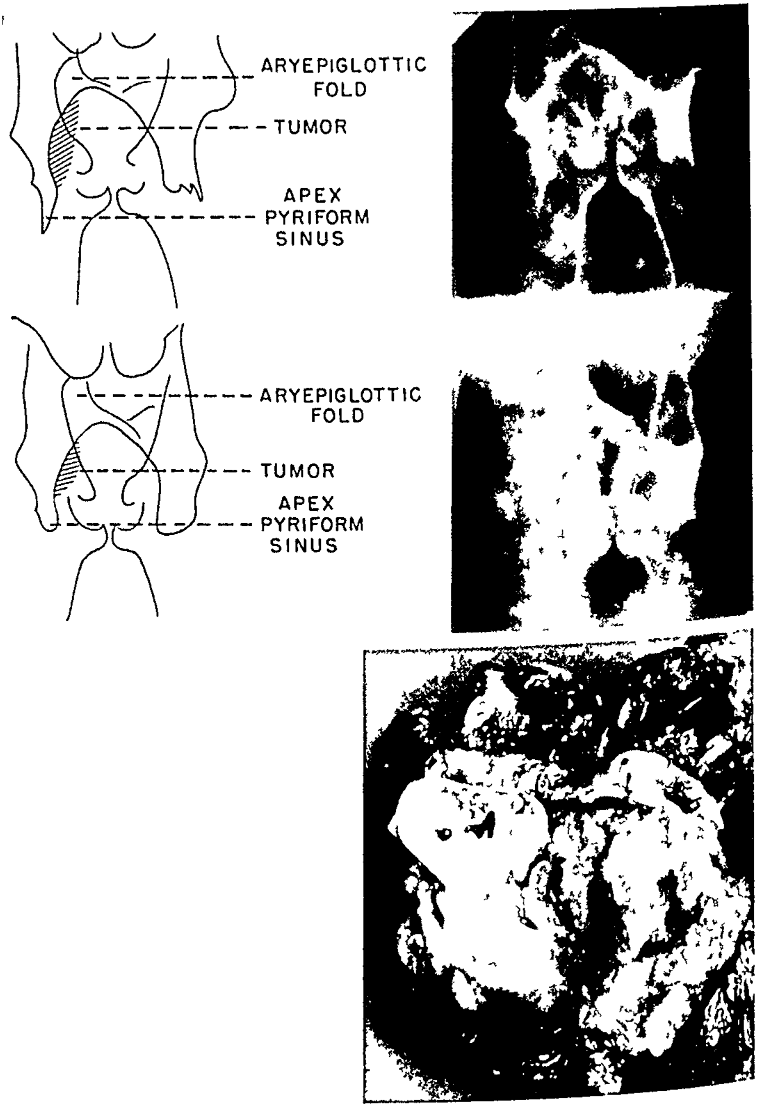


Fig 11 Pyriform Sinus Frontal view—There is tumor distortion of the inferior medial wall of the right pyriform sinus The apex of the pyriform sinus is free of tumor The laryngeal structures are normal except for marked edema of the right ary-epiglottic fold and arytenoid eminence

Specimen—A —arytenoid There is an adequate margin of excision on the specimen

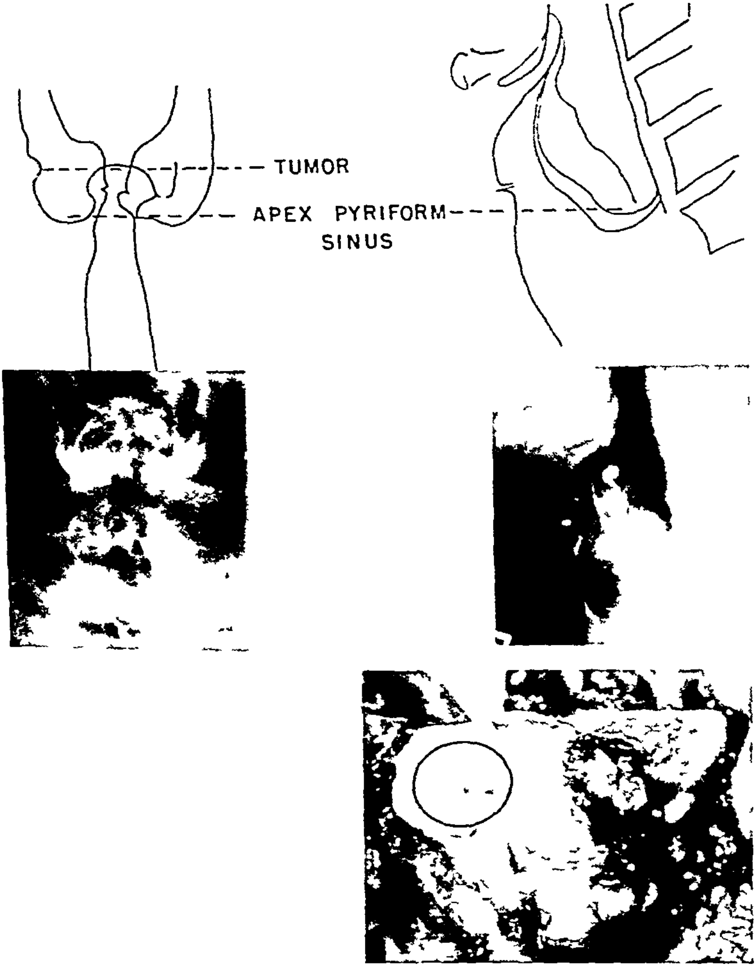
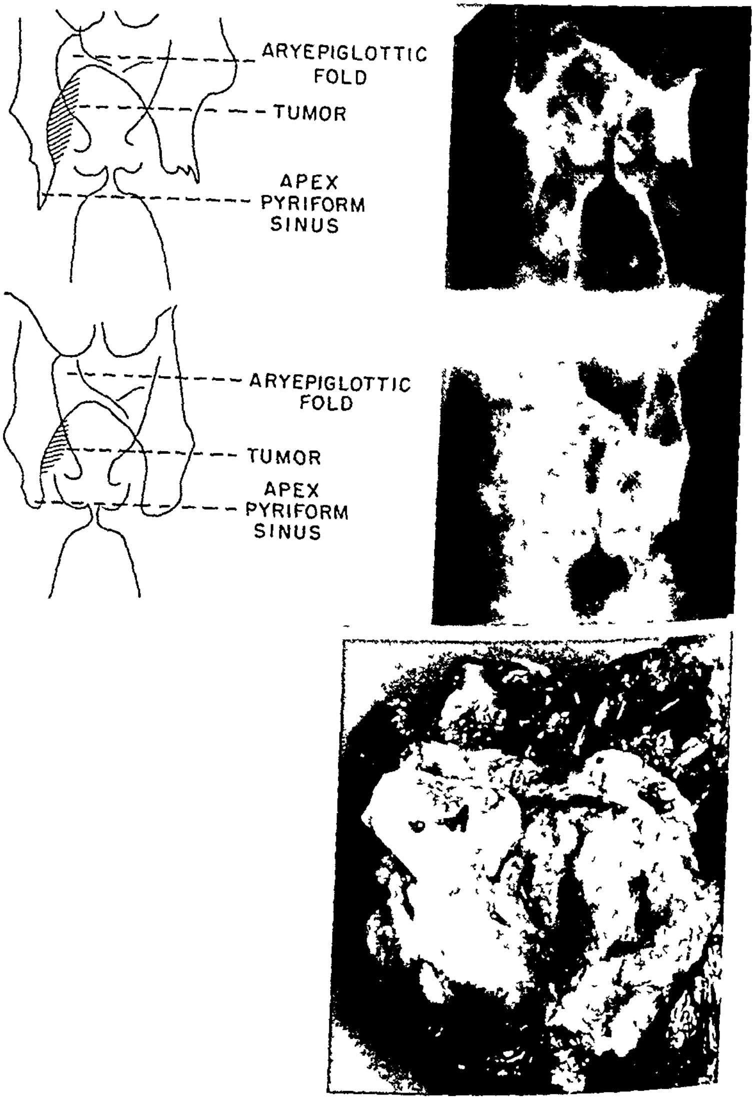


Fig 12 Pyriform Sinus Frontal view—A small (1 cm) irregularity is present in the lateral wall of the hypopharynx (modified Valsalva) The apex of the pyriform sinus is free of tumor

Specimen—Note the sharp elevated tumor margins

Glottic

From Table III, one can see that the size of the tumor seen on laryngogram varies as to location Since glottic lesions are confined to an area between the floor of ventricle to a point where the lower border of the intrinsic muscles end



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Specimen—A—arytenoid There is an adequate margin of excision on the specimen

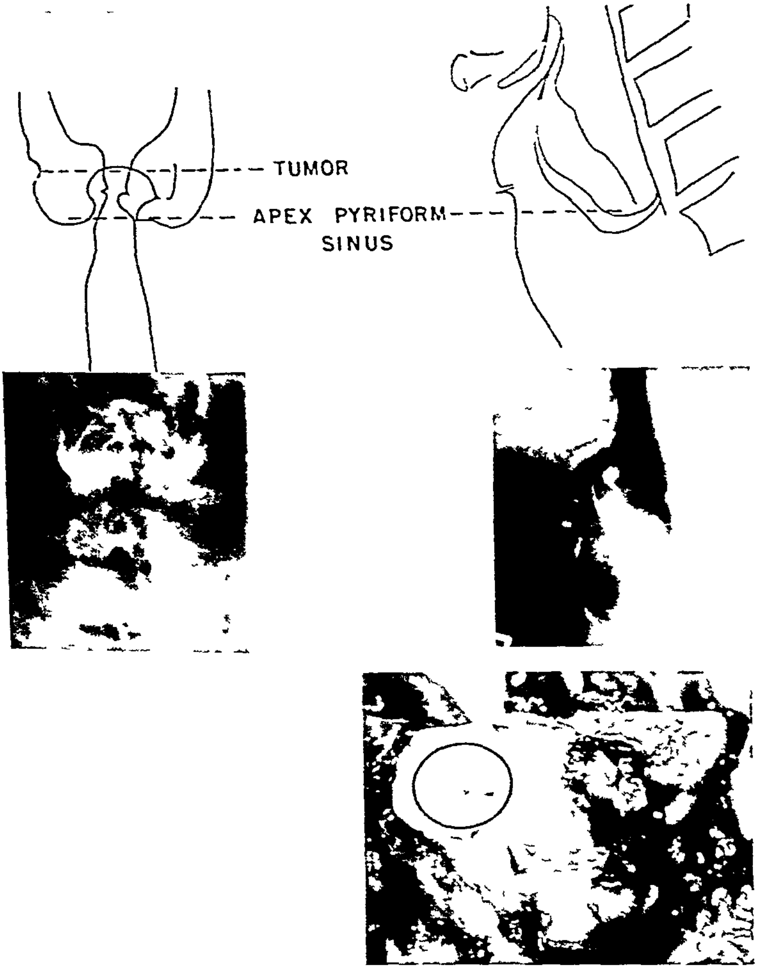

Fig 12 Pyriform Sinus Frontal view—A small (1 cm) irregularity is present in the lateral wall of the hypopharynx (modified Valsalva) The apex of the pyriform sinus is free of tumor

Specimen—Note the sharp elevated tumor margins

Glottic

From Table III, one can see that the size of the tumor seen on laryngogram varies as to location Since glottic lesions are confined to an area between the floor of ventricle to a point where the lower border of the intrinsic muscles end

(1 cm), no measurements were made Anterior-posterior extent of the tumor of the cord could not be accurately determined from lateral views

Infraglottic

There was an actual variation in size of tumors from 1 3-2 6 cms The margin of error is least in this group, with a ±0 3 cm on variation from the true size There was little difference in error whether the tumor was greater or less than 2 cms in the superior-inferior dimension

TABLE II

Classification Errors

	Clinical and Direct Laryngoscopy	Laryngograms	Total Pathology
Glottic	1 subglottic	1 false cord	13
Infraglottic	5 false cords 2 subglottic	4 { ventricle false cord 1 subglottic	14
Supraglottic	7 true cord	1 true cord	29
Transglottic	6 { 3 base epiglottis 2 false cord—ventricle 3 subglottic	0	16
Pyriform Sinus	1 missed tumor	1 A E fold	27
Total	22	8	99

TABLE III

Size of Primary Lesion

	Actual Size	Average Laryngogram Variation
Glottic	—	—
Infraglottic	1 3 2 6 cms	±0 3 cm
Supraglottic	1 0 5 0 cms	±0 6 cm
Transglottic	1 8 5 0 cms	±0 4 cm
Pyriform Sinus	1 - 6 cms	±0 6 cm

Supraglottic

The size of the tumor varied from 1-5 cms in greatest length in the superior-inferior extent, and the margin of variation was ± 0 6 cm

Transglottic

Tumors in this group vary from 1 6-5 0 cms on the specimen with a mean error variation of ±0 4 cm by laryngograms

Pyriform Sinus

These tumors varied in actual size from 1-6 cms with an error of ±0 6 cm by laryngograms

DISCUSSION

Limitations of Laryngogram Interpretation (See Table II)

The successful interpretation of laryngograms depends on the skill and speed of performance of the procedure and the experience and interest exhibited by the radiologist At the present time there are four areas where some difficulty has been experienced in the accurate interpretation

Anterior Commissure of True Cord When the anterior commissure area was deformed on laryngogram in glottic, infraglottic and transglottic groups, tumor was demonstrated pathologically in seven of eight instances On the other hand, when the laryngograms were negative, there was invasion microscopically in five of 13 instances This degree of error can only be explained by submucosal or relatively superficial invasion, insufficient to give gross distortion visible by laryngograms One can conclude that this area is not correctly assessed by laryngograms

True Cord Mobility There was a high correlation between clinical and radiologic findings in every instance when the cord was mobile or completely fixed When lag of the cord was evident by neuromuscular involvement or mass tumor weight, such a cord appeared mobile to the radiologist

False Cords The most frequent error was in interpretation of the false cords in the infraglottic group It is important to stress that more errors were made clinically as well on this particular structure Since biopsies were taken from these areas, postoperative edema will result The majority of these laryngograms were made after biopsy examination, and it is logical to assume that some of these errors in interpretation could have been avoided if laryngograms were done prior to biopsy.

There are additional anatomical factors to explain some of these errors. Examination of these specimens demonstrated that the false cord displacement superiorly by the cordal subglottic lesion obliterated the ventricle. Review of these errors on laryngograms showed that actually this was the case, and it is likely that these errors of interpretation will be less in the future.

Aryepiglottic Fold. When edema or distortion of this fold was present it was difficult to rule out tumor invasion. Tumor can be suspected only when definite scalloping and irregularity of radiographic outline is present.

Postcricoid. Laryngograms were never designed for the diagnosis of lesions in this area, and no interpretation can be given by the method of examination being discussed.

Significance of Laryngogram Findings

Laryngograms have demonstrated their great value as an adjunct diagnostic procedure for laryngeal cancer. When these findings are correlated with indirect and direct laryngoscopy and biopsy report, together with cervical node findings, the otolaryngologist can separate his cases into groups with high and low metastatic rate and arrive at a definite scheme for treatment.

Accuracy of laryngogram classification was high, 92 per cent as contrasted to 78 per cent by conventional clinical appraisal. There was an 8 per cent error in laryngogram classification, with the majority of the errors in the infraglottic group. There was a much greater error rate by clinical means (22 per cent). With continued refinement in laryngogram technique and greater experience in interpretation, the false cord error rate should be less in the future. Other errors of interpretation are extremely low and are to be expected.

Certain clinical situations have always made accurate clinical classification difficult. Bulky supraglottic tumors are an-way problems and the precise lower margin is always difficult to determine. While lesions above the petiole of the epiglottis are easily determined and one can perform the supraglottic subtotal laryngectomy operation with confidence, the greatest problem confronting the surgeon who wishes to conserve function is with lesions of the false cord and base of the epiglottis. In the latter group, laryngograms can very accurately discern freedom of involvement of the true cord. Bulky tumors of the false cord that overhang the true cord can be precisely visualized by laryngograms. It is in this situation that clinically one has most frequently mistaken tumor to involve the true cord.

The accuracy of the radiologist in classifying a lesion as supraglottic was high. In 17 instances where the supraglottic operation was performed, 12 were listed as wide surgical margins of safety and proven by the examined specimens. In five cases where tumor was in the false cord area and petiole, these were accurately visualized and again correlated well with the safety margin of the surgical specimen. Of the 12 patients with laryngectomy and neck dissection performed, six cases could have had a supraglottic subtotal operation. The latter operation was not performed because in the clinical preoperative appraisal, it was felt there was "insufficient margin of safety."

The purpose of bringing this point up is to emphasize that accuracy of diagnosis is necessary for the purpose of evaluating whether conservation of function operation can be performed rather than write the case off to the more easily executed total laryngectomy. While this paper does not deal with end results of the supraglottic subtotal operation, these highly selected cases are quite successful for a one to six-year follow-up.[14,1] Perhaps the high frequency of the "pushing" type of lesion in the supraglottic group accounts in part for success with this relatively narrow surgical margin of safety.

Clinical estimation of extent of cancer involving the infraglottic and transglottic groups has been frequently in error. The error in judgment in both groups is explained by fixation of the cord which masks the size of the tumor present

Size and extent of subglottic involvement have been difficult to appraise even with a mobile or somewhat paretic cord Tumors of the transglottic group that have involved the false and fixed true cord frequently obscured marked infraglottic extension In addition the base of the epiglottis has been sometimes overlooked In all of these situations, the laryngograms have proved their value by precise localization and sharp delineation of the lower extent of the tumor

Tumor crossing the midline was correctly determined by laryngogram with every lesion in the supraglottic, infraglottic and transglottic groups

There was a high accuracy index for pyriform sinus lesions from the clinical and radiological standpoint The majority of these cases required total laryngectomy and neck dissection, with modification such as skin grafting or other reconstructive procedures with larger tumors, however, there is a small number of selected cases where conservation of function procedures can be performed after a detailed clinical and radiological evaluation This should help the surgeon decide what surgical procedures can be instituted

No one can appreciate more than the surgeon the great importance of knowing where the lesion exactly is He can plan his surgical procedure rather than complicate this with an ill-planned exploratory approach

Size of tumor can be accurately determined for all classification sites with a mean variation from ±0 3-0 6 cm depending upon tumor site It is impossible to estimate size by laryngoscopy, but it should not be surprising how frequently exact actual tumor size correlates with laryngograms Size of tumor, degree of differentiation and location of the lesion have a direct bearing on the metastatic rate

Since the transglottic group has the highest metastatic rate for apparent and inapparent metastases when the tumor is over 2 cms in diameter, it follows that neck dissection with total laryngectomy is mandatory in this situation If a palpable node is present on one side with a 2 cm or larger transglottic or supraglottic lesion, consideration of bilateral neck dissection is warranted

It should be apparent that there are other therapeutic implications Patients with small glottic cancer and possibly minimal lesions of the epiglottis where the metastatic rate is low might better be handled by irradiation With improved methods of treatment by radiotherapists, conservation of function will apply to radiotherapy and the parameter of surgical treatment will be better defined Until the time when this study is again reviewed and specific conclusions drawn, the surgical management will continue to be selected on the basis of accurate clinical and radiologic diagnosis

SUMMARY AND CONCLUSIONS

1 Accurate clinical classification of cancer of the larynx is necessary since the choice of therapy is governed by the probability and location of metastases

2 Analysis of data obtained from a study on routine *en bloc* neck dissection with total or subtotal laryngectomy has given us very valuable information as to certain characteristics of the primary lesion

3 A pathologic classification based on location of the primary lesion has been used for laryngographic and clinical use The value of the proposed classification indicates that there is a progressive increase in metastatic rate from glottic, infraglottic and transglottic cancers

4 Size of tumor, degree of differentiation, and location is related to apparent and inapparent cervical metastases

5 Laryngograms can more accurately localize and objectively document the size of the cancer than clinical evaluation

6 Size of the primary tumor can be estimated on laryngograms to within ±0 3-0 6 cm of actual tumor size

7 The laryngogram is necessary for accurate diagnosis

8 Logical treatment will necessarily follow *a* Conservation of function by radiotherapy for early lesions of the cord, *b* larynx-sparing operations for selected cases of supraglottic or pyriform sinus lesions, *c* more radical operations (total laryngectomy to include neck dissection when the inapparent metastatic rate is high

9 The therapeutic conclusions drawn from this study are necessarily tentative The decision test by which all therapy is judged is a careful follow-up of patient survival

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ent species was found, giving an average of 1 45 species per culture

An interesting observation is that the number of gram negative organisms found in cultures was relatively high Escherichia coli, Klebsiella aerogenes, Neisseria catarrhalis, Pseudomonas aeruginosa and Proteus vulgaris accounted for 26 82 per cent of the organisms found The combined staphylococcus and streptococcus forms had the highest relative abundance of 67 33 per cent but the gram negative group was surprisingly numerous

TABLE I

Number of Species in 1 000 Serial Cultures

ecies	1	2	3	4	Number of Strains	Relative Abundance Per Cent
	Per Cent of Species in Given Mixture					
l Fecalis	50	50	0	0	6	0 41
ubtilis	23	62	15	0	13	90
dida Albicans, etc	14	57	29	0	7	48
lococcus						
neumoniae	29	71	0	0	7	48
htheroids	20	60	20	0	5	34
Coli	77	18	5	0	104	7 19
bsiella	69	26	5	0	140	9 68
sseria	0	43	46	11	37	2 56
udomonas	72	23	5	0	75	5 18
teus Vulgaris	63	31	6	0	32	2 21
monella Paratyphii	100	0	0	0	5	34
gella Dysent		0	0	0	1	07
Albus	39	41	18	2	304	21 01
Aureus	62	28	9	1	278	19 21
ep Viridans	25	46	26	3	205	14 18
ep B Hemo	16	56	26	2	114	7 88
ep Anhemo	16	57	26	1	70	4 84
ep Anaerobic	100	0	0	0	3	21
moph Influenzae	0	68	26	5	19	1 31
tero Cocci	0	79	21	0	19	1 31
scellaneous	33	0	67	0	3	21
mber	641	552	234	20	1,447	100 00
lative Abundance Per Cent	44 3	38 1	16 2	1 4		100 00

An inspection of the percentage of strains of a given species found in pure cultures and in mixed cultures shows extreme variation from species to species For example, over 60 per cent of the strains of Escherichia coli, Klebsiella aerogenes, Pseudomonas aeruginosa, Proteus vulgaris and Staphylococcus aureus were found in pure cultures On the other hand no strains of Neisseria catarrhalis, Hemophilus influenza or enterococci were found in pure culture Particularly noteworthy is the fact that Staphylcoccus albus was found in pure cultures only 39 per cent of the time as contrasted with Staphylococcus aureus which was found 62 per cent of the time Pure cultures of streptococci were relatively rare

NUMBER OF POSSIBLE MIXTURES

These interspecies differences in the relative numbers found in pure and mixed cultures suggested an investigation of the species comprising the mixtures The 12 most common clearly defined species were selected for the investigation The maximum number of species recorded in a given culture was four With 12 species and a random distribution the maximum possible numbers of mixtures and the numbers observed were

Number of Species in Culture	Number of Possible Mixtures	Number of Mixtures Cultured
1	12	10
2	66	45
3	220	32
4	495	5
Total	793	92

A much smaller number of mixtures of the different species is observed than would be expected on the basis of a random distribution By a random distribution we mean that only factors of a chance nature cause the mixtures, factors of antagonism, commensalism, symbiosis and different environmental requirements are not considered The relatively small number of observed mixtures as compared with the possible mixtures would suggest that some or all of the above and possibly other factors are at work

To check this possibility a coefficient of association (Ken-

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*Species with less than ten strains recorded are not considered in this discussion dall,[5] 1950, p 30) was computed for each possible pair of organisms, and the results are given in Table II.

The coefficient of associations, commonly designated by the symbol "Q" is a measure of the degree of association A value of a +1 0 would indicate that the two species were always found together, never apart, which might mean a symbiosis or commensalism A value of a –1 0 would mean that the two species were never found together which could indicate a profound antagonism A value of 0 0 would indicate that the species were found together about as often as would be expected on the basis of pure chance

It must be realized that these values were derived from cultures grown on agar plates and that one culture might overgrow another, or that antagonisms or symbioses not present in tissue might be present on the plates in the laboratory, nevertheless, the derived values suggest some interesting possibilities

An inspection of the Table shows that the majority of the values is negative, indicating that an effective antagonism exists between the various pairs of species This is not too surprising, however, the high negative values for some of the common mixtures such as Staphylococcus albus, Staphylococcus aureus (–0 62) and Staphylococcus aureus, Streptococcus viridans (–0 49) are interesting

The positive associations are of the most interest because they indicate that the two species are found together more often than would be expected by chance.

Neisseria catarrhalis, Streptococcus viridans and Hemophilus influenzae showed a tendency towards having the most positive associations Particularly noteworthy are the following

Neisseria catarrhalis - Streptococcus viridans	–0 82
Neisseria catarrhalis - Staphylococcus albus	–0 53
Staphylococcus aureus - Hemophilus influenzae	–0 57
Sterptococcus viridans Beta Hemolytic Streptococcus	–0 48

It is interesting to note that Hemophilus influenzae and

TABLE II

Association Between Different Species

	6 E Coli	7 Klebsiella	8 Neisseria	9 Pseudomon	10 Proteus	16 St Albus	18 St Aureus	20 Strep Virid	21 Strep B Hemo	22 Strep Anhemo	26 Hemoph Influenzae
B Subtilis	-1 00	-1 00	-1 00	-1 00	+0 44	+0 68	-0 36	1 00	-1 00	-0 05	-1 00
E Coli		-0 48	-1 00	-0 80	-0 57	-0 58	-0 92	0 76	-1 00	-0 46	-1 00
Klebsiella			-1 00	-0 51	-0 07	-0 72	-0 85	-0 51	-0 66	-0 85	-1 00
Neisseria				-1 00	-1 00	+0 53	-0 72	+0 82	0 10	-0 47	+0 52
Pseudomon					+0 29	0 60	-0 82	-0 74	-1 00	-0 71	-1 00
Proteus						0 74	-1 00	-1 00	-1 00	-0 41	-1 00
St Albus							-0 62	+0 20	-0 04	+0 26	0 03
St Aureus								-0 49	-0 29	0 02	+0 57
Strep Viridans									+0 48	-0 17	+0 29
Strep B Hemo										-0 17	-0 68
Strep Anhemo											-1 00
Hemoph Influenzae											

Neisseria catarrhalis were not observed in pure culture and that Streptococcus viridans was found in pure culture only 25 per cent of the time

In our clinical experience Proteus and Pseudomonas are frequently found together in chronic mastoid infections Staphylococcus aureus sinusitis often follows Hemophilus influenzae infection of the upper respiratory tract and Streptococcus viridans infections often are left as the residue of a Beta Hemolytic streptococcus infection These are especially interesting in view of the positive association between them *in vitro* as illustrated in Table II, as it may indicate a conversion from a relative saprophyte to a parasite when they occur together

RESULTS OF SENSITIVITY TESTS AGAINST PURE CULTURES

The results of the sensitivity tests of the antibiotics and chemotherapeutics are given for the various species of bacteria in Table III The results are expressed in percentage of strains inhibited or killed by each antibiotic

An inspection of the response of each species of bacteria to the various antibiotics shows similarity between certain groups of the antibiotics There is naturally some variation, but the over-all pattern is clear

By grouping together those antibiotics having similar responses, a profile of per cent response versus species of bacteria can be constructed The profiles so obtained are shown in Fig 1

A total of six qualitatively distinct groups was found as follows

Group I consists of Penicillin, Erythromycin, Bacitracin Novobiocin and tentatively Oleandomycin The last is only tentative because of insufficient data This group has little effect on most gram negative bacteria but acts rather successfully against the gram positive group

Group II consists of the Tetracyclines and Nitrofurantoins While drug house advertising leaves the impression of varying

TABLE III A

Summary of Results of Tests of Pure Cultures of Bacteria Against Various Antibiotics
Per Cent of Strains Sensitive to Gram Negative Organisms

Antibiotics and Bacteriostatics	Bacillus Subtilis	Escherichia Coli	Klebsiella Aerogenes	Pseudomonas Aeruginosa	Proteus Vulgaris
Penicillin	35	1	5	1	23
Erythromycin	65	2	6	1	3
Tetracycline	75	43 (383)	38	7	13
Oxytetracycline	82 (11)	43 (281)	34 (101)	7 (118)	12 (60)
Chlortetracycline	82 (11)	43 (281)	33 (101)	7 (118)	10 (60)
Chloramphenicol	85	91	69	42	82
Streptomycin	70	56	42	59	68
Bacitracin	25	1	8	4	4
Neomycin	55	56	72	32	56
Polymixin B		54 (61)	28 (97)	54 (46)	0 (15)
Novobiocin	85 (13)	4 (341)	5	3 (160)	8 (110)
Nitrofurantoin	40 (10)	65 (269)	34 (105)	3 (119)	18 (82)
Oleandomycin		1 (82)	6 (100)	2 (46)	0 (14)
Cyclamycin					
Gantrisin Sulfisoxazol	25	28	28	3	12
Elkosin Sulfaclimetine	18 (17)	22 (300)		0 (117)	8 (107)
Triple Sulfa	12 (17)	21 (300)		9 (117)	10 (107)
Sulfadiazine	6 (17)	22 (300)		18 (117)	6 (107)
Sulfamerazine	12 (17)	26 (300)		2 (117)	9 (107)
Thiosulfyl	12 (17)	15 (300)		0 (117)	5 (107)
Sulfathiazole		15 (300)			8 (107)
Number of Strains Tested	20	384	109	174	117

Figures in parentheses indicate the numbers of strains upon which the percentage was based when the number of strains differs from the total given at the bottom of page

TABLE III B

Summary of Results of Tests of Pure Cultures of Bacteria Against Various Antibiotics
Per Cent of Strains Sensitive to Gram Positive Organisms

Antibiotics and Bacteriostatics	Staphylococcus Albus	Staphylococcus Aureus	Streptococcus Viridans	Streptococcus Beta Hemolytic	Streptococcus Anhemolytic	Streptococcus Anaerobic
Penicillin	67	15	87	92	86	94
Erythromycin	81	75	83	85	81	90
Tetracycline	10	11	78	58	77	87
Oxytetracycline	38 (166)	39 (209)	76 (58)	65 (23)	76 (62)	83 (21)
Chlortetracycline	39 (166)	38 (209)	79 (58)	61 (23)	76 (62)	88 (21)
Chloramphenicol	86	86	96	95	93	100
Streptomycin	53	15	11	22	49	68
Bacitracin	85	82	78	85	88	87
Neomycin	61	66	9	20	23	16
Polymixin B	2 (92)	5 (171)	2 (16)	6 (17)	0 (7)	
Novobiocin	85 (221)	68 (213)	73 (71)	61 (38)	72 (82)	71 (28)
Nitrofurantoin	83 (76)	79 (17)	28 (74)	95 (18)	69 (18)	65 (17)
Oleandomycin	73 (92)	51 (170)	36 (72)	88 (17)	57 (7)	
Cyclamycin	83 (21)	92 (25)	86 (7)	70 (10)		
Gantrisin Sulfisoxazol	28	16	8	12	0	3
Elkosin Sulfadimetine	12 (125)	18 (82)	5 (19)	2	2 (80)	3
Triple Sulfa	12 (125)	13 (82)	5 (19)	2	4 (80)	3
Sulfadiazine	10 (125)	13 (82)	5 (19)	2	1 (80)	3
Sulfamerazine	12 (125)	15 (82)	5 (19)	2	2 (80)	3
Thiosulfyl	23 (125)	17 (82)	5 (19)	5	6 (80)	
Sulfathiazole	21 (125)	36 (17)	5 (19)	5	1 (25)	
Number of Strains Tested	236	256	77	10	88	31

Figures in parentheses indicate the numbers of strains upon which the percentage was based when the number of strains differs from the total given at the bottom of page susceptibility of organisms to the three Tetracyclines, the figures in Table III do not bear this out It would seem the Tetracycline, the oxy- or the chlor-forms can be used indiscriminately with little variance in sensitivity results Interestingly, the germicidal furan derivative Nitrofurantoin shows a profile so similar to the Tetracycline that it is best considered with this group in studying sensitivity results They are moderately effective against Escherichia coli, Klebsiella aerogenes and the gram positive organisms

Group III consisting of Chloramphenicol is the only good broad spectrum antibiotic as judged by this method

The various organisms tested show a uniformly high sensitivity to this antibiotic with the exception of Pseudomonas aeruginosa which is not highly susceptible to any of the antibiotics tested Of all of the antibiotics considered in this study Chloramphenicol is the most efficacious broad spectrum antibiotic as judged by the disc test results As will be shown later, it overlaps most of the other antibiotics and often includes strains sensitive to other types as well as many of the insensitive types In terms of its broad spectrum and the high percentage of strains sensitive to it, Chloramphenicol would be the antibiotic of choice

Certain serious side reactions have occasionally been reported for this drug,[6 7] and while these are disputed or minimized by some writers,[8,9] it would seem wise to reserve Chloramphenicol for use when the other antibiotics fail

Group IV consists of Streptomycin, Dihydrostreptomycin and Neomycin Although the latter was found by Waxman[10] during his search for an antibiotic to handle the Streptomycin resistant bacteria, its total profile closely matches that of the actinomycete derivatives It is interesting that there was no difference observed between Streptomycin and Dihydrostreptomycin The Group IV profile is a comparatively flat curve moderately successful against all the bacteria tested except streptococci There were insufficient cases of the anaerobic bacteria such as Clostridium and Bacteroides for evaluation in this study

Group V consists of Polymixin B only It is unique in that it has a high antibiotic value against Pseudomonas aeruginosa and Escherichia coli, though poor against the other organisms Although primarily a topical medication because of its toxicity when used parenterally it is of value because of the increasing difficulty with pseudomonas skin and mucous membrane infections

Group VI is made up of the various sulfa derivatives It has not been plotted because disc test does not seem as reliable for the sulfas as it does for the antibiotics An investigation of some of the sulfa results showed internal inconsistencies It may be that the reason lies in poor solubility or slow diffusion of the drug in agar or it may be due to a more complex reason In any event, we do not consider that the sulfa data are worth analyzing further

The placing of the various antibiotics into the groups discussed above does not necessarily have any biological or theoretical significance, though similar spectra might imply similar actions The main use of the profile curve is in selecting an antibiotic group for therapy with a given organism When there are several antibiotics in the group, then the final judgment must be made in terms of the sensitivities of the particular bacterial species and strain, and in terms of that unanalyzable faculty called "clinical judgment"

It seems to us that the data presented in this paper may give some aid in selecting the antibiotic for a given pure or mixed bacterial infection If the species of bacteria causing the infection are known and the sensitivities are known, a judgment of the most probably useful antibiotic can be obtained as follows For a pure infection with one species of pathogen *1* Consult Fig 1 and compare the sensitivity of the various antibiotic groups for the species in question and select the group or groups showing the best effect on the organism For example, consider a Proteus vulgaris infection of the ear canal Groups I, II and V show very little effect against this organism Group III, Chloramphenicol, shows that 82 per cent of the Proteus strains respond to this drug, and for Group IV about 62 per cent responded Selecting Group IV, a check is made in Table III where it is found that Streptomycin had a percentage value of 68 and Neomycin a value of 56 per cent *2* A check of the laboratory tests shows (let us say), that the particular strain involved in the infection has the following sensitivities

Antibiotic	Sensitive
Chloramphenicol	Yes
Streptomycin	No
Neomycin	Yes

The negative response for streptomycin does not mean that this drug is not usable but it does mean that the chances of its being effective are lessened Alternatively, the positive value for neomycin means that the probabilities of its being effective are increased as compared with the situation where the sensitivity is negative

From this point on, a value judgment of possible dangers from side effects must be made and the clinician's judgment brought into play

As another example, consider pneumonia due to Staphylococcus albus A check of Fig 1 shows that four groups are potentially useful In order, they are

Group III	86 per cent
Group I	74 per cent
Group IV	57 per cent
Group II	50 per cent

A test of the specific strain of the organism shows the following sensitivities

Group	Antibiotic	Sensitive	Average Per Cent Sensitivity of Staphylococcus Aureus
III	Chloramphenicol	Yes	86
I	Penicillin	No	67
	Erythromycin	Yes	81
	Bacitracin	No	85
	Novobiocin	Yes	85
	Oleandomycin	No	73
	Cyclamycin	Yes	83
IV	Neomycin	No	64
	Streptomycin	Yes	53
II	Tetracycline Group	Yes	38
	Nitrofurantoin	No	83

Certain of the antibiotics are ruled out immediately because of their restriction to topical use Of the others, the antibiotics to which the strain tested showed no sensitivity should be abandoned at least temporarily The remaining ones that might be suitable are

	Average Per Cent Response
Chloramphenicol	86
Erythromycin	81
Oleandomycin	73
Cyclamycin	83
Tetracyclines	39
Streptomycin	53

The average per cent response of strains of Staphylococcus albus to each of these antibiotics is comparatively meaningless as each of these showed a sensitivity So, a priori, one might argue that the probability of the strain responding to any one of the above antibiotics is 100 per cent In actual fact, there is a high, but not a 100 per cent correlation between the disc test and clinical response Presumably the higher the per cent of strains responding, the better the chance of a given strain responding clinically and hence the better chance if the antibiotics are used selectively

SUMMARY

A study of the comparative *in vitro* evaluation of the various antibiotics was attempted This study was not aimed at the accuracy of the disc method, but rather to examine the results available to the average medical practitioner

Routine antibiotic sensitivity reports were serially abstracted from laboratory day books, in two groups In the first, 1,000 consecutive reports were used to obtain relative abundance of various organisms and their mixtures (see Table I) The second group was used to obtain larger numbers of the rarer organisms for statistical study

These statistical analyses showed some interesting possibilities as to antagonism and symbiotic relationships (see

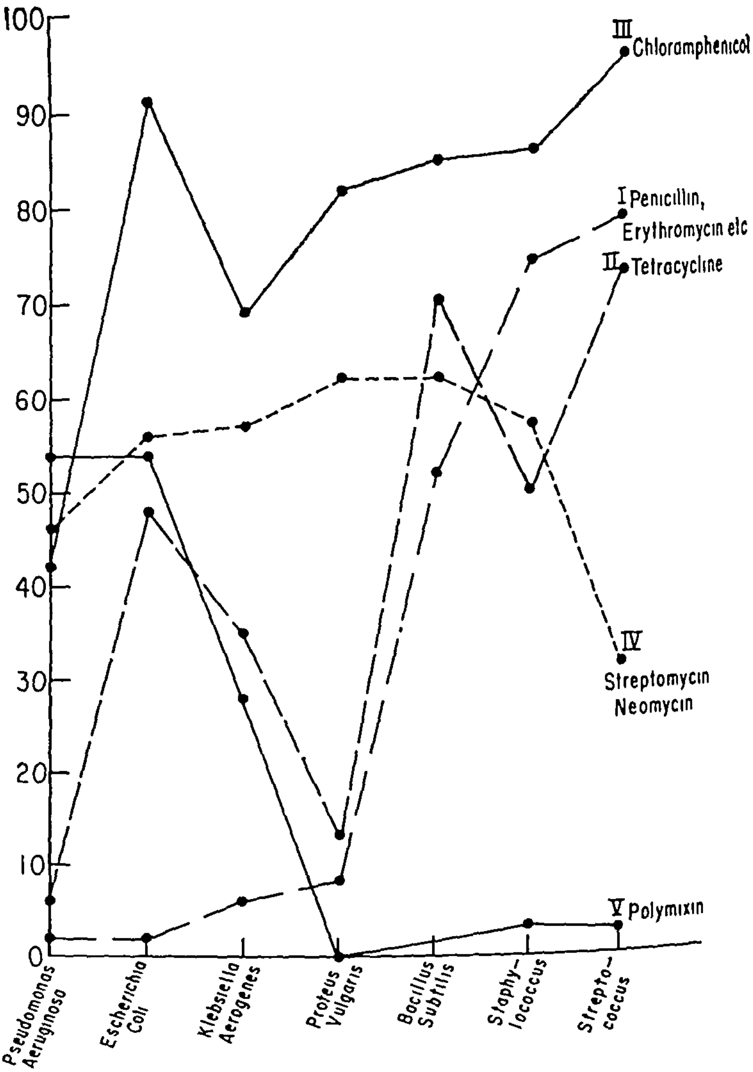

Fig 1

Table II) Staphylococcus albus and aureus, and Staphylococcus aureus and Streptococcus viridans showed potential antagonisms Catarrhalis Neisseria and Streptococcus viridans, Staphylococcus aureus and Hemophilus influenzae and finally Streptococcus viridans and Beta Hemolytic streptococci had a tendency for positive association, suggesting some symbiosis This was interesting in view of their occasional association in clinical disease

The results of the sensitivity tests against pure cultures of organisms was shown in Table III A profile of drug response versus species of bacteria was presented in Fig 1 This showed six groups, the significance of which is not understood

It would seem that these data would aid in the selection of the antibiotic of choice for either pure or mixed infection The method of combining the results of the sensitivity tests with the curves to select the drug with the statistically best possibilities of killing the infection, is demonstrated

CONCLUSION.

If Tables such as are here presented are prepared and used, the antibiotic therapy best able to care for an infection can be chosen on a statistical basis.

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PITFALLS IN THE DIAGNOSIS OF CANCER OF THE LARYNX*

WALTER H MALONEY, M D ,†

Cleveland, O

INTRODUCTION

Early diagnosis in cancer of the larynx must start with an appreciation of the importance of minimal complaints referable to this organ, in fact, there may be no complaints whatsoever, or they may appear unrelated, such as dysphagia, ear pain or a swelling in the neck The typical complaints of hoarseness or change in the character of the voice may not be constant or progressive They may vary in intensity as the associated benign or systemic disease varies with time and treatment Neglect of persistent minimal complaints and the tendency to attribute them to other causes such as vocal abuse, infection, sinus disease or allergy should be avoided

EXAMINATION

A high index of suspicion and awareness of the hidden areas of the larynx must be coupled with a thorough and accurate examination Double primary lesions are not limited to the related histologic types of epithelium but may be found distally The late diagnosis of this distal lesion will greatly effect the survival of your patient A benign appearing polypoid degeneration of the vocal cord will mask an early malignant change An inadequate mirror examination may be worse than none at all

Delay in direct laryngoscopy is another pitfall in the early diagnosis of cancer of the larynx This is the only way to examine adequately the hidden areas of the larynx Minimal

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mucosal changes or other benign appearing lesions which do not clear up under conservative therapy in the time interval you have come to expect, *i e* two to three weeks should be inspected and biopsied by direct laryngoscopy Cord motion is usually best evaluated by mirror examination but direct examination may confirm a suspected impairment of motion and find a lesion infiltrating the cricoarytenoid joint The pooling of secretions in the pyriform sinus may be the only clue to a lesion of the post-cricoid larynx or cervical esophagus

I believe the initial biopsy in an early suspicious lesion of the cord edge should be such that if it is benign you have not made the patient permanently hoarse It is better to rebiopsy even at the inconvenience of the patient than to destroy his speaking voice unnecessarily This does not mean that deep biopsy should not be done if the lesion persists or the pathologist reports activity indicative of malignant changes in adjacent areas These deeper and more radical biopsies should be reserved for the second and third examinations in early lesions For lesions located other than on the cord edge deep biopsy is acceptable and is to be preferred

While local anesthesia is used in a majority of cases, the apprehensive patient and those with short thick necks may require general anesthesia, and I have found that Fluothane, preceded by the ordinary local anesthesia, is best in my hands, however, general anesthesia is no substitute for good endoscopic technique It will not eliminate the need for appreciation of minimal mucosal change, a careful unhurried examination, and a precise, accurate and adequate biopsy One must guard against inadequate instrumentarium and untrained assistants Retrograde and fore-oblique telescopes are useful in visualizing the hidden parts of the larynx Be certain to inspect all of the larynx and not be fooled into stopping the examination at the first appearance of pathology Remember the multiple primaries and malignancies hidden under benign disease

There are other modes of examination of the larynx, and failure to use them will delay the early diagnosis of cancer of the larynx The first is the use of laminograms These are especially valuable in the detection of subglottic and ventricular lesions Double contrast studies and cinefluography using bronchographic solutions such as Dionosil are of value Stroboscopic examinations have been used by some to good advantage Cytologic examination of secretion taken directly from the cords may help Finally, exploratory thyrotomy may be necessary to establish the diagnosis where all else has failed

RECURRENCE AFTER IRRADIATION

Previous irradiation complicates the early diagnosis of recurrence of malignancy in the larynx This is even more true if you did not have the opportunity to see the original lesion The picture usually resolves to one of edema, hyperplasia, distortion of the soft tissues and continued hoarseness, pain, and dysphagia This progresses to the point where you cannot be reasonably certain that there is residual disease, recurrent disease or only the benign complications of irradiation In these problems I feel we must continue to pursue a course of diagnostic work to establish the exact state of disease

If within eight to ten weeks after completion of therapy there is abnormal tissue visualized, biopsy should be done again and repeated if negative until the suspicious lesion is resolved If there is only edema and swelling which persists, the timing of repeat biopsy must be correlated with the ultimate surgery If there is a possibility of partial laryngectomy, we must be early in our biopsy to make the diagnosis before the lesion passes beyond the limits of partial resection If total laryngectomy is contemplated we usually have time to allow further resolution of the edema since the danger of metastasis is lessened by the obliteration of lymphatic pathways by the primary irradiation and conservation of function of the larynx is no longer an issue

RECURRENCE AFTER SURGERY

Another problem is that of diagnosis of recurrent disease after previous surgery The careful histologic examination of the operative specimen may point toward the site most likely to have recurrence or residual disease, *i e*, the cut edge with hyperactivity of the basal layer of cells, abnormal nuclei, and hyperkeratosis In these cases, early biopsy of any abnormal tissue is indicated and if positive allow a more conservative approach to therapy Previous surgery may also complicate the problem by burying the cut edge with residual disease deep in normal tissue It may distort the anatomy so that casual inspection is not feasible, and each examination necessitates inconvenience and discomfort to the patient The practice of pulling arytenoid mucosa into the larynx to cover denuded areas often complicates the examination and airway, therefore, an effort should be made at surgery to leave an operative field which is accessible to re-examination

Both post-irradiation and post-surgical problems are complicated further when there is obstruction of the airway by edema and scarring Repeated attempts at biopsy may very likely precipitate the need for tracheotomy In such cases I prefer to defer biopsy for as long as possible and reduce the edema to its fullest prior to examination

When direct laryngoscopy and biopsy fail, the next step is exploratory thyrotomy with frozen section and a previous agreement with the patient as to contemplated surgery Again, we must consider that if the tissue is benign that there is a possibility that the postoperative edema may decrease the airway to the point where tracheotomy may be necessary Surgical exploration of an irradiated larynx may cause necrosis of the cartilage

CHRONIC LARYNGITIS

The chronic irritated larynx raises a special problem First, careful periodic examination is essential to establish a base line In turn, deviation from this basic picture then becomes important and warrants investigation I believe that the equivocal lesion which does not resolve must be biopsied and rebiopsied until it either resolves or a positive diagnosis is made If it does resolve then we must remember that there may be multiple foci or pre-malignant change and periodic re-examination becomes important and rational, moreover, the precursors of malignancy, *i e*, excessive smoking, infection, and vocal abuse must be guarded against

PREVIOUS BIOPSIES

The problem of previous biopsies raises both a technical problem and a personal problem, inasmuch as we now must consider the capability of the referring endoscopist I do not think that anything but an obvious gross lesion should be biopsied by other than the physician charged with the decision as to ultimate therapy Biopsy may complicate the picture by doing the following things *1* introduce infection, *2* cause edema and swelling, thus limiting motion, *3* remove and eliminate the only suspicious tissue which might urge you on to further studies As a result this latter presents your consultant with an apparently benign picture but in reality one with a hidden lesion which must await further growth before it can be discovered Finally, it may destroy the margins of the lesion and make it necessary to do more radical surgery than might have been necessary if the true edge of the lesion could have been adequately visualized

PATHOLOGY CONSULTATION

Close co-operation with your pathologist may make the difference between success and failure in early diagnosis Take the time to give him a careful history and discuss with him the general factors in the case Make him aware of other associated disease such as hypothyroidism, tuberculosis, and biopsy material from other seemingly unrelated procedures Evaluate the degree of infection present so that he in turn can evaluate its relationship to cellular activity We must be aware that cellular changes such as dyskeratosis, hyperkeratosis and atypical maturation, hyperactivity of the nuclei and irregularity of the basal layer are changes seen next to malignancy and indicate the need for repeated biopsies in the adjacent areas Careful orientation of the biopsy specimen in relation to the whole larynx will be of great help to him This is done by careful labeling of specimens, use of drawings, and even the attendance of the pathologist in the operating room at the time of examination

Previous positive biopsy done elsewhere should be reviewed by you and your own pathologist If not absolutely conclusive a second biopsy should be obtained by the operating surgeon since the medical-legal responsibilities are such that he must be certain beyond any doubt that he has an accurate diagnosis This same index of suspicion applies to the benign diagnosis as well as to the malignant one

The pathologist is often placed in a most difficult position when we expect him to deliver us a clear cut unequivocal diagnosis The borderline problem of hyperkeratosis, atypical maturation, associated infection and irregular sectioning of the tissue block may make it impossible to be didactic, therefore, we should make this problem one for continued orientation and study with the pathologist He should be encouraged to state his observation precisely, accurately and objectively, then he should draw his conclusions We in turn must be prepared to take these same findings with his impression, make our own diagnosis and assume the responsibility for the final decision

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THE JAMES E NEWCOMB AWARD

Dr Joel J Pressman, of Los Angeles, was chosen by the members of the American Laryngological Association at its annual meeting recently, to be the recipient of the James E Newcomb Award for his outstanding contribution to research in otolaryngology examination becomes important and rational, moreover, the precursors of malignancy, *i e*, excessive smoking, infection, and vocal abuse must be guarded against

PREVIOUS BIOPSIES

The problem of previous biopsies raises both a technical problem and a personal problem, inasmuch as we now must consider the capability of the referring endoscopist I do not think that anything but an obvious gross lesion should be biopsied by other than the physician charged with the decision as to ultimate therapy Biopsy may complicate the picture by doing the following things *1* introduce infection, *2* cause edema and swelling, thus limiting motion, *3* remove and eliminate the only suspicious tissue which might urge you on to further studies As a result this latter presents your consultant with an apparently benign picture but in reality one with a hidden lesion which must await further growth before it can be discovered Finally, it may destroy the margins of the lesion and make it necessary to do more radical surgery than might have been necessary if the true edge of the lesion could have been adequately visualized

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If treatment was based on radical extirpation of the larynx alone in every extensive lesion regardless of location or extent of tumor, one could say that any data on accuracy of diagnosis is immaterial since any other surgical procedure would not be considered, however, conservation of laryngeal function by varied surgical approaches to the supraglottic and hypopharyngeal areas should be possible if accurate appraisal of the extent of the primary lesion can be made How often have we been impressed by freedom of involvement of tumor in the true cords area with supraglottic cancer after total laryngectomy, and wonder whether a supraglottic operation might not have been better?

In order that laryngograms demonstrate their true value to the clinician, graphic objective findings should highly correlate with pathology specimens, furthermore, if laryngograms are that important, one should be able to separate lesions which can be operated upon by less radical procedures (supraglottic subtotal laryngectomy or transhyoid partial laryngo-pharyngectomy) which conserve function

Conversely, if such radiographic findings might indicate a more radical operation (inclusion of neck dissection), it would follow that this diagnostic measure is of immense importance To back these assumptions, a brief review of surgical pathology data will be presented from our cases operated upon by uniform treatment during the last decade

PATHOLOGIC SIGNIFICANCE OF LOCATION AND SIZE OF PRIMARY LESION

Classification of laryngeal lesions differs in various sections of the country and over the entire world[1-11] One with any experience in laryngeal cancer will admit that we have

LARYNGOGRAMS THEIR VALUE IN THE DIAGNOSIS AND TREATMENT OF LARYNGEAL LESIONS

A Study Based on Clinical, Radiographic and Pathologic Findings on 99 Patients with Cancer of the Larynx*†

JOSEPH H OGURA, M D,
WILLIAM E POWERS, M D, (By Invitation),
SUMNER HOLTZ, M D, (By Invitation),
MALCOLM H MCGAVRAN, M D, (By Invitation),
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and
RICHARD VOORHEES, M D,‡ (By Invitation),

St Louis, Mo

The laryngogram, a method for objective radiographic demonstration of laryngeal lesions, has been used by the radiologist to delineate masses within and about the larynx, to indicate extent of the lesion and to demonstrate impaired function of adjacent structures This method of examination should not be used as a screening procedure, but it should serve as an ancillary aid in diagnosis and treatment Its value to the radiotherapist is to document objectively the effects of radiation on malignant tumors

The otolaryngologist has resorted to indirect and direct examination and biopsy procedures to establish the nature of the lesion and institute appropriate surgical measures On occasions, he has utilized tomography to show soft tissue densities, but this has the disadvantage of not clearly demonstrating the detail of certain specific structures under question, *e g*, false cord and ventricle with false cord tumors

Many benign lesions within the larynx have been adequately

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I Glottic—cancers limited to the true cord

II Infraglottic—cancers of the subglottis and glottis

III Supraglottic—cancers involving the false cord, fixed and free portion of the laryngeal surface of the epiglottis

IV Transglottic—cancers that cross the ventricle, thus involving two or three of the above sites The rare ventricular tumor is included in this group

This classification has been based on areas of involvement and does not include the hypothetical extrapolation from size and location as to the point of origin It is based on where tumor *is* rather than a supposition as to where it began The glottic, infraglottic and supraglottic types are similar to that used by Pietrantoni[4 5]

This classification area scheme has the value of separating lesions according to two major divisions of lymphatic drainage Of the 96 patients studied, the following facts were noted

There was a progressive increase in metastatic incidence from glottic, infraglottic, supraglottic and transglottic, 0, 19, 33 and 52 per cent respectively The metastatic rate of the transglottic group represents the addition of the infraglottic and supraglottic, confirming the expected results derived from theoretical considerations of the lymphatic drainage of the larynx

The transglottic group yielded a higher incidence of both clinical apparent and inapparent metastases The metastatic rate for inapparent metastases for the infraglottic group was 5/23 or 22 per cent, the supraglottic 1/25 or 4 per cent and the transglottic was 5/16 or 31 per cent Supraglottic and infraglottic cancers larger than 2 cms and of moderate or poor differentiation also constituted a high incidence of metastases (15/39) Cancers in these two locations that are well differentiated of any size, those of 2 cms or less and moderately or poorly differentiated provided a poor yield of metastases to the neck (two of 20) Of the 23 patients with supraglottic or transglottic lesion shown to have cervical two serious problems first, the clinical classification of cancer of the larynx, second, its relationship with the metastatic rate Each proposed classification scheme has its advantages and disadvantages We can best illustrate this situation if we have 100 laryngeal cancers and they are split into five or ten groups, and then into clinical stages

Such information derived, while specific, will be based on a small limited experience of that individual While several methods for study of metastatic spread have been reported, seldom has the information been derived from a homogenous sample where the treatment has been uniform

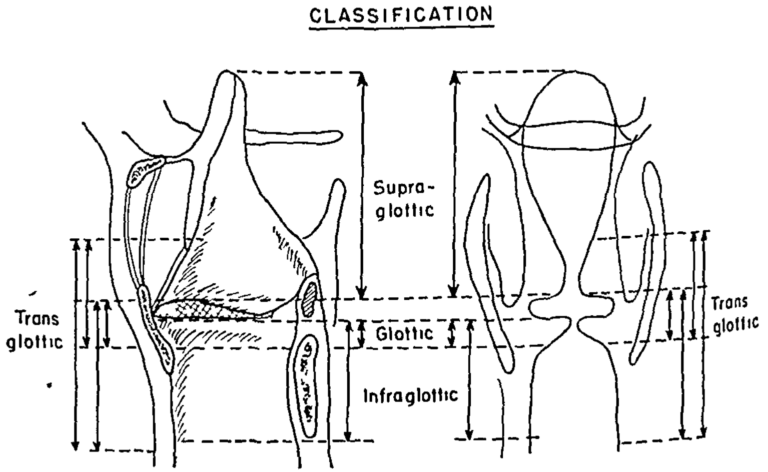

Fig 1 Clinical, pathologic and laryngographic classification

Recently McGavran, Bauer and Ogura[12] have reviewed the specimens of laryngeal cancers where *en bloc* total or supraglottic subtotal laryngectomy with incontinuity neck dissection was performed between 1950-1959 No case with prior radiation treatment or other surgical intervention has been included All ary-epiglottic fold and pyriform sinus cancers known to have a high metastatic rate and cancers earlier than 1950 are excluded in this pathologic study

The proposed classification (see Fig 1) based on pathologic data are diagnostic validity of laryngograms, it necessarily follows that the clinical findings, radiographic, and pathologic data are documented separately.

Ninety-nine laryngograms from 1957-1959 were examined on proven epidermoid carcinoma of the larynx where subsequent surgery was performed, and the specimens were available for comparison Six of 13 glottic lesions were hemilaryngectomies, and the remainder was incontinuity neck dissection with total or subtotal laryngectomy Without regard to previous interpretation these films were reviewed by the radiologists

All of the laryngograms were re-examined without regard to known classification or knowledge of clinical data There was never any major difference of opinion between the radiologists, however, if opinions differed over certain small structural involvement, *e g*, false cords or anterior commissure, such differences were resolved into one opinion and recorded together with a drawing of the size and shape of the tumor. The following facts were noted

I *Classification*

1 Glottic
2 Infraglottic
3 Supraglottic
4 Transglottic
5 Pyriform sinus

II *Area of Involvement*

1 Size
2 Structures involved
3 Adjacent structures invaded or free
4 Upper and lower border of tumor
5 Mobility of cords
6 Cartilage destruction
7 Anterior commissure and across midline
8 Pyriform sinus, ary-epiglottic fold and base of tongue

These were then placed into various classifications, the size estimated in the superior-inferior extent, and notations were metastases, 11 (48 per cent) had proven positive nodes in the contralateral side of the neck on follow-up surgery Consideration in these cases should be given to bilateral neck dissection (one or two stage)

Supraglottic tumors tended to be the "pushing" type rather than the "infiltrating" type, particularly when they are relatively well differentiated They had sharp margins of extent, and showed less tendency to metastasize

Thus cases of laryngeal cancer can be separated into groups with high and low metastatic rates by using preoperative information about site, size, biopsy differentiation and clinical evaluation of cervical lymph nodes

TECHNIQUE OF LARYNGOGRAMS

The procedure used was essentially that as reported by Powers, McGee and Seaman[13] Since 1958, however, we have modified the technique by using 10 per cent cocaine for topical anesthesia rather than 5 per cent cyclaine The standard method of anesthesia of the hypopharynx and larynx is used with the Jackson cross-action forceps and 2 cc of cocaine is dropped into the larynx The use of oily dionisil gives the best coating and has the theoretical advantage of minimizing granuloma formation in the lung

Ten to 20 cc of oily dionisil is dropped slowly over the tongue intermittently during inspiration Frontal and lateral spot films are made using high kilovoltage during phonation, inspiration, Valsalva and modified Valsalva maneuvers

There are some difficulties inherent with this simple method of performing laryngograms (In 2 per cent of our cases, it was impossible to perform a laryngogram because of the apprehensiveness of the patient) It is important to stress that laryngograms should be performed *before* biopsy Edema following laryngoscopy and biopsy will cause some distortion on the films

METHOD OF STUDY

Since this study was designed to appraise accurately the diagnostic validity of laryngograms, it necessarily follows that the clinical findings, radiographic, and pathologic data are documented separately

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8 Pyriform sinus, ary-epiglottic fold and base of tongue

These were then placed into various classifications, the size estimated in the superior-inferior extent, and notations were made of the adjacent structural involvement Comment was made whether radical excision in supraglottic or pyriform sinus lesions was necessary or conservation of function operation could be performed (supraglottic subtotal or transhyoid partial laryngo-pharyngectomy) The clinical and direct laryngoscopic observations were then recorded from the hospital record on a master sheet and final comparisons made with the gross photograph of the specimen and the pathology records

During the 1957-1959 period, the following cases were excluded from this study

Seven laryngograms made on laryngeal cancer during this period were discarded for the following reasons *1* poor coating, two cases, *2* poor quality or insufficient views, two cases, *3* following irradiation with edema and distortion, two cases, *4* tracheotomized patient—insufficient coating and poor airway, one case Four laryngograms were done and surgery performed, but these could not be found in the X-ray file It was impossible to do laryngograms on two patients because they were uncooperative

Twenty-five patients had laryngograms, but these were not included since irradiation was the treatment of choice, and no specimen was available Ten other cases had far advanced cancer and surgery was not considered

Twenty patients with laryngeal cancers underwent surgery without laryngograms during this period Three laryngograms were done where neither surgery nor X-ray therapy was given to the patient

These represented a total of 170 cases of all forms of laryngeal cancer during this three-year period

RESULTS

The lesions demonstrated by laryngograms were classified on the basis of certain structural involvement according to the foregoing proposed pathologic classification Accuracy was verified by correlating these findings with the surgical specimens and gross photographs

Errors were listed if the laryngographic interpretation resulted in a different classification because of certain adjacent structural involvement incorrectly appraised

Clinical information was derived from the hospital records as to physical examination and direct laryngoscopic examination These were listed as to structures involved These findings represented the combined notes of the residents and the attending surgeon The same criteria for accuracy and errors were used as were used for laryngograms

TABLE I

Classification	Clinical and Direct Laryngoscopy	Laryngograms	Surgical Pathology
Glottic—			
Correct	12	12	13
Errors	1	1	
Infraglottic—			
Correct	7	9	14
Errors	7	5	
Supraglottic—			
Correct	22	28	29
Errors	7	1	
Transglottic—			
Correct	10	16	16
Errors	6	0	
Pyriform Sinus—			
Correct	26	26	27
Errors	1	1	
Total—			
Correct	77 } 22%	91 } 8%	99
Errors	22	8	

Glottic (See Table I)

Clinical evaluation of true cord involvement as to extent, and fixation of the cord was accurate in 12 of 13 cases One error was recorded Subglottic extension was absent in the specimen

Laryngograms on cordal lesions were accurately demonstrated in 12 of 13 cases (see Fig 2) In one, the false cord was thought to be involved It is important to stress the side

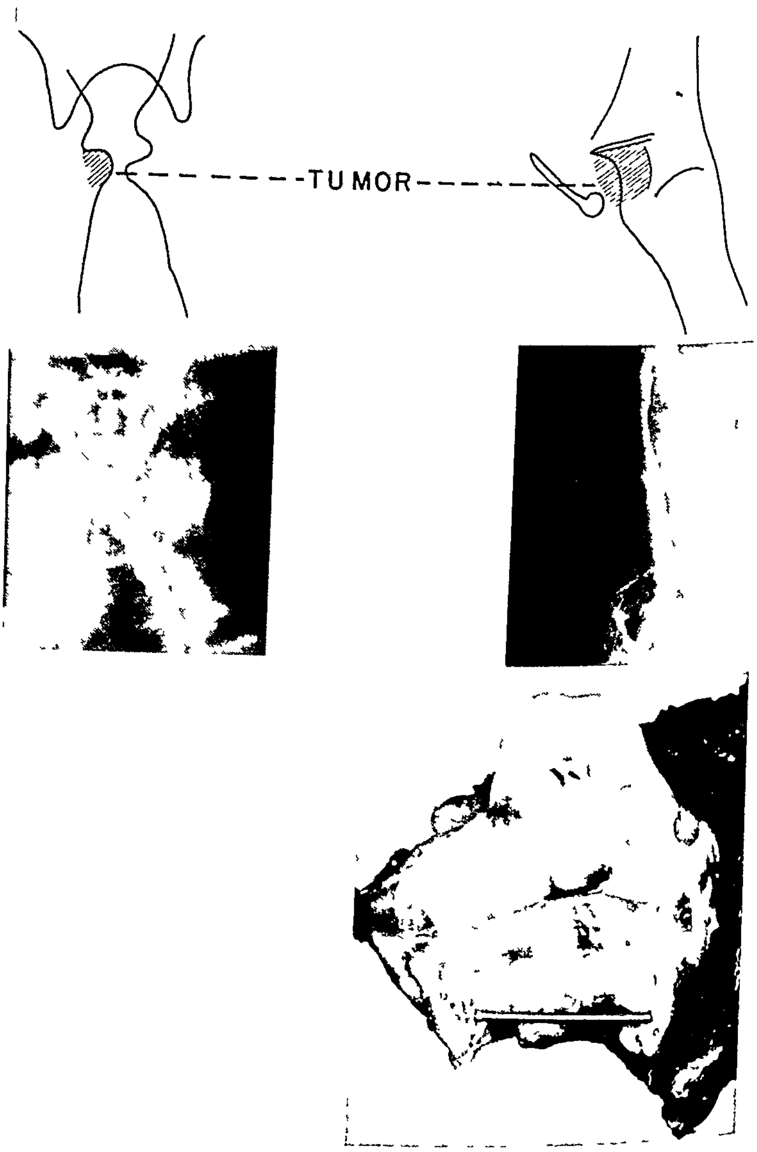

Fig 2 Glottic Frontal view—Note the thick right true cord The right ventricle is smaller than the left There is no subglottic extension and the false cords are normal

Lateral view—There is irregularity of the anterior portion of the true cord and a double contour anteriorly indicating anterior commissure involvement

Specimen—Tumor is confined to the anterior two-thirds of the true cord and extends several millimeters into the subglottis of involvement was called in every instance The mobility of the cord as demonstrated by indirect examination correlated with the laryngograms in every instance

Infraglottic (See Table I)

Accuracy both as to clinical and laryngogram data was least in this group Clinically, a correct diagnosis was made in seven of 14 patients There were seven errors In two instances of cordal fixation, the subglottic mass was not suspected until a biopsy was taken blindly from the subglottic area In five instances, false cord involvement was suspected, and the true nature demonstrated by the negative biopsy of this area

Laryngogram accuracy was demonstrated in nine of 14 cases (see Fig 3) Four errors were primarily made in classifying these lesions as transglottic since the false cords were thought to be involved Non-filling of the ventricle and edema distortion of the false cord (post laryngoscopic biopsy) may account for some of these errors One small subglottic tumor was missed by laryngograms In five cases, the tumor was demonstrated across the midline by laryngograms and in the specimens

Supraglottic (See Table I)

From clinical information on 29 patients, 22 were accurately placed in this group Five of the 29 extended into the vallecula or anterior pyriform sinus (see Fig 4) All seven errors were in the true cord area, where true cordal involvement was thought to be present

With laryngograms, 28 of 29 were correctly placed in the supraglottic group, and one error was recorded where the true cord was thought to be involved In 17 instances, the radiologist was able to appraise that conservation of function operations could be performed These 17 cases correlated with the actual number of conservation of function operations performed All 17 cases had a good margin of safety in the surgical specimen Twelve of this group were in the area of the petiole and higher (see Fig 5) and five were in the false cord area and above In three instances of the five inferior

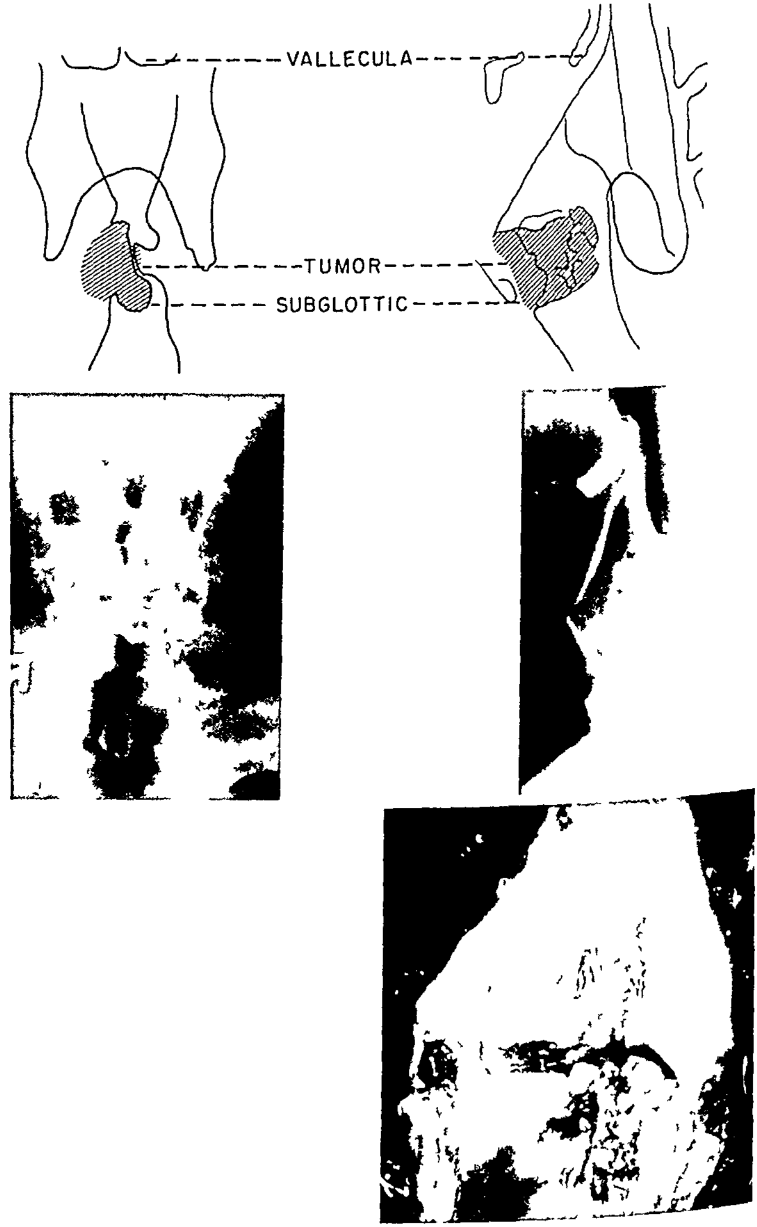

Fig 3 Infraglottic Frontal view—An exophytic tumor replaces the right true cord and involves the subglottic area The ventricle is partially obliterated by the mass but the lateral portion of the ventricle and false cord appears normal

Lateral view—The bulky tumor is seen in its full subglottic extent The anterior commissure is involved with tumor

Specimen—Note the obliteration of the ventricle by the cordal-subglottic tumor

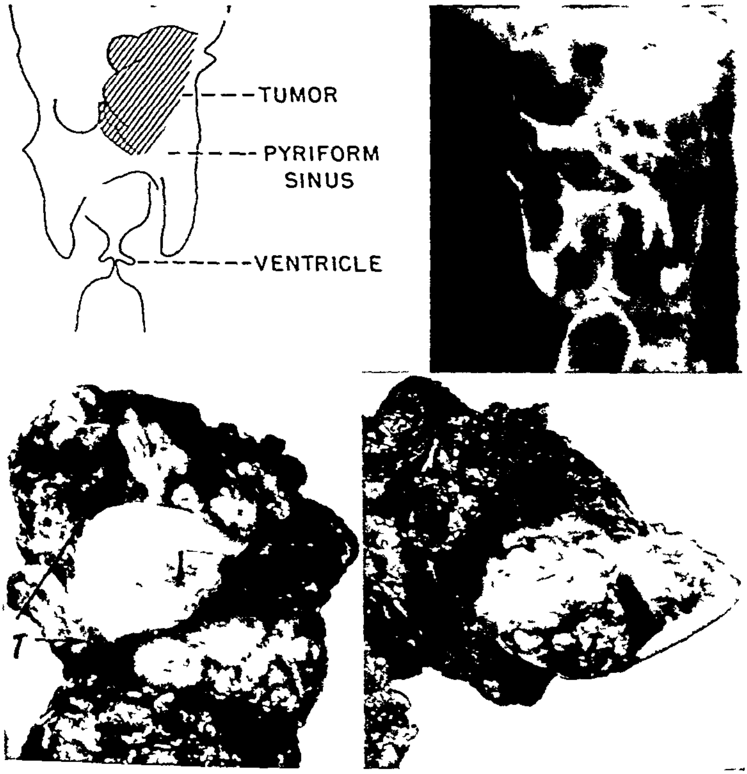

Fig 4 Supraglottic Frontal view—The right vallecula is distorted and obliterated by a tumor mass that extends down onto the left medial margin of the epiglottis The tumor extends to the midline The true and false cords are normal There is an adequate inferior margin of safety for the conservation operation

Specimen—Note the tumor (T) extends from the lateral wall of the epiglottis to the left vallecula

supraglottic tumors operated upon by the supraglottic operation, the laryngogram findings gave the surgeon confidence that the operation could be performed (see Fig 6)

Of the remaining 12, obviously the surgeon felt that laryngectomy and neck dissection was the safest procedure From the laryngographic evidence it would have been theoretically possible to perform a supraglottic subtotal operation in 11, and only one, invasion of the anterior commissure area, was so close a subtotal operation could not have been considered There was an error in clinical evaluation in six of the 11 surgical specimens (These were not listed as additional

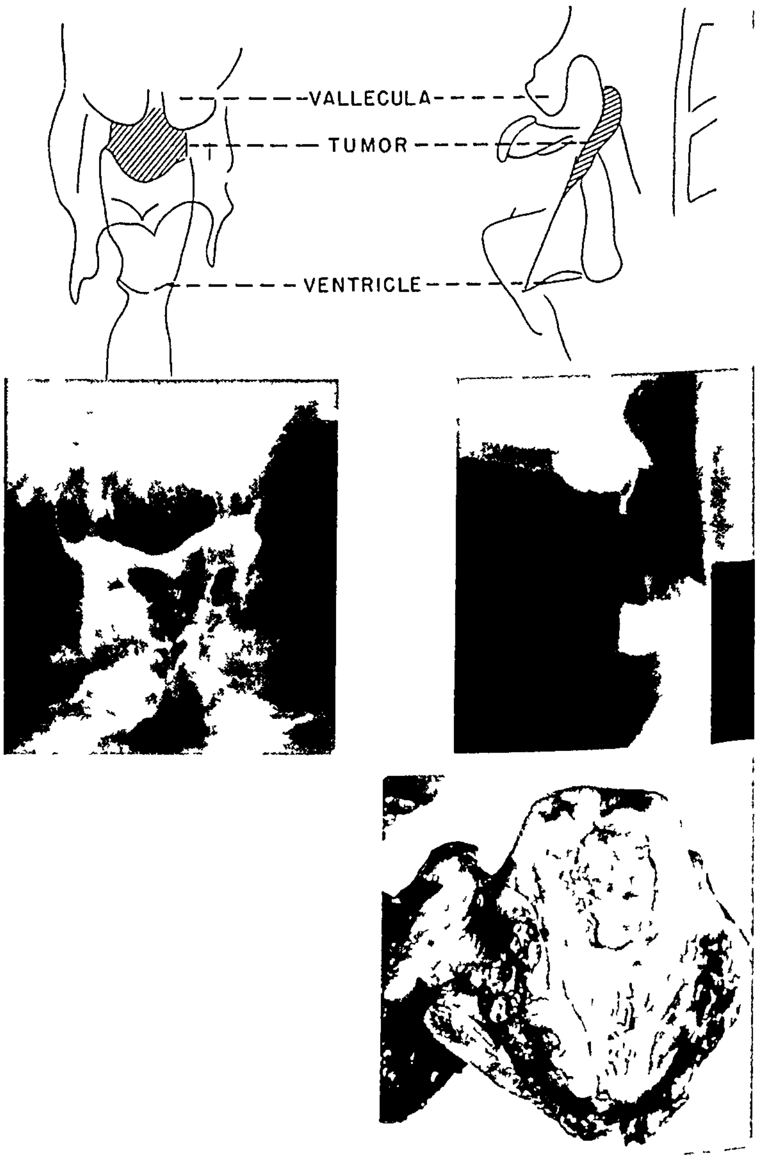

Fig 5 Supraglottic Frontal view—There is an ill-defined density in the midline on the free portion of the epiglottis The lower border of the tumor is high above the cords

Lateral view—The laryngeal surface of the epiglottis is irregular and the epiglottis is thickened The tumor is seen to extend to just superior to the petiole

Specimen—Note that there is a wide margin of safety in the specimen

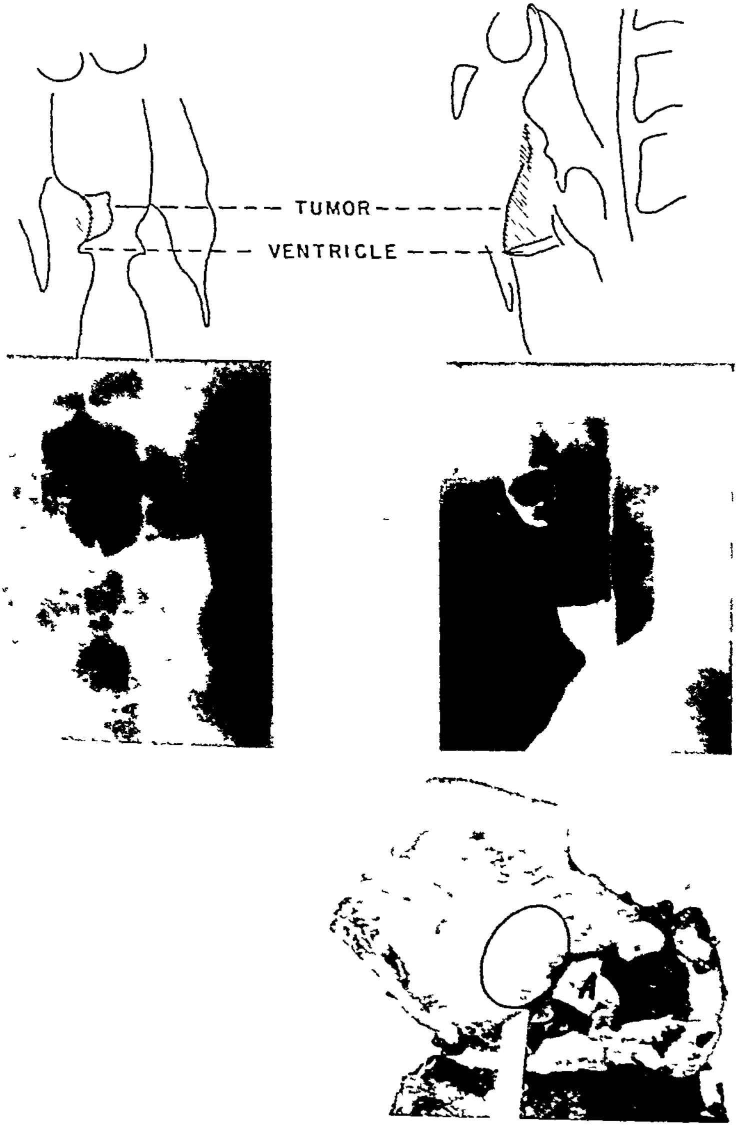

Fig 6 Supraglottic Frontal view—A tumor mass is present on the right false cord The ventricle and true cord are free of tumor There is an adequate margin of safety for a conservation operation

Lateral view—Note the tumor extension on the false cord

Specimen—The wooden applicator is inserted into the ventricle A—arytenoid errors) An adequate margin of safety to the true vocal cords was present in those subjected to total laryngectomy (see Fig 7), however, these operations were performed before the significance of laryngogram findings was apparent In the remaining six instances of total laryngectomy, the surgical margin to the true cord area was too narrow to justify this conservation operation (see Fig 8)

In 23 instances the tumor was in the midline In six instances, the unilateral position of the tumor was demonstrated both by laryngograms and in the specimen

Transglottic (See Table I)

In the group of 16 patients, clinical accuracy was assessed correctly in ten, but it was missed in six instances Subglottic involvement was not suspected when the true cord was fixed in three cases False cord and ventricle involvement was missed in two instances, and base of epiglottis invasion was missed three times In two cases, two adjacent area involvements were simultaneously present and not suspected In two additional instances only "slight subglottic extension" was suspected when actually extensive infraglottic involvement was demonstrated by laryngograms These two were not listed as additional errors to the six listed

Laryngogram accuracy was highest in this group There were no errors in classification (see Fig 9) The four errors listed under the infraglottic group were actually called transglottic because of suspected false cord invasion

The majority of these tumors were unilateral In four instances, the tumor crossed the midline on laryngograms and this was confirmed on the specimen

Pyriform Sinus (See Table I)

There were 27 pyriform sinus cancers in this group While these tumors are hypopharyngeal and are not strictly laryngeal in origin, these have been included in this study since surgical treatment employed is most frequently laryngectomy and neck dissection (see Fig 10), use of partial or complete skin grafts for complete reconstruction of the pharynx, or

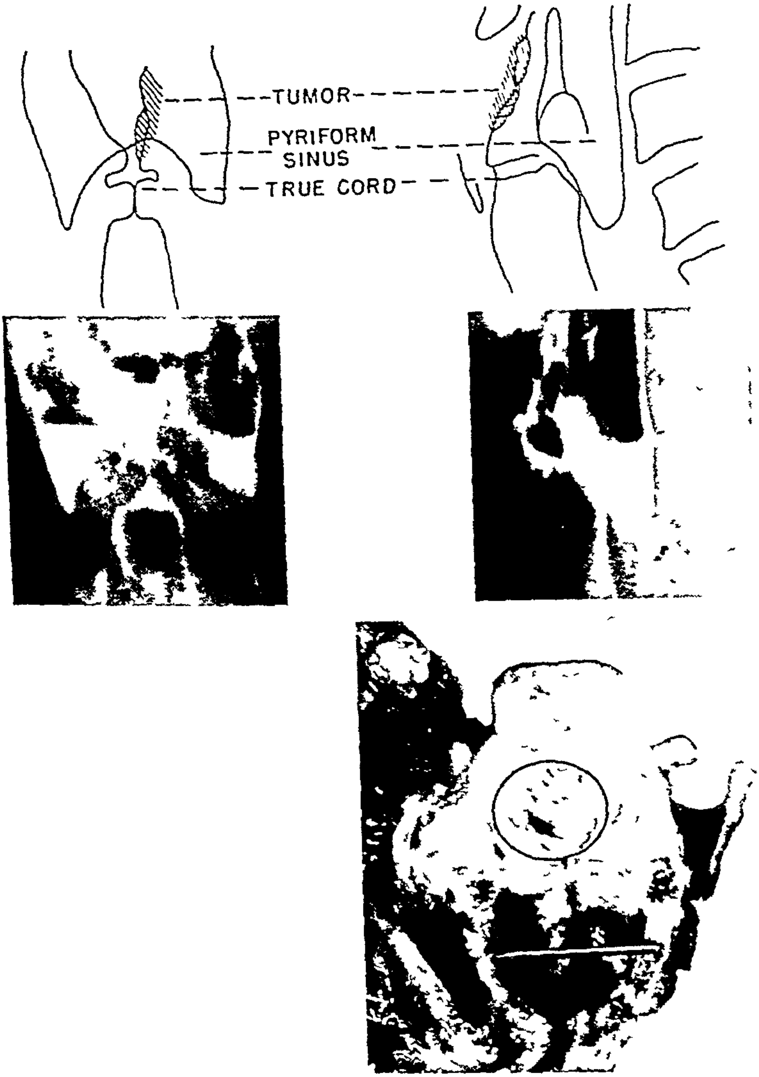

Fig 7 Supraglottic Frontal view—On the superior portion of the false cords there is narrowing of the vestibule and irregularity of the mucosal surfaces indicating presence of tumor The true cords subglottic area and ventricles are free of tumor There is a good margin of safety for a conservation operation

Lateral view—The laryngeal surface of the epiglottis is lobulated and irregular The tumor extends below the petiole but well above the ventricles

Specimen—Note that it was possible to perform a supraglottic operation

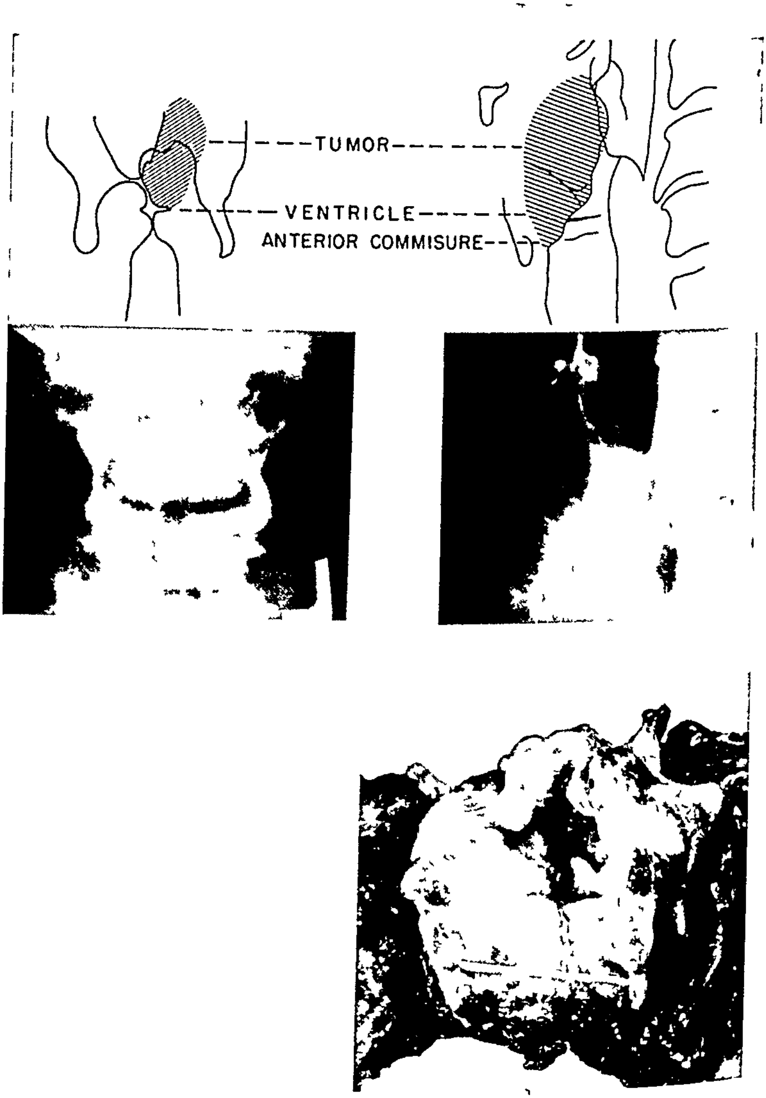

Fig 8 Supraglottic Frontal view—A tumor mass distorts the laryngeal vestibule bilaterally but mainly on the right The true cords are free of tumor

Lateral view—A bulky ulcerated tumor involves nearly the entire laryngeal surface of the epiglottis with invasion of the pre-epiglottic space The inferior margin of the tumor extends into the anterior commissure There is no margin of safety for a conservation operation

Specimen—Note extensiveness of the tumor with marginal invasion of the anterior commissure

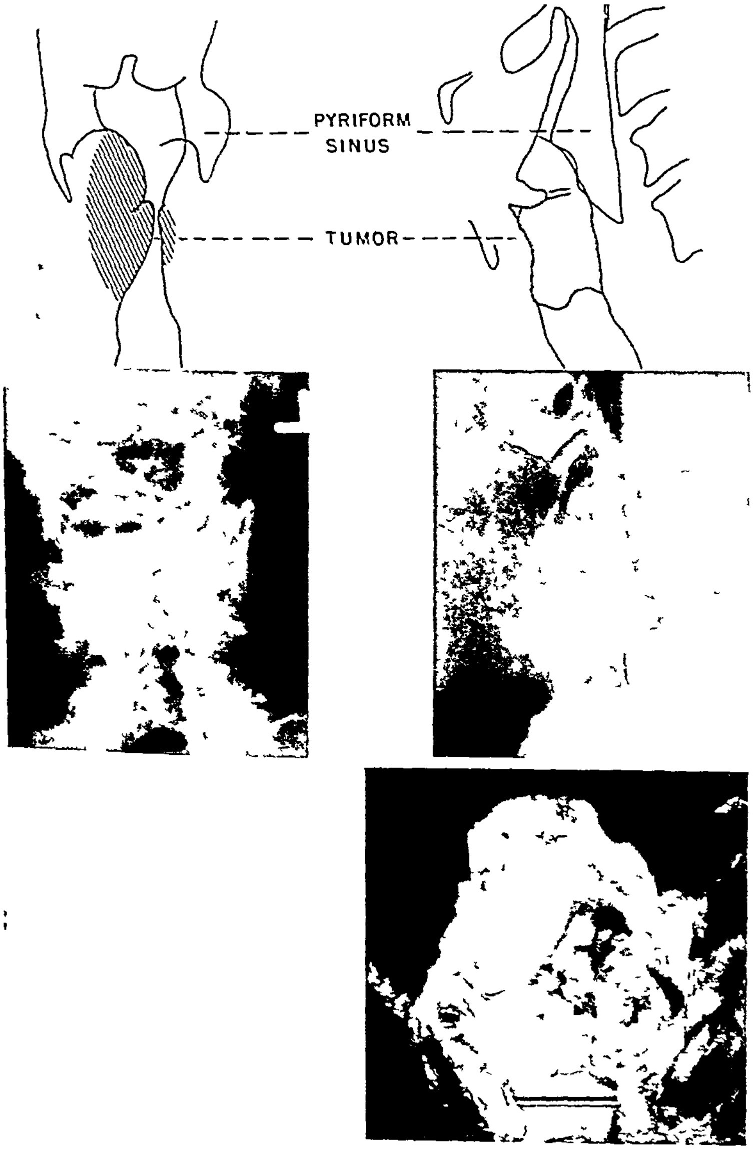

Fig 9 Transglottic Frontal view—The right laryngeal ventricle is obliterated by tumor which extends from above the false cord to a point two centimeters into the subglottis The cord is fixed

Lateral view—The tumor mass begins above the false cord The inferior margin is near the lower border of the cricoid cartilage Note that the anterior subglottic area is irregular The thyroid cartilage is destroyed

Specimen—Note the extensive tumor crossing the ventricle and cords

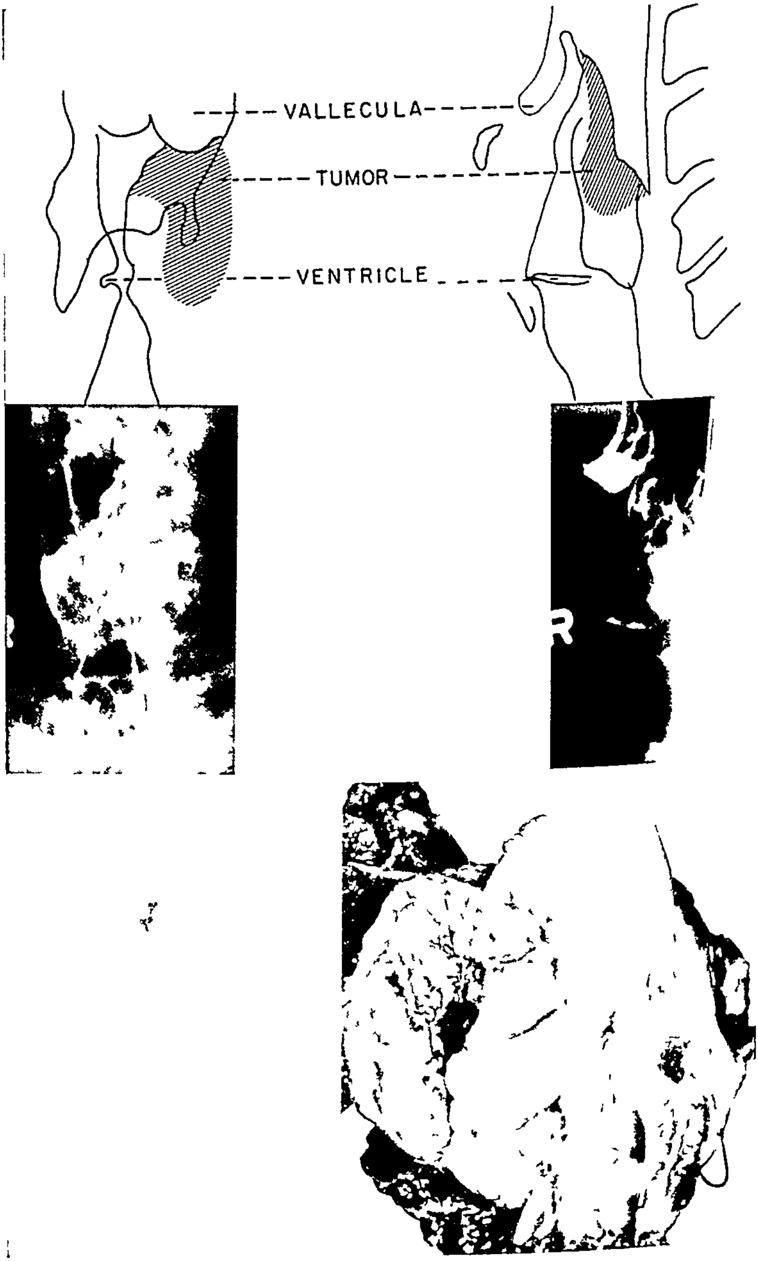

Fig 10 Pyriform Sinus Frontal view—The left pyriform sinus is almost completely replaced by a circumferential tumor that extends from the level of the vallecula to the apex of the pyriform sinus There is tumor involvement of the ary-epiglottic fold The vestibule false cords and true cords are displaced and edematous

Lateral view—The free edge of the epiglottis and the ary-epiglottic fold are irregular due to tumor The anterior wall of the pyriform sinus is involved

Specimen—Note the large size of the pyriform sinus carcinoma conservation of function, *i e*, transhyoid partial laryngopharyngectomy

Accuracy of diagnosis in this group has been high Clinically and radiologically in 26 of 27 cases the lesions were accurately placed In six of the 27, conservation of function operation was performed These were correctly described in five of the six cases by the radiologist

The following criteria were used for conservation of function operation These tumors should be small (under 2 1/2 cms), be free of thyroid cartilage invasion, demonstrate mobility of the arytenoid, and have a tumor-free apex in the pyriform sinus (see Fig 11) Preferably they should be placed on the lateral hypopharyngeal wall Direct examination of the posterior cricoid area should show no evidence of tumor

Clinically one error was found A small 1 cm lateral wall tumor was missed completely and was picked up by the radiologist (see Fig 12) The lone laryngogram error was an edematous ary-epiglottic fold together with non-filling of the pyriform sinus The tumor was interpreted primarily on the ary-epiglottic fold, actually it was on the medial wall of the pyriform sinus

SIZE OF PRIMARY LESION FROM LARYNGOGRAMS

The correction factor for magnification of the laryngeal structures on roentgenograms was determined by using a radio-opaque measuring ruler or a metal object of known size This factor for distortion was 20 per cent for the frontal views and 33 1/3 per cent for the lateral views All laryngogram measurements of the tumor were made of the superior-inferior extent, and no attempt was made to measure the transverse or anterior-posterior dimension

All laryngographic measurements were recorded first and final accuracy compared with the pathology records and gross photographs for orientation

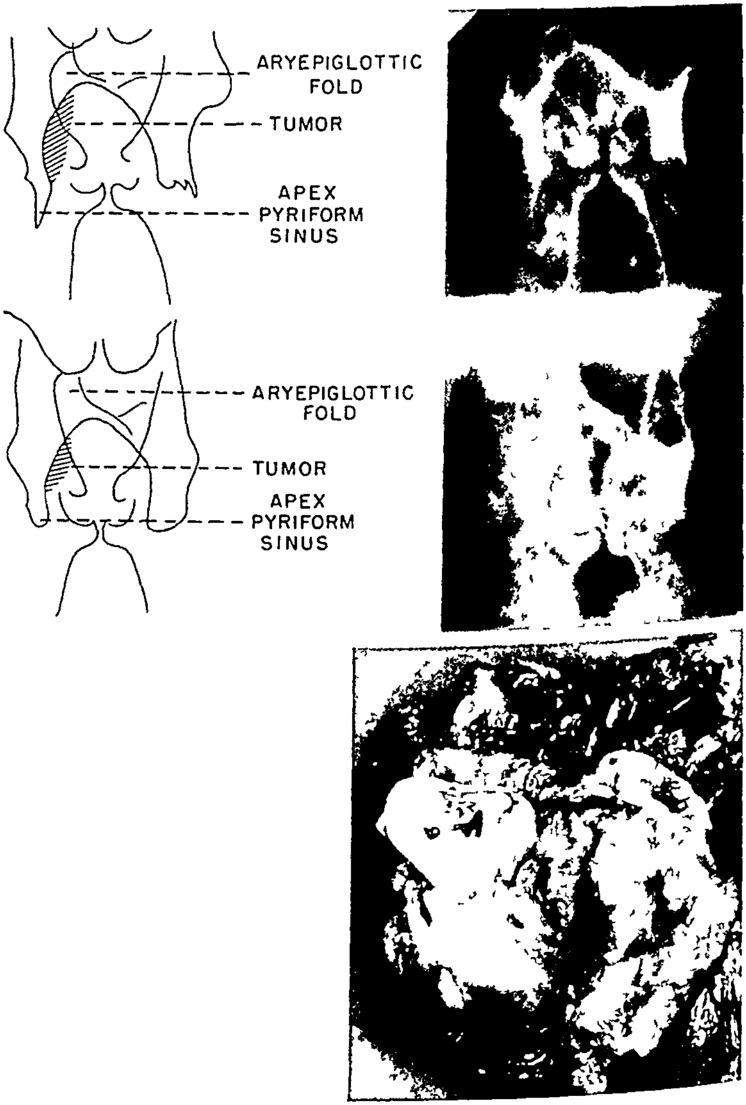

Fig 11 Pyriform Sinus Frontal view—There is tumor distortion of the inferior medial wall of the right pyriform sinus The apex of the pyriform sinus is free of tumor The laryngeal structures are normal except for marked edema of the right ary-epiglottic fold and arytenoid eminence

Specimen—A—arytenoid There is an adequate margin of excision on the specimen

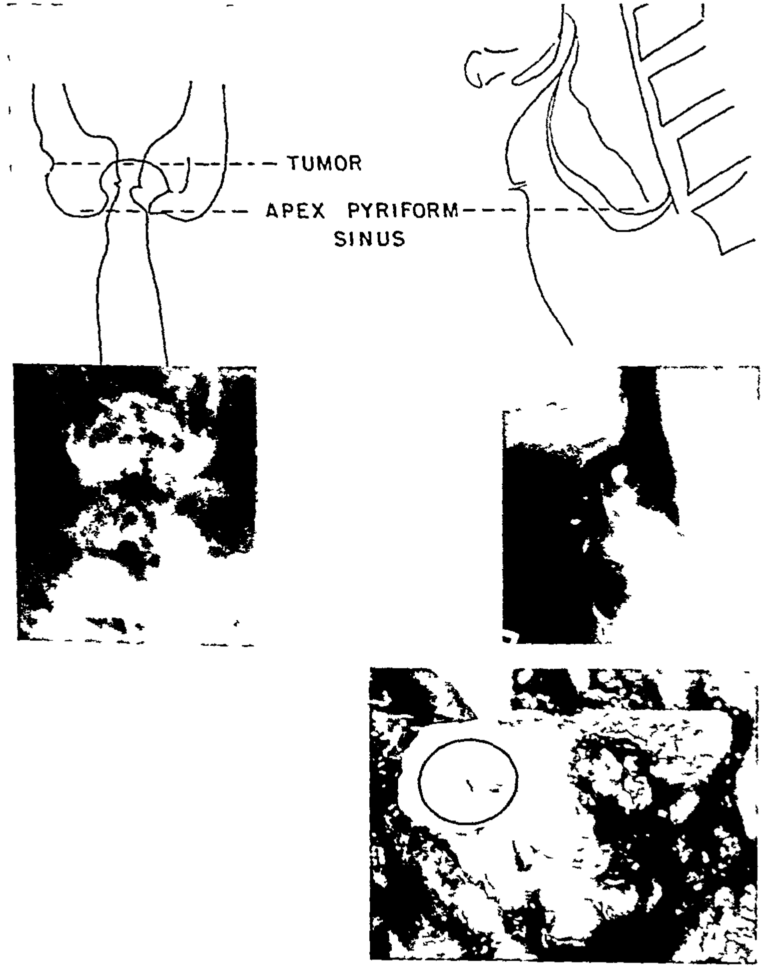

Fig 12 Pyriform Sinus Frontal view—A small (1 cm) irregularity is present in the lateral wall of the hypopharynx (modified Valsalva) The apex of the pyriform sinus is free of tumor

Specimen—Note the sharp elevated tumor margins

Glottic

From Table III, one can see that the size of the tumor seen on laryngogram varies as to location Since glottic lesions are confined to an area between the floor of ventricle to a point where the lower border of the intrinsic muscles end

(1 cm), no measurements were made Anterior-posterior extent of the tumor of the cord could not be accurately determined from lateral views

Infraglottic

There was an actual variation in size of tumors from 1.3-2.6 cms The margin of error is least in this group, with a ±0.3 cm on variation from the true size There was little difference in error whether the tumor was greater or less than 2 cms in the superior-inferior dimension

TABLE II

Classification Errors

	Clinical and Direct Laryngoscopy	Laryngograms	Total Pathology
Glottic	1 subglottic	1 false cord	13
Infraglottic	5 false cords 2 subglottic	4 {ventricle / false cord} 1 subglottic	14
Supraglottic	7 true cord	1 true cord	29
Transglottic	6 {3 base epiglottis 2 false cord—ventricle 3 subglottic}	0	16
Pyriform Sinus	1 missed tumor	1 A E fold	27
Total	22	8	99

TABLE III

Size of Primary Lesion

	Actual Size	Average Laryngogram Variation
Glottic	—	—
Infraglottic	1.3-2.6 cms	±0.3 cm
Supraglottic	1.0-5.0 cms	±0.6 cm
Transglottic	1.8-5.0 cms	±0.4 cm
Pyriform Sinus	1-6 cms	±0.6 cm

Supraglottic

The size of the tumor varied from 1-5 cms in greatest length in the superior-inferior extent, and the margin of variation was ± 0.6 cm

Transglottic

Tumors in this group vary from 1 6-5 0 cms on the specimen with a mean error variation of ±0 4 cm by laryngograms

Pyriform Sinus

These tumors varied in actual size from 1-6 cms with an error of ±0 6 cm by laryngograms

DISCUSSION

Limitations of Laryngogram Interpretation (See Table II)

The successful interpretation of laryngograms depends on the skill and speed of performance of the procedure and the experience and interest exhibited by the radiologist At the present time there are four areas where some difficulty has been experienced in the accurate interpretation

Anterior Commissure of True Cord When the anterior commissure area was deformed on laryngogram in glottic, infraglottic and transglottic groups, tumor was demonstrated pathologically in seven of eight instances On the other hand, when the laryngograms were negative, there was invasion microscopically in five of 13 instances This degree of error can only be explained by submucosal or relatively superficial invasion, insufficient to give gross distortion visible by laryngograms One can conclude that this area is not correctly assessed by laryngograms

True Cord Mobility There was a high correlation between clinical and radiologic findings in every instance when the cord was mobile or completely fixed When lag of the cord was evident by neuromuscular involvement or mass tumor weight, such a cord appeared mobile to the radiologist

False Cords The most frequent error was in interpretation of the false cords in the infraglottic group It is important to stress that more errors were made clinically as well on this particular structure Since biopsies were taken from these areas, postoperative edema will result The majority of these laryngograms were made after biopsy examination, and it is logical to assume that some of these errors in interpretation could have been avoided if laryngograms were done prior to biopsy

There are additional anatomical factors to explain some of these errors Examination of these specimens demonstrated that the false cord displacement superiorly by the cordal subglottic lesion obliterated the ventricle Review of these errors on laryngograms showed that actually this was the case, and it is likely that these errors of interpretation will be less in the future

Aryepiglottic Fold When edema or distortion of this fold was present it was difficult to rule out tumor invasion Tumor can be suspected only when definite scalloping and irregularity of radiographic outline is present

Postcricoid Laryngograms were never designed for the diagnosis of lesions in this area, and no interpretation can be given by the method of examination being discussed

Significance of Laryngogram Findings

Laryngograms have demonstrated their great value as an adjunct diagnostic procedure for laryngeal cancer When these findings are correlated with indirect and direct laryngoscopy and biopsy report, together with cervical node findings, the otolaryngologist can separate his cases into groups with high and low metastatic rate and arrive at a definite scheme for treatment

Accuracy of laryngogram classification was high, 92 per cent as contrasted to 78 per cent by conventional clinical appraisal There was an 8 per cent error in laryngogram classification, with the majority of the errors in the infraglottic group There was a much greater error rate by clinical means (22 per cent) With continued refinement in laryngogram technique and greater experience in interpretation, the false cord error rate should be less in the future Other errors of interpretation are extremely low and are to be expected

Certain clinical situations have always made accurate clinical classification difficult Bulky supraglottic tumors are an-way problems and the precise lower margin is always difficult to determine While lesions above the petiole of the epiglottis are easily determined and one can perform the supraglottic subtotal laryngectomy operation with confidence, the greatest problem confronting the surgeon who wishes to conserve function is with lesions of the false cord and base of the epiglottis In the latter group, laryngograms can very accurately discern freedom of involvement of the true cord Bulky tumors of the false cord that overhang the true cord can be precisely visualized by laryngograms It is in this situation that clinically one has most frequently mistaken tumor to involve the true cord

The accuracy of the radiologist in classifying a lesion as supraglottic was high In 17 instances where the supraglottic operation was performed, 12 were listed as wide surgical margins of safety and proven by the examined specimen' In five cases where tumor was in the false cord area and petiole, these were accurately visualized and again correlated well with the safety margin of the surgical specimen Of the 12 patients with laryngectomy and neck dissection performed, six cases could have had a supraglottic subtotal operation The latter operation was not performed because in the clinical preoperative appraisal, it was felt there was "insufficient margin of safety"

The purpose of bringing this point up is to emphasize that accuracy of diagnosis is necessary for the purpose of evaluating whether conservation of function operation can be performed rather than write the case off to the more easily executed total laryngectomy While this paper does not deal with end results of the supraglottic subtotal operation, these highly selected cases are quite successful for a one to six-year follow-up[14,1] Perhaps the high frequency of the "pushing" type of lesion in the supraglottic group accounts in part for success with this relatively narrow surgical margin of safety

Clinical estimation of extent of cancer involving the infraglottic and transglottic groups has been frequently in error The error in judgment in both groups is explained by fixation of the cord which masks the size of the tumor present

Size and extent of subglottic involvement have been difficult to appraise even with a mobile or somewhat paretic cord. Tumors of the transglottic group that have involved the false and fixed true cord frequently obscured marked infraglottic extension. In addition the base of the epiglottis has been sometimes overlooked. In all of these situations, the laryngograms have proved their value by precise localization and sharp delineation of the lower extent of the tumor.

Tumor crossing the midline was correctly determined by laryngogram with every lesion in the supraglottic, infraglottic and transglottic groups.

There was a high accuracy index for pyriform sinus lesions from the clinical and radiological standpoint. The majority of these cases required total laryngectomy and neck dissection, with modification such as skin grafting or other reconstructive procedures with larger tumors, however, there is a small number of selected cases where conservation of function procedures can be performed after a detailed clinical and radiological evaluation. This should help the surgeon decide what surgical procedures can be instituted.

No one can appreciate more than the surgeon the great importance of knowing where the lesion exactly is. He can plan his surgical procedure rather than complicate this with an ill-planned exploratory approach.

Size of tumor can be accurately determined for all classification sites with a mean variation from ±0.3-0.6 cm depending upon tumor site. It is impossible to estimate size by laryngoscopy, but it should not be surprising how frequently exact actual tumor size correlates with laryngograms. Size of tumor, degree of differentiation and location of the lesion have a direct bearing on the metastatic rate.

Since the transglottic group has the highest metastatic rate for apparent and inapparent metastases when the tumor is over 2 cms. in diameter, it follows that neck dissection with total laryngectomy is mandatory in this situation. If a palpable node is present on one side with a 2 cm. or larger transglottic or supraglottic lesion, consideration of bilateral neck dissection is warranted

It should be apparent that there are other therapeutic implications Patients with small glottic cancer and possibly minimal lesions of the epiglottis where the metastatic rate is low might better be handled by irradiation With improved methods of treatment by radiotherapists, conservation of function will apply to radiotherapy and the parameter of surgical treatment will be better defined Until the time when this study is again reviewed and specific conclusions drawn, the surgical management will continue to be selected on the basis of accurate clinical and radiologic diagnosis

SUMMARY AND CONCLUSIONS

1 Accurate clinical classification of cancer of the larynx is necessary since the choice of therapy is governed by the probability and location of metastases

2 Analysis of data obtained from a study on routine *en bloc* neck dissection with total or subtotal laryngectomy has given us very valuable information as to certain characteristics of the primary lesion

3 A pathologic classification based on location of the primary lesion has been used for laryngographic and clinical use The value of the proposed classification indicates that there is a progressive increase in metastatic rate from glottic, infraglottic and transglottic cancers

4 Size of tumor, degree of differentiation, and location is related to apparent and inapparent cervical metastases

5 Laryngograms can more accurately localize and objectively document the size of the cancer than clinical evaluation

6 Size of the primary tumor can be estimated on laryngograms to within ±0 3-0 6 cm of actual tumor size

7 The laryngogram is necessary for accurate diagnosis

8 Logical treatment will necessarily follow *a* Conservation of function by radiotherapy for early lesions of the cord, *b* larynx-sparing operations for selected cases of supraglottic or pyriform sinus lesions, *c* more radical operations (total laryngectomy to include neck dissection when the inapparent metastatic rate is high

9 The therapeutic conclusions drawn from this study are necessarily tentative The decision test by which all therapy is judged is a careful follow-up of patient survival

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DISTINGUISHED SERVICE AWARD GIVEN TO NEARLY DEAF EAR PHYSICIAN

Dr Kenneth M Day of Pittsburgh, Pa, is recognized by his medical colleagues as one of the most successful ear physicians in the country.

He attributes his success to the fact that he has been nearly deaf himself for many years, and has served as a model of what the hearing handicapped can really do

In honor of his service to medicine and mankind, Dr Day received a distinguished service award May 20, 1960, from the Pennsylvania and New Jersey Academies of Ophthalmology and Otolaryngology at a joint annual meeting in Atlantic City, N J, attended by eye ear, nose and throat physicians from six middle Atlantic states

THE APPLICATION OF IN VITRO CYTOTOXIC REACTIONS TO CLINICAL DIAGNOSIS OF FOOD ALLERGY*

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and
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The need for improved diagnosis of food allergy is widely recognized The paper by Black[3] in 1956, strongly suggested the use of viability of leucocytes as a sign of allergic reaction His observations dealt with the behavior of living leucocytes in the simultaneous presence of both allergen and plasma from a sensitized individual If specific antibodies to the allergen were present, the polymorphonuclear leucocytes showed toxic reactions with death of the cell occurring within 15 minutes to several hours If the reaction was prompt and strong, clinical sensitivity to the allergen was suspected Glaser[illegible] and co-workers attempted to evaluate the test in 1958 They reported 186 tests on 13 patients with 27 known sensitizations They questioned the validity of the test since it revealed reaction to only one allergen in 24 "tested," but 19 had poor controls, and no interpretation could be made The application of Black's cytotoxic test with minor variations to 107 patients with wide variety of possible or definite allergies to food, forms the basis of this report

The destruction of leucocytes by the antigen-antibody reaction has been observed for many years from several points of view As early as 1909 Achard and Bernard[1] studied specific reactions of leucocytes In 1947 Squier and Lee[13] reported a maximum fall of 43 per cent in leucocytes during *in vitro* reactions of untreated ragweed sensitive blood with ragweed extract Attempts to use this for quantitative measurements

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of the strength of house dust extracts were made by Berkowitz and Scherago[3] with only partial success Franklin and Lowell[9] in 1949, failed to find destruction of leucocytes from ragweed patients, using a total count method Tuberculin was shown to cause a similar fall in lymphocytes separated from plasma by Favour [8] A preliminary report from Finland (Pettay[11]) estimated leucocytolysis *in vitro* caused by different allergen extracts Extracts giving three plus skin reactions caused a 23 per cent drop in the total leucocyte counts, two plus reactions gave a 17 per cent drop and weak reactions a 13 per cent drop Negative extracts caused no decrease Waksman[15] reported specific white cell lysis produced by the combination of rabbit antiserum to a principal protein (ovalbumin, bovine gamma globulin) with homologous antigen Hartman and Hock[10] observed in careful animal experiments that the same cytological changes were observed in blood from both sensitive and non-sensitive animals but that the manifestations of leucocytic injury were greater in blood from sensitive animals, which indicated that injury to blood leucocytes can result from an antigen-antibody reaction A scholarly review of available literature on the toxic effect of the antigen-antibody reactions on cells of hypersensitive reactors is given by Waksman[16] in "Cellular and Humoral Aspects of the Hypersensitive States" It is thus evident from the literature that the antigen-antibody reaction does give a leucocytotoxic effect Vaughan[14] used the post-prandial fall in the leucocyte count as a sign of food allergy, and Rinkel[12] incorporated this into his method for testing foods (Chap VII)

The technique for observing the leucocytic toxic reaction, as described by Black, is simple in plan but rather difficult to carry out, fortunately, however, with experience it becomes easier and errors in technique less frequent

The first problem is to obtain an abundance of viable leucocytes For a year our sporadic efforts were not consistently successful Now we take from 5 to 10 cc of blood, add 0 07 cc of transfusion citrate per cc of blood, and centrifuge in small glass tubing for at least one hour at medium speed The tubing is pulled slightly in the middle to narrow the center part of the tubes so that the buffy coat will be elongated and thereby easier to remove (see Fig 1) With this technique the resulting buffy coat is usually adequate The use of capillary tubing requires a meticulous technique which proved not only to entail more work but also to be more apt to induce error It is now reserved for cases in which veni-puncture is not practical

After the buffy coat is obtained about two-thirds of the

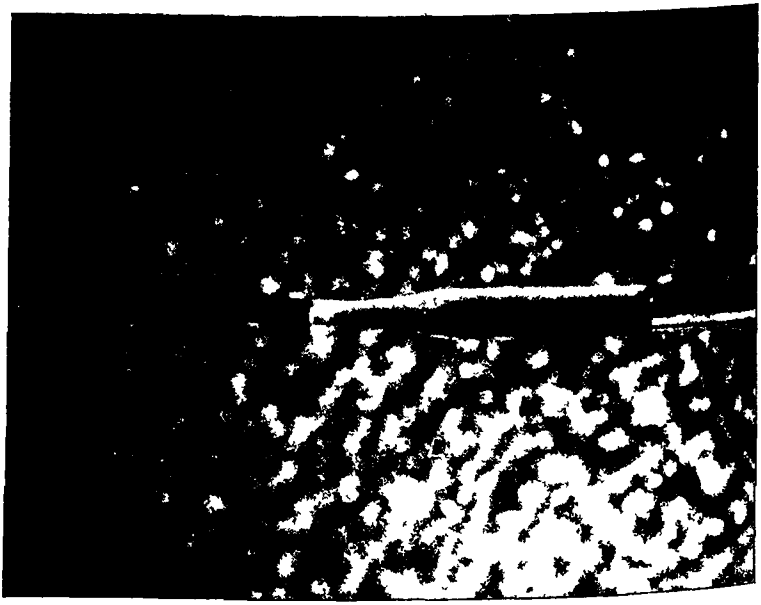

Fig 1 Glass tubing with narrow middle portion in which citrated blood has been centrifuged to obtain buffy coat and serum

serum is removed by an appropriate capillary pipette, then the buffy coat is withdrawn and mixed with the serum so that there will be between 15 and 40 leucocytes per high power field

Special care is necessary in mixing the leucocytes and serum This is accomplished by drawing the mixture back and forth in the tube, stirring and rotating it five or more times A slide with concave depressions such as is used for blood agglutination work provides satisfactory mixing chambers

Since the cells settle rapidly, the mixing must be done just before the cells are put on the slides to insure even distribution in all the samplings In order to avoid drying, the mixture can be saved for long intervals in the tube, but before putting out further tests it must be thoroughly mixed

The antigens used were dry extracts of foods from Hollister-Stier Solutions of 0 1 per cent were made with pyrogen-free

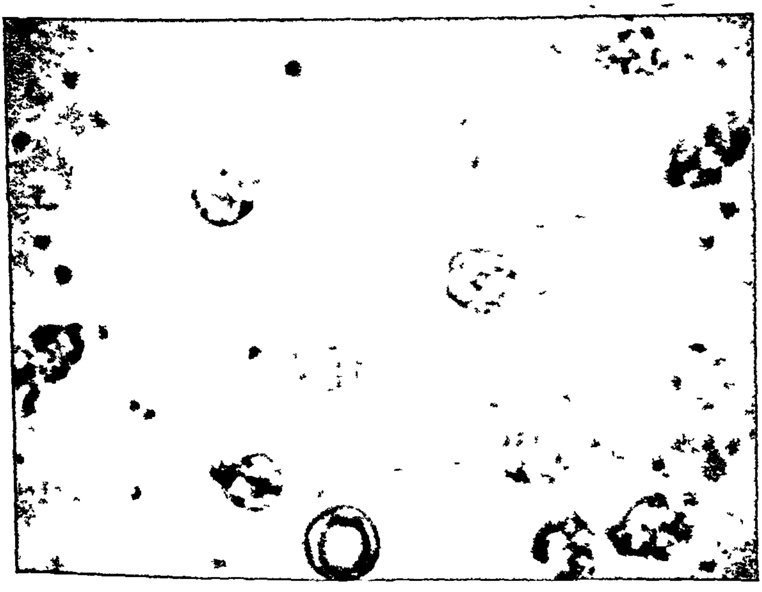

Fig 2 Normal leucocytes with pseudopods

distilled water and allowed to stand for 24 hours The solutions were dropped on numbered slides (three to a slide) so that the resulting area of dried food would be slightly larger than the cover slip This preparation of the food deviated slightly from Black's method which was to take a barely visible amount of dried protein allergen picked up with the small end of a fresh flat toothpick and to add the powder to a drop of distilled water on the slide It seemed advisable to use known dilutions of the antigen to control the quantities and obtain more nearly uniform concentrations of food With this method large numbers of uniform slides easily can be prepared in advance

It is well to prepare all the glass used in the test with "Silicad," which is a silicone product With its use the controls recently have remained good for over an hour, and one was observed for 11 hours This is practically a prerequisite for performing the test

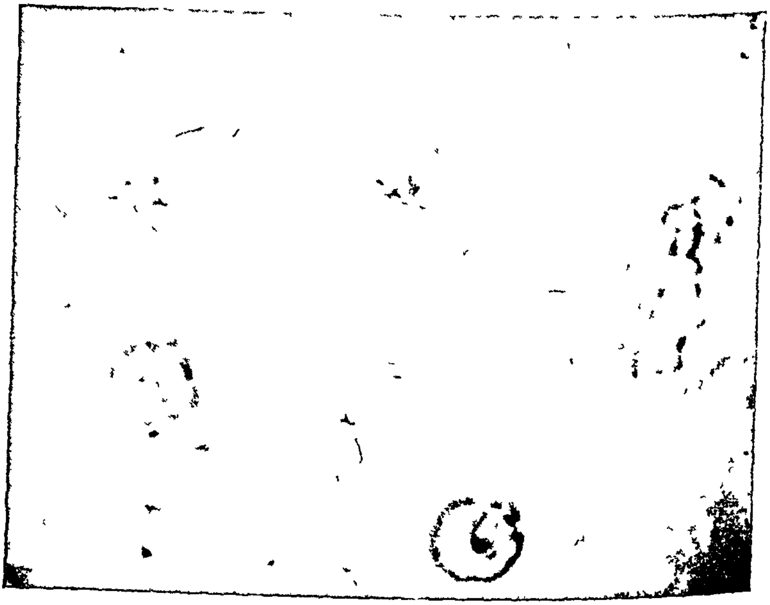

Fig 3 Normal active leucocytes showing shape variations Note platelets

The leucocytes are easily visible under a good high-dry objective We find the 60x apochromatic objective satisfactory, making the use of oil-immersion unnecessary The neutrophils are irregular in shape due to their numerous pseudopods (see Figs 2 and 3) With close observation they show streaming of granules and change of shape with amoeboid-like motion Eosinophiles are easily identified by the large refractile granules They are much less motile than the neutrophils The lymphocytes show very little visible change in cell contour or movement

Cell counts, as Black recommended, have not been done, but usually about 12 different fields of cells are studied When the distribution of cells is good, each field contains between 15 and 40 leucocytes After a little experience the healthy, viable condition of the leucocytes is very clear The deviations from the normal, however, require careful observation and experience with judging cells in the fresh unstained condition The slides are observed within ten minutes after they are made A second examination is made after 30 or 40 minutes Sometimes, when the second examination was questionable, a third has been made at the end of an hour

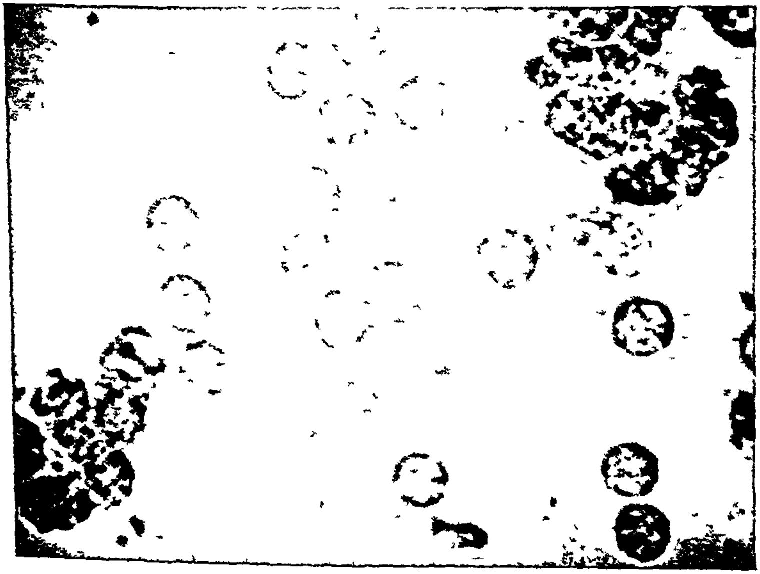

Fig 4 Round leucocytes indicating reaction to food allergen

The reactions of the neutrophils, as we have seen them, present the following changes

1 Loss of amoeboid movement and cessation of streaming of cytoplasmic granules

2 Rounding of the cell contour with or without small spine-like projections (see Fig 4) method large numbers of uniform slides easily can be prepared in advance

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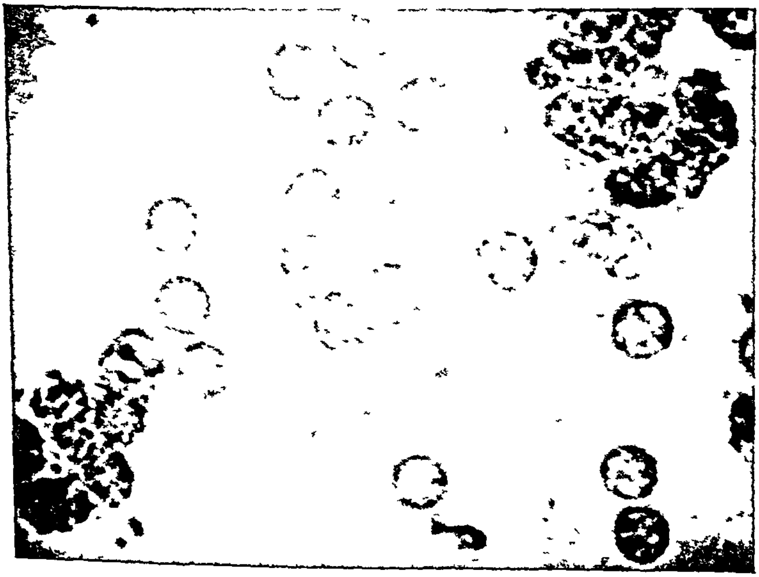

Fig 4 Round leucocytes indicating reaction to food allergen

The reactions of the neutrophils, as we have seen them, present the following changes

1 Loss of amoeboid movement and cessation of streaming of cytoplasmic granules

2 Rounding of the cell contour with or without small spine-like projections (see Fig 4)

3 Vacuolization and spreading of the cytoplasm The cell may become thin and flat (see Fig 5)

4 Fragmentation of the cell with rupture of the membrane and dispersion of the granules (see Fig 6)

Frequently, in the immediate marked reactions, not mentioned by Black, the cells begin to disintegrate before they can be observed Along with the disintegration of the neutrophils there is also a rapid clumping, agglutination and disappearance of the platelets The red blood cells agglutinate and appear to be mushy, with loss of distinct limiting cellular membranes In slightly less severe reactions, the red cells are crenated (see Fig 7) The eosinophiles may remain intact after the other cells are gone, thus they seem to be more resistant to the toxic effects than are the neutrophils The lymphocytes are also somewhat resistant, similar to the eosinophiles, however, in severe reactions they, too, are difficult to find, so it is suggested that they also share to some extent in

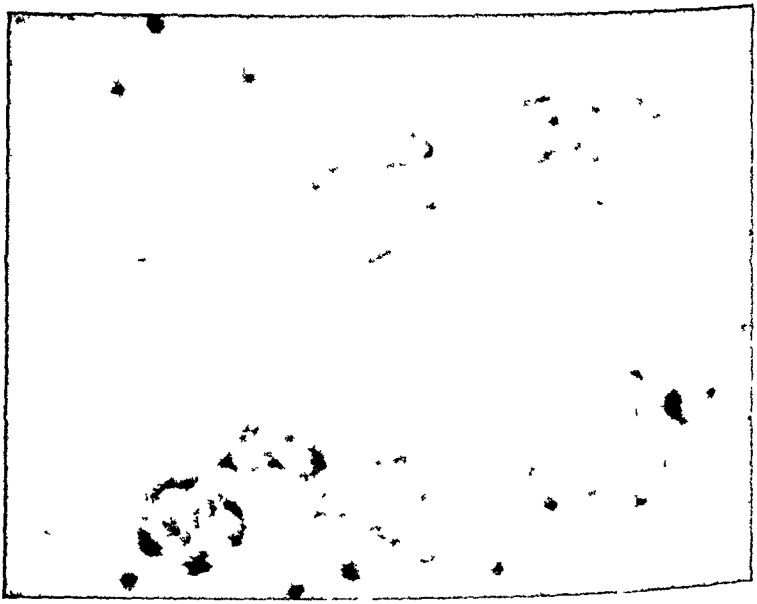

Fig 5 Beginning disintegration of leucocytes—reaction to food allergen was marked

the cytotoxic reaction (Favour[2] noted that the lymphocytes were the first affected in the reaction of tuberculin on cells without serum)

In our experience it is not necessary to incubate the cells at 37° C to observe reactions, however, the room in which we work was warm (80 to 85° F) For the most part the tests were not observed after 45 minutes to one hour

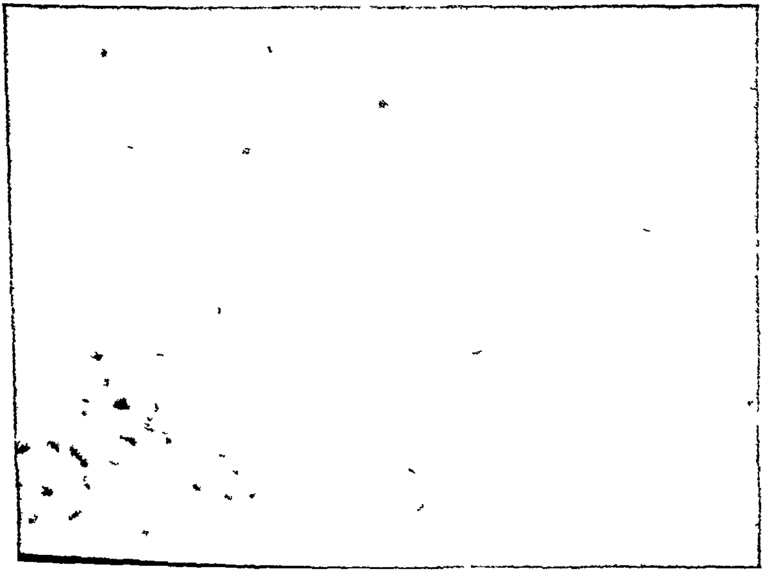

Fig 6 Complete disintegration of several leucocytes in the presence of offending allergen

The reactions of these tests have been evaluated in terms of marked, moderate, slight and negative A total of 3,828 reactions were studied, which were divided according to severity as shown in Table I and as follows

Marked—466—majority of leucocytes disintegrated, platelets diminished, and red cell reactions, of which 95 had occurred by the first observation (called immediate marked, M-1) and 371 by the second (M-2)

Moderate—522—all leucocytes round with only a few disintegrating

Slight—533—approximately half of the leucocytes round and half active in ten to 15 fields examined

Negative—2,307—viable normal appearing neutrophils and platelets in all fields examined A comparison of these with the positive reactions often formed the basis of the diagnosis of negative, because the controls, until recently, showed more rapid deterioration of the cells than did those with compatible food A remarkable improvement of the controls followed the use of Silicad on slides and cover slips

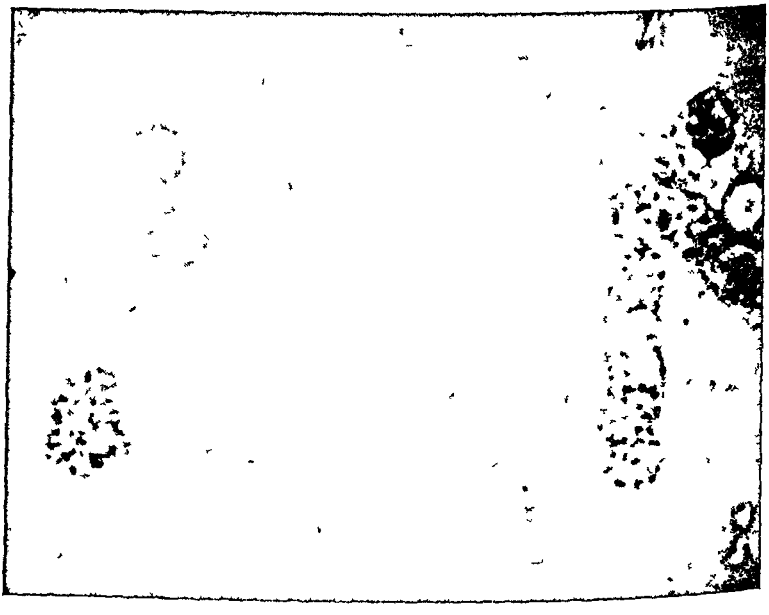

Fig 7 Round leucocytes and crenated red blood cells

A few in which definite judgment was difficult were classified as questionable

Two-hundred-thirty-two individual food tests were repeated When compared, we found 124 correlated exactly, 66 varied only one degree (as from slight to moderate or

TABLE I

Reactions to Allergens

Allergen	M-1	M-2	Mod	Sl	Neg	Total	Allergen	M-1	M-2	Mod	Sl	Neg	Total
Corn	3	16	31	22	62	134	Rice	–	5	8	4	37	54
Wheat	3	18	29	19	65	135	Brazil Nut	1	5	7	4	32	49
Egg	3	15	29	24	72	143	Cashew	1	–	11	2	38	52
Milk	3	17	21	27	63	131	Coconut	–	2	2	8	39	51
Beef	2	11	21	18	61	113	Peanut	1	3	4	10	34	52
Pork	–	8	13	23	78	122	Pecan	–	–	2	4	35	41
Lettuce	5	11	22	27	47	112	Black Walnut	–	3	3	4	28	38
Potato	3	7	20	18	62	110	Cherry	–	3	2	2	28	35
Tomato	–	7	14	19	57	97	Grapefruit	–	3	4	1	26	34
Orange	–	4	11	17	58	90	Apple	2	2	2	1	30	37
Peach	1	2	10	23	49	85	Banana	2	9	2	4	16	33
Strawberry	1	4	5	19	56	85	Pear	–	–	1	1	30	32
Cocoa	4	7	12	12	51	86	Asparagus	–	–	1	2	26	29
Malt	11	31	20	7	19	88	Beet	–	1	–	1	29	31
Yeast	10	18	15	9	34	87	Cabbage	–	–	–	2	24	26
Soybean	1	3	10	19	55	88	Cauliflower	–	–	–	6	22	28
Coffee	1	6	11	12	41	71	Sweet Potato	3	6	6	4	12	31
Garlic	4	9	13	8	36	70	Turnip	–	6	3	3	14	26
Onion	3	22	13	11	21	70	Black eyed Peas	–	–	–	2	18	20
Pepper	2	1	13	7	48	71	Broccoli	–	–	–	2	18	20
Chicken	1	8	16	10	36	71	Carrot	–	–	1	1	19	21
Tuna	1	7	11	10	47	76	Cucumber	–	–	1	1	15	17
Celery	1	8	4	6	14	33	Mushroom	1	–	4	2	11	18
Lima Bean	–	–	–	1	29	30	Squash	–	–	–	1	10	11
Pea	1	8	1	2	19	31	Olive	–	1	2	2	4	9
String Bean	1	6	3	2	21	33	Tea	–	1	2	3	7	13
Pineapple	1	–	3	3	28	35	Apricot	–	1	–	2	4	7
Cantaloupe	–	6	6	6	21	38	Blackberry	–	3	1	1	3	8
Honey Dew	–	1	5	3	30	39	Blueberry	–	1	–	–	6	7
Turkey	–	3	7	10	52	72	Date	–	–	2	–	5	7
Shrimp	2	3	9	5	50	69	Fig	–	–	–	1	5	6
Lamb	1	12	1	6	31	57	Grape	–	–	1	1	4	6
Goat Milk	3	9	5	3	37	57	Lemon	–	–	1	–	4	5
Oat	1	8	7	6	36	58	Plum	–	2	–	–	3	5

normal, or marked to moderate), 34, two points of discrepancy, seven had three points, one, four (see Table II)

These errors are partly unexplained and some may be due to faults in technique Extreme cleanliness of all glassware is essential, and as mentioned above, the use of Silicad on both slides and cover slips should decrease errors in the future Variability in drying due to different thickness of the size of drops of the cell suspension may be avoided by sealing the cover slip with vaseline Any break in meticulous cleanliness may introduce a noxious substance Pyrogens (bacterial products that cause fever) may be found in ordinary distilled water Experiments are being made to determine the optimal quantities of the antigens

TABLE II

Total Tests Repeated on Same Day—232

Number Agreed	124—53%
Number Disagreed—	
Slightly	66—28%
Definitely	42—18%

The selection of patients for the cytotoxic tests is often facilitated by the cytologic findings In previous papers the authors[6] showed a correlation between increased numbers of mast cells and allergy to food This finding frequently gives a clue to the existence of food allergy Of the 47 patients of this series who had nasal cytology, 30 had mast cells Fifteen of those with mast cells were in the group having chronic rhinitis Thirteen of these correlated with their cytotoxic food tests One had only a slight improvement, and one did not correlate It seems that nasal cytology should be done when possible

Patients were also selected in whom the diagnosis was obscure and symptoms were of long duration, based upon the hypothesis that allergy might be the undiscovered etiology of otherwise undiagnosed diseases The various symptoms or conditions studied are given in Table III These number 133 and were gathered from the 107 patients tested It is seen that the symptoms of known allergic etiology improved with allergic measures, as did some others of questionable allergic etiology In the group of Ménière's disease, four of the six patients were shown to have allergy to food Two of them appear to be "cured"

The results of a test are told to the patient verbally and in writing The patient is advised to discontinue all foods that gave marked or moderate reaction, but to reintroduce the foods, one at a time, beginning with lunch on the fifth day This has rarely been carried out completely, for many patients have either felt so much better from the diet that they were not interested in eating the food again, or lost interest due to the severity of the advised change of diet The patients are advised to watch for the "slight reactors" and to observe other possible reactions

TABLE III

Symptoms and Syndromes Studied

	Totals	Improved	Sl	Unimp	Unknown
Chronic Rhinitis Sinusitis	36	20	7	7	2
Headache	21	15	2	3	1
G-I	13	10	1		2
Asthma and Cough	13	2	4	2	5
Infections acute	7	5	2		
Angioneurotic Edema Urticaria, Dermatitis	7	6	1		
Conjunctivitis and Palpebral Edema	7	6	1		
Ménière's	6	2	2	2	
Migraine	5	3	1	1	
Polyps	5	2	2		1
Myalgia	4	2	1	1	
Fatigue	4	2		1	1
Serous Otitis	2	2			
Hoarseness	1	1			
Iritis	1	1			
Arthritis (gout)	1	1			
	133	80	24	17	12

DISCUSSION

The Black test has proved useful in many cases There are apparently false positive reactions and some errors There normal, or marked to moderate), 34, two points of discrepancy, seven had three points, one, four (see Table II)

These errors are partly unexplained and some may be due to faults in technique Extreme cleanliness of all glassware is essential, and as mentioned above, the use of Silicad on both slides and cover slips should decrease errors in the future Variability in drying due to different thickness of the size of drops of the cell suspension may be avoided by sealing the cover slip with vaseline Any break in meticulous cleanliness may introduce a noxious substance Pyrogens (bacterial products that cause fever) may be found in ordinary distilled water Experiments are being made to determine the optimal quantities of the antigens

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SUMMARY

The cytotoxic food allergy test, originally described by Black has been used in 107 patients whose ages varied from nine to 80 years They had a wide variety of possible or definite allergies to food The technique used varied only slightly from the method first reported This test, while time consuming and requiring a cytologist for accurate interpretations of the cellular variations, has proved reliable to a degree that it may be employed in selected cases as a diagnostic method in which food allergy is a possibility The presence of mast cells in nasal secretions is often a useful indicator of food allergy with nasal pathology and may suggest the advisability of doing the leucocytoxic test

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are some cases in which the symptoms have not been due to the food allergy, whereas others with nasal symptoms relieved were surprised that other seemingly unrelated symptoms were also improved

Ménière's disease has been difficult to cure Occasionally it has been known to respond to non-specific allergic medication, and a few cases have been reported to be due to food allergy By means of much more efficient food testing possible with this test, it may be possible to diagnose the food factors in many more cases

Another unique value is the diagnosis of allergy to many foods both in severe and in very mild cases In both of these groups the introduction or omission of a single food may cause no change in symptoms, since the severe cases have maximal severity from the other foods, and the mild cases may notice symptoms only when several allergens are present

The test is perfectly safe for the patient since no antigen is administered

The actual accuracy of the test is yet to be determined The key of the test, the leucocyte, is a very delicate structure influenced by minor changes in environment

If the tests are not given a preliminary observation before incubation certain irregularities sometimes occurring in distribution of the cells may be mistaken later for the disappearance of cells, and the immediate marked reactions would be missed

The degree of reaction apparently can be influenced by the amount of antigen used in the test It may also be influenced if the test is made during a food reaction Blood taken during the time of a reaction has been found to be non-reactive to the allergen, whereas blood taken the day before or the day after (one observation) gave strong reactions

The more efficient diagnosis of food allergy offered by cytotoxic tests makes it possible to determine allergy to food in cases that otherwise could not or would not submit to long dietary experimentation

PSEUDO NEURAL HYPACUSIS IN CHILDREN[*]

SEYMOUR J BROCKMAN, M D ,[†]
and
GLORIA H HOVERSTEN M A ,[‡]
(By Invitation),
Los Angeles, Calif

Since World War II there has been an increased interest and awareness in factors of and problems related to hearing difficulties Initially, this interest was directed toward problems of diagnosis, treatment and rehabilitation of organic hearing losses

As facilities, techniques and consultants became more adept and facile in the various otologic centers established by the armed forces, more knowledge was acquired of organic difficulties and also of functional components as factors in hearing loss Several studies (Johnson, Work and McCoy,[1] Truex[2]) have shown that as many as 40 per cent of the members of the armed forces evaluated for hearing difficulties had a part or most of their hearing disability secondary to functional rather than organic causes

The armed forces' centers and, after the war, the veterans' centers, using a team approach of the otologist, psychiatrist or psychologist, and the audiologist were able to establish more definite criteria for such evaluations An excellent article on just such problems was presented by Truex[2] with regard to findings at the Deshon Center during and after the war

With the end of the war, much of this work was published

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[*] Read at the meeting of the Western Section American Laryngological Rhinological and Otological Society Inc Denver Colo Jan 22 1960

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[‡] Audiologist in practice with Drs Victor Goodhill Seymour J Brockman and Irwin Harris Los Angeles

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and was used as a guide in the diagnosis of such problems as they were presented to the private physician

The problems of functional hearing loss encountered in the armed forces as compared to those encountered in private practice are different in some respects In the armed forces' and veterans' centers, a good number of the cases were true psychogenic hearing losses due to battle trauma A smaller portion was judged to be malingering, either for benefit of non-return to duty, pension or transfer In private practice, more of these losses in adults are due to medico-legal problems following accidents and are placed in the category of malingering rather than true psychogenic hearing loss The physician in private practice is aware of this, and if he does not have the facilities for testing will usually refer the patient for more complete diagnostic evaluation Practically all of this work has been done on adults

The otologist with some pediatric-otologic practice, having become cognizant of functional problems as encountered in adults, has encountered similar problems in children

The child's problem is different from that faced in adults Whereas adults with functional loss are attempting in many instances to shirk a duty or exact a monetary gain, no such problems exist in children The parents are not usually aware of the hearing problem until the school brings it to their attention, in fact, it may be due to the expansion of the excellent testing programs in schools that these problems, which we are sure previously existed, are now coming to our attention

In our practice, we have had occasion to see a number of these children and have perused the literature for comments considering the frequency of this problem There has been a dearth of material on the subject The most recent complete report has been published by Dixon and Newby[3] Juers[4] and Myklebust[5] have been the other main contributors

DESCRIPTION OF SUBJECTS

We have selected nine of our more recent cases for discussion These children ranged in age from eight to 15 years, six were female, three were male In all nine of the cases, referral to our office was brought about by a school hearing test showing the hearing to be impaired Prior to the school test, there had been no awareness or concern on the part of the parents or child as to the presence of a loss

In none of the cases were behavior problems, academic difficulties or speech problems relayed as a complaint by the parent on initial examination None of the children was observed to have other than excellent conversational ability All had normal speech

The following criteria were used in the initial determination of the possibility of a functional component or completely functional hearing problem necessitating audiologic study

1 Discrepancy between the voluntarily obtained speech reception threshold and the average loss for pure-tones in the speech range (500-2000 cps)

2 Inconsistency in conversational ability outside of the test situation as compared with the pure-tone audiometry test results

3 Pure-tone threshold responses inconsistent and variable

TESTING PROCEDURES

In our practice, pure-tone audiometry generally precedes speech audiometry except in the case of children five years or under With the children under discussion, it has not been possible to establish any single uniform set of procedures, the audiologist preferring to introduce various tests dependent upon the child's previous responses, however, we find that a few generalizations as to test procedure have emerged as a result of these experiences

1 Speech Audiometry

Since it has been our observation that the speech reception threshold usually is much closer to the true hearing level than the pure-tone audiogram, the audiologist begins with speech audiometry, simply continuing informally in the sound-field through the speaker, the conversation which was originally started in the same room with the patient The child is given the impression that testing has not begun

For example, he may be told that the audiologist is simply getting ready for the test and needs to know the answers to such questions as, "How do you spell your last name?" and "How old are you?" To add to the child's impression that testing has not begun, the light in the control room which enables the audiologist to be seen can be left on (even though the patient is not given the opportunity to lip-read)

The level at which this conversation and questioning is performed is dependent upon the observed ability to follow conversational speech prior to testing If this appears normal, as it has in all our cases, conversation is then presented at soft levels of 15 to 25 db Care should be taken not to begin with intensities much greater, as the child will use this as his base-reference, and it will be much more difficult to secure responses at the soft levels which may have been achieved much more easily initially

Continuing at the same intensity level of 15 to 25 db, the child is simply told, "Now I want you to repeat some words" Spondaic words may then be presented and the speech reception threshold obtained After testing several of these children, the technique evolved of omitting the speech reception threshold and instead, going directly to the speech discrimination tests A PB word-list is given at the same level at which the audiologist and child carried on conversation thus leading the child to respond unsuspectingly at this level Note that no intensity decrease has taken place and, therefore, the impression of "testing" is minimized

Six children gave normal discrimination scores (90 to 100 per cent) at levels of no greater than 30 db It was possible to assume from this that the actual speech reception thresholds for these children were considerably better than their admitted speech reception thresholds measured following the speech discrimination tests

Three of the children apparently made deliberate errors, their discrimination scores showing wide variability For example, one child scored 32 per cent at 40 db and on another word-list given two days later scored 72 per cent at the softer level of 25 db

In all of our cases, informal conversation was carried out without difficulty at a better level than the admitted measured speech reception threshold In the nine children, initial speech reception thresholds were obtained of 10 to 25 db

At this point, the audiologist has objective evidence that the child's hearing is certainly near-normal and that he is probably under no strain in ordinary listening situations Speech audiometry, using a similar presentation, can then be continued with earphones

2 *Pure-Tone Audiometry*

Pure-tone audiometry is attempted in the standard manner Presentation of tones and method of determination of thresholds will vary from child to child As a general rule, we think it advisable again to *expect* the response at softer intensity levels so that the child is not given a loud tone as a base-reference Only when all efforts are exhausted at getting a pure-tone response at these levels (approximately 30 db or below) should the audiologist present tones at louder intensity levels We think much of the error on school tests may have been simply in "cranking" up the sound to high levels until the child responded

The pure-tone testing is introduced with some such casual remark as, "You will have no trouble hearing these tones, they are easy to hear" A tone is then presented at 25 to 30 db and, if the child does not respond, the audiologist's attitude is one of surprise The audiologist may suggest to the child that he hears the tones and that he simply did not understand the directions Often a child can be coaxed into a response in this manner Sometimes the ascending threshold, starting with minus 10 db and interspersing coaxing, can result in a nearer true threshold response If such coaxing fails at these levels, it becomes necessary to present tones of higher intensity

Factors which should be noted during this pure-tone testing are

1 Variability of "threshold" responses for a single tone These frequently cover as wide a range as 30 to 35 db and can be influenced by the method of presentation, varying the frequency and/or loudness-contrast of tones

2 Comparison of the ascending threshold and the descending threshold as proposed by Dean A Harris[6] Harris states that, in the adult malingerer, a difference of 10 db or greater between the ascending and descending thresholds is highly indicative of non-organic involvement

3 Reaction to surprise directions interspersed throughout the pure-tone test For example, one child was moving the earphone cords while making no response to pure-tones at 55 to 60 db A sharp rebuke, "Don't do that!" at 25 db caused the child to drop the earphone cords immediately Several children raised their hands to the earphones to remove them when told, "We're finished You can take the earphones off" at levels at which they had not responded to spondees or in formal conversation

It is helpful for the younger child, eight to nine years, whose loss seems to be on more of an unconscious basis, an "inability to listen," rather than a willful malingering, to be given the opportunity to respond to play-audiometry Frequently, this simple concretizing of the abstract listening activity (by dropping a marble in a box or putting a peg into a hole when the tone is heard) enables this child to center his attention and thus respond at softer levels We have one child eight years of age, whose threshold moved from 70 db to 5 db by changing to this simple method of testing usually reserved for children six years or under (see Fig 1)

These children may do even better if they are trained and encouraged to respond to softer levels We have a glass between the test and control room so that the child can see the audiologist Initially, this visual situation is maintained, the audiologist nodding approval when the child questions or hesitates in response to a tone and sometimes indicating by a gesture of "shh" or perhaps a finger gesture that the next tone will be very soft When the child is able to go ahead on his own, then the light is dimmed so that the audiologist can't be seen, and the test continues

In the child where these pure-tone and speech techniques do not result in formal establishment of normal hearing thresholds, it is sometimes necessary to resort to special tests such as the Stenger test for pure-tone, the modified Stenger for speech, the Doerfler-Stewart test and psychogalvanic skin resistance audiometry These have proved most helpful

PURE-TONE AVERAGE (500 2000 cps)

	C.A Age 10	
June 18 1959—		
No 1	Air conduction—RE	40 db
No 2	Air conduction—RE	30 db
No 3	Air conduction—LE	20 db
No 4	Air conduction—RE	10 db
No 5	Bone conduction - LE	10 db
July 2 1959—		
No 1	Air conduction—RE	15 db
No 2	Air conduction—RE	0 db
No 3	Air conduction—LE	–10 db
No 4	Air conduction—RE	–10 db

Fig 1 Air conduction thresholds for the same ear of one child obtained on the same day by the same examiner using two methods

DISCUSSION AND CASE REPORTS

On initial otologic examination two of the nine children had a transient otitis media which subsided rapidly on medical management In no instance was the otitis media recurrent nor did it interfere with any of the subsequent test findings We eventually were able to achieve both pure-tone and speech reception threshold results on all of the children indicating hearing within the normal range (15 db or less) Most commonly, the audiograms were of the perceptive type, bone-conduction and air-conduction being similar If test results revealed an air-conduction loss greater than 60 db, the bone-conduction in these instances was superior Five of the nine children (four females, 10 years of age or under, and one male, 14 years of age) were judged as having an inability to listen similar to those described by Myklebust Myklebust[5] discusses the fact that some children with otherwise normal hearing show poor conscious response to pure-tone stimulation This he explains on the basis that the pure-tone is too abstract and meaningless to arouse cortical evidence and recognition on the part of the child He further states that in the process of auditory maturation the child first shows evidence of response to speech or complex meaningful sounds or noise and that visible evidence of response to pure-tones is in some instances delayed We concur with these statements Some children respond to pure-tones at a much earlier age than others This does not mean that the organ of Corti or the central pathways are not stimulated by a pure-tone, but merely that recognizable conscious evidence of response is delayed in development

It is an accepted fact that some individuals will regress to infantile patterns psychologically in certain areas of their behavior when subjected to induced stress Since the function of hearing to some extent represents a psychological reaction, it also is possible that some individuals are so sensitized or conditioned as to regress in the auditory area under certain situations of psychological stress A greater loss in pure-tones in these cases would more logically be a regression than a suppression

These five children appeared to be in need of training and encouragement in order to learn to respond to faint tones and speech They did not reveal evidence of willful malingering but rather tended to be entirely cooperative and straightforward in the test situation Further indication of the inability to listen and the advisability of training these responses to faint tones is illustrated by close examination of one child's audiograms (see Fig 2)

With the exception of the 14-year-old male, four of these children apparently had made entirely satisfactory home and school adjustments, the parents having *no* complaints or anxieties There were no gross emotional disturbances or speech problems apparent on visits to our office

The remaining four of the nine children were strikingly different in test behavior and histories from the five children just discussed All four of these remaining children appeared to be willfully malingering, and on consultation, were found to be the source of considerable anxiety to their parents

One of the four, an 11 year-old girl, had been in many foster homes with considerable psychological trauma in early life Hypochondriacal behavior was reported by the mother The mother was entirely receptive to psychiatric referral When the child returned nine months later for testing a normal audiogram was obtained (see Fig 3)

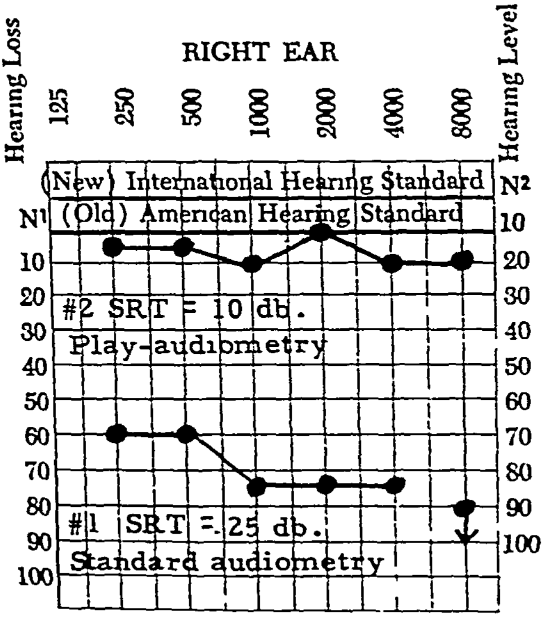

Fig 2 Results of tests on one child showing consistent increase in listening ability The numbers refer to order of test

A boy, age 14 was failing in school and a poor reader The mother reported an I Q test had been given and the child found to have normal intelligence Early psychological trauma was present (this boy's father and older brother dying one month apart when the patient himself was three and one-half years of age) The boy has had six months of psychiatric treatment (see Fig 4)

A girl 14 years of age, had been given a hearing test at school was permitted to see the audiometer and was able to observe that her hearing was poor in the left ear This proved so upsetting to her that she stayed home from school unknown to her parents for ten weeks dressing in the morning and calling in for assignments No response to pure-tone or speech at maximum levels in the left ear was secured on her first audiometric examination in our office Spondees were repeated at 5 db in the right ear, but there were no pure tone responses in that ear until levels of 60 to 90 db

Return visits revealed wide fluctuations in speech reception thresholds as well as pure tone thresholds For the right ear, the ascending speech reception threshold was 50 db, the descending threshold 20 db with later correct spondee repetition at 2 db Pure tone threshold responses for the right ear, depending upon whether ascending or descending presentation was used, varied over a 20 to 35 db range A few left ear responses were secured at 80 to 100 db with no response to spondees, but surprise directions were followed at levels as low as 20 db in that ear

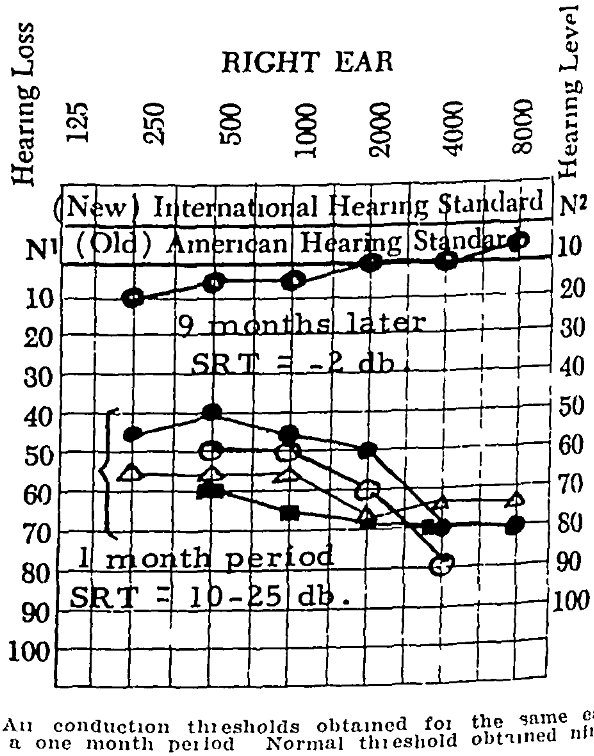

Fig 3 Air conduction thresholds obtained for the same ear of one child over a one month period Normal threshold obtained nine months later

The Stenger tests and PGSR measurement were made, both tests revealing near normal hearing in the left ear Following the PGSR, the child evidently realized that the test had been beyond her control and that it had revealed better hearing than she previously had admitted She then gave voluntary responses to pure tones in the left ear at 10 to 25 db Final discrimination tests were normal (96 98 per cent) in both ears when speech was presented at 25 db (see Fig 5)

On conclusion of the voluntary tests revealing normal hearing the audiologist commented, "That was much better How do you explain that?" The child answered, "I guess my nose was stuffed up, and it cleared "

Following the school test, the child had been transferred to an integrated program for hard of hearing and deaf children She had a teacher accompany her to the classes with normal hearing children, the teacher writing down directions and explanations for her The teachers felt that her hearing fluctuated

The child was seen in a psychiatric clinic for evaluation and was found to be functioning at a bright level intellectually Noted were the following

1 Poor social adjustment with peers

2 Basic insecurity, tense and anxious

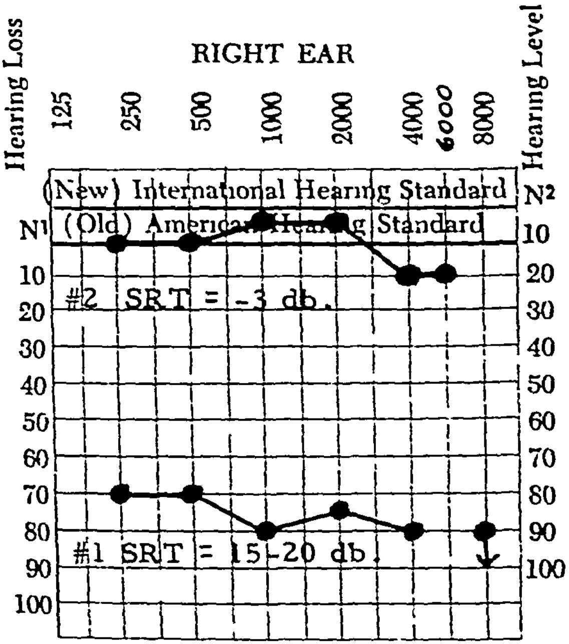

Fig 4 Air conduction thresholds for the same ear of one child obtained on the same day Normal threshold obtained following suggestion to the child that he had misunderstood directions or was pretending

3 Strained relationship with mother

4 Immature patterns of behavior

Psychotherapy was recommended if difficulties in social adjustment persisted

The fourth child a 15 year-old male was reported to have been failing in all subjects and expelled from a public school and a private school because he would not study Temper tantrums at school had occurred with subsequent referral to a psychiatrist for six months treatment Four successive audiograms over a two month period indicated a 65 to 95 db pure-tone bilateral loss with speech reception thresholds of 10 to 25 db

Speech discrimination test errors and test behavior were markedly different from those normally expected with organically impaired ears

This child told the audiologist that he did not hear the sound (l) and proceeded to repeat "fap" for *flap*, "fing" for *fling* When given the word, *puff*, he said, "I got the 'uff" (followed by a long pause) and then the question, "Did you say puff?" When given the word, *wire*, he was overheard to whisper to himself "wire, wire" before saying into the microphone the word, *fire*

It was requested that he return for follow up studies, but he was not seen for two years An interview with his father prior to the audiologic testing at this time revealed his general psychological and emotional state had improved He was described as being an entirely different boy, happier at home with fewer temper tantrums Normal pure tone and speech audiometric results were obtained without difficulty (see Fig 6), however, on completion of the testing, the boy stated, "I wish I could see as well as I can hear My eyes get weaker and weaker I can't see anything in here clearly "

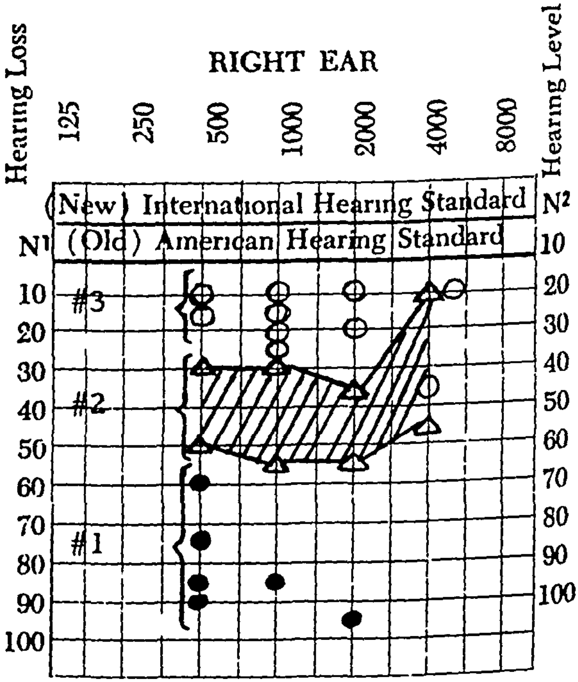

Fig 5 Range of variability in air conduction thresholds for same ear of one child obtained on three successive visits Shaded area indicates difference in ascending and descending thresholds on second visit

It should be emphasized that clues to non-organicity occur within the tests for speech reception threshold and speech discrimination as well as in the completed test scores themselves The response pattern of three children in this group was atypical, that is, differing from the responses of the normal or organically impaired ear

Half-word spondees were frequently given, such as "boy" for *cowboy* A grouping of errors might occur, the child always giving the first syllable of a spondee as "foot" for *football* "earth" for *earthquake*

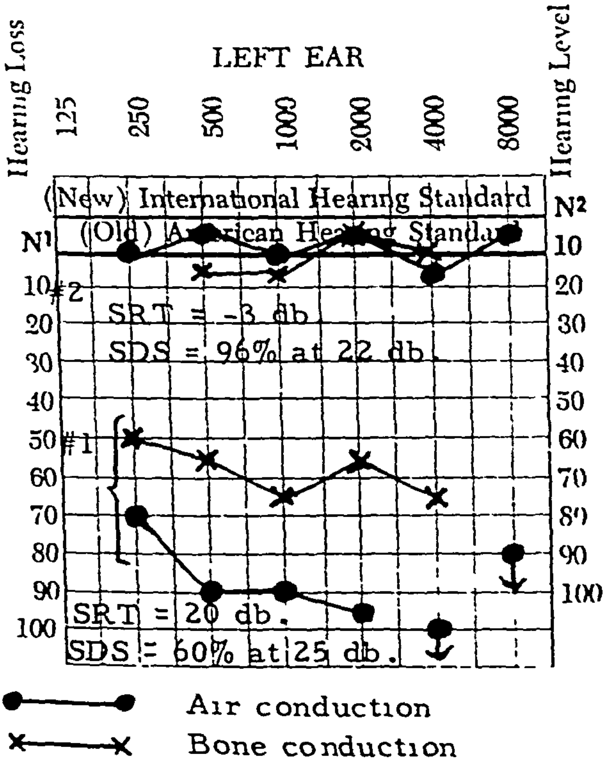

Fig 6 Initial audiogram (No 1) for same ear of one child compared with final audiogram (No 2)

When given speech discrimination tests, several children were observed to substitute consistently words that rhymed with the test word (without relation to relative intelligibility). Long silences frequently followed the audiologist's word presentation, the child later repeating the word hesitantly and with a rising inflection A grouping together of identical types of error was observed, such as repeating seven words in a series each with the final consonant omitted

CONCLUSION

Otologists and school physicians who make recommendations for special classes should be aware that non-organic hearing problems can and do exist, and that the first clue to this is the discrepancy between the pure-tone audiogram and conversational-speech ability

A diagnosis on the basis of the pure-tone audiogram alone is not sufficient, and speech audiometry should be considered absolutely essential in children's hearing tests The speech reception threshold in most instances can be relied upon to come closer to the patient's actual loss

The impression and final diagnosis of normal hearing is best made after complete otologic and audiologic evaluation and psychologic or psychiatric evaluation when indicated

Full knowledge of the hearing mechanism and testing technique, together with adequate equipment is necessary

The audiologist should possess sufficient background in the testing of normal ears and patients with organic losses so that the response pattern and test behavior of the patient with a non-organic loss will stand out as markedly different

On the basis of our experience, we feel that the more deliberate and willfull the non-organic loss appears, the more closely scrutinized must be the entire school and home adjustment picture, and the more likely it is that psychiatric referral should be made

The otologists may hesitate in some instances to confront a willfully malingering child directly with the statement that he hears normally, face-saving may be of great importance to a particular child After testing, the suggestion to the child that his hearing is improved and much better can act as a necessary face-saving device Frequently, simply telling the child in a matter-of-fact way that the tests showed that he had normal hearing and that he must simply have misunderstood directions or perhaps was pretending may be indicated The child may feel better having this out in the open and be somewhat relieved of the necessity to continue the pretense This is not meant to imply that the underlying basic reasons for this behavior are in any way altered by such superficial treatment In the conscious malingerer, this of course can be explored only through psychiatric evaluation

With the child who is not willfully malingering and who exhibits no symptomatology other than the apparent inability to listen in the test situation, it would appear desirable to stop all testing at school Testing should be performed as with the willfully malingering child only to the extent necessary in the otologist's office Comments, both at home and at school, with reference to the child's hearing are probably best discontinued Further elaboration or generalization as to causal factors, significance of this behavior or prognosis is not within the scope of this paper We can recommend only continued observation and gathering of data by others

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BILATERAL TONSILLECTOMY FOR PERITONSILLAR ABSCESS[*]

BENJAMIN M VOLK, M D,
and
EDWARD C BRANDOW, JR, M D,
(By Invitation)
Albany, New York

In our opinion, bilateral tonsillectomy for peritonsillar abscess is the procedure of choice Incision and drainage for peritonsillar abscess has been the standard accepted therapy literally for centuries This procedure consists of the evacuation of pus, perhaps daily reopening of the incision and, some weeks later, tonsillectomy

The patient, however, during the acute stage of the illness usually has marked dysphagia, drooling, severe pain, elevation of temperature and considerable dehydration All of these symptoms, on the average, may persist for two to five days We all have had the experience of not obtaining pus in incising what we thought surely was an abscess We also may have repeated this frustrating experience daily for a few consecutive days Eventually, the abscess would rupture spontaneously, as it does in the majority of cases, or extend laterally, posteriorly or inferiorly Occasionally, troublesome bleeding would occur Fortunately, a fatality would rarely result although it is a probability

Historically, it is interesting to note the evolution of the surgical treatment of peritonsillar abscess In the Fourteenth Century, Guy de Chauliac,[1] a French surgeon, described the procedure for incision and drainage Essentially, this has changed in no respect up to the present time As time passed, various progressive surgical modifications were recommended,

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[*] Read at the Sixty-Third Annual Meeting of the American Laryngological Rhinological and Otological Society, Inc Miami Beach, Fla March 16 1960

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among them, incision and drainage with removal of a small piece of tonsil, the removal of only the upper half of the involved tonsil, partial dissection of the upper pole with luxation and removal of the entire tonsil a few days later when the severity of the acute infection had subsided, packing of the abscess-cavity for a few days with removal of the offending tonsil, and finally, bilateral tonsillectomy In 1859, Chassaignac[2] performed the first complete tonsillectomy for peritonsillar abscess and was the first to describe an abscess forming in the loose connective tissue between the tonsil and the superior constrictor muscle

The great volume of literature concerning this surgery emanated in Europe in the latter part of the Nineteenth Century and in the early decades of the Twentieth Century Oddly enough, there are only a few articles on this subject in American literature, and none in the last quarter of a century Dr Harry Baum[3] of Denver gave a very enthusiastic and comprehensive paper in 1926 before the Midwestern Section of the American Laryngological, Rhinological and Otological Society in Omaha, Neb The interest in what might be termed radical surgery was largely stimulated by many case reports of fatalities in this disease which occurred at this time Many septic infections, therefore, were treated by tonsillectomy, with recovery, which resulted in many contributions to the literature of otolaryngology It should be emphasized that all of this pioneer work was done before the introduction of chemotherapy and antibiotics

Impressed by these large series of cases without any complications attributable to the surgery, we began this procedure in January, 1957 Since that time, it has been used routinely in all cases of peritonsillar abscess We wish to present our experiences with 36 cases, being aware that the number of cases is statistically small and that no definite final conclusion can be made at the present time, therefore, this is considered a preliminary report It is difficult to accumulate a large number of cases Apparently, this acute condition is occurring less frequently because of the improving health of the general population and also through use of chemotherapy and antibiotics

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120 cases In our series, the same age group incidence was 80 per cent

Peritonsillar abscess is not always located anteriorly at the superior pole, therefore, the usual diagnostic criterion of a markedly injected bulging upper pole with deviation of the uvula is not always reliable These signs may not be present If the abscess or abscesses are located elsewhere in the tonsillar fossa, one must rely on history and the presence of trismus Virtanen's[6] series of 379 cases shows only 51 per cent had an upper pole abscess and 0 9 per cent was multilocular In the Bateman-Kodicek series of 120 cases, 50 per cent was located at the upper pole, 5 per cent was multilocular Virtanen also found 14 per cent had bilateral abscess involvement Grahne also found bilateral involvement in 7 per cent In our series, 62 per cent had an abscess in the upper pole, 10 per cent was multi-locular or multiple and 8 per cent or three cases were bilateral, all masked on the uninvolved side and found incidentally The remainder was located in different areas in the fossa

In this series, the duration of symptoms varied from two to seven days Twenty-two patients stated that they had one or more attacks of tonsillitis, and four others had had one or more attacks of peritonsillar abscess, each having had one or more episodes of incision and drainage One patient had had a tonsillectomy performed seven years previously Two patients had extension of the infection to the para-laryngeal region with swelling of the epiglottis

Preoperatively, all of the patients had some elevation of temperature ranging from 99 4° F to 102° F, with two having 103° F and one 104° F Postoperatively, all patients, with three exceptions, had normal temperatures within four to 24 hours These all subsided within 48 to 72 hours The majority of patients operated upon at the Albany Hospital was discharged within 24 to 48 hours, none remaining for more than 96 hours Bacteriologically, only 17 cases were reported as follows five—beta hemolytic streptococcus, ten—streptococcus viridans, and two—diplococcus pneumonia

No fatalities or complications were reported either in

The surgery was performed by members of the attending and the resident staff in both the Veterans Administration Hospital and Albany Hospital. All of these cases were handled in similar fashion, each being given penicillin four or more hours before surgery. Another antibiotic was used if there was any question of penicillin sensitivity. If necessary, intravenous fluids were also administered. As indicated, routine preoperative medication was used.

Twenty-two of the 36 cases received local anesthesia, and 14 were done under endotracheal anesthesia. The type of anesthesia used depended on the patient's age and degree of apprehension, local anesthesia being preferable. In those patients receiving general anesthesia, it is important that a competent anesthesiologist be available and that the Rose position be used so that in case of spontaneous rupture of the abscess the dependent head position will preclude the possibility of aspiration.

The surgical technique was identical with that of a routine interval tonsillectomy and was no more difficult. The abscess actually separates a portion of the tonsil from its bed. After incision, one usually finds the tonsil more than half dissected. Local injection keeps bleeding at a minimum and is also used in conjunction with a general anesthetic. Trismus is not an inhibiting factor for the removal of the tonsil. Removal of the opposite tonsil is uneventful. We feel, however, that this is also necessary because of the high percentage of recurrence of an abscess and because of finding unsuspected asymptomatic pockets of pus.

Many of these patients were treated as emergencies, the surgery being frequently performed at night. Postoperatively, antibiotic therapy was continued for five days. The care was similar to that of a routine procedure. No patient had any associated medical condition that contraindicated the surgery.

Peritonsillar abscess is primarily a disease occurring in the prime of life. Grahne,[4] reporting 725 cases, found that 79 per cent were in the ten to 40 year age group, Bateman-Kodicek[5] found approximately 85 per cent incidence in the group of

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Grahne's 725 cases or Bateman-Kodicek's 120 cases These patients, postoperatively, reacted similarly to those having an elective tonsillectomy, and no unusual bleeding occurred These series are being presented because they are recent and occurred in the post-antibiotic era In our series, no primary or secondary hemorrhage occurred

Not all of the cases came directly under our care but were first managed conservatively by others These cases help illustrate the advantage of the one-stage immediate tonsillectomy Two cases had spontaneous rupture with abatement of symptoms, yet pus was found at the operation the next day One case had incision the day before with no pus A large pocket was found behind the inferior pole during tonsillectomy Another had incision and drainage with complete subsidence of symptoms, yet seven days later, pus was found at the operation Without tonsillectomy, each of these cases would have had residual pus left in the throat Certainly, the three cases with pus behind the opposite and unsuspected side were much benefited

Tonsillectomy for peritonsillar abscess conforms to the basic surgical principles of adequate incision and dependent drainage The entire medial wall is excised Simple incision for drainage is not comparable Drainage is not dependent for residual pockets may be left behind, possibly resulting in more abscess formation or possible extension to the parapharyngeal or the paralaryngeal region Operative trauma or injection in an area of cellulitis has not caused either local or systemic spread of infection If a single cavity has been successfully incised and drained, the patient may either not return in the optimum time for interval tonsillectomy, or may not return at all This results in the tonsil's becoming exceedingly scarred in the fossa, making dissection exceedingly difficult As a result, excessive scar tissue may leave tonsillar tissue behind which in itself may cause a recurrence of infection or an abscess

COMMENT

Immediate tonsillectomy as a one-stage procedure is the treatment of choice in the management of peritonsillar abscess This has many advantages over incision and drainage The only method of obtaining adequate and dependent drainage is by removal of the tonsil Incision alone may leave behind residual pockets of pus, particularly if the abscess is multi-loculated Incision may fail to give pus as the abscess is not located behind the upper pole in a significant number of cases After incision, additional procedures for further evacuation of pus may be necessary on the second and third days Enough patients have pus on the opposite side to warrant doing a bilateral tonsillectomy The abscess separates the tonsil from its bed and after adequate incision, the tonsil is usually more than half dissected out Bleeding is kept at a minimum by local injection Antibiotics protect against further spread of infection The complications from the operation are no higher than with interval tonsillectomy These patients, postoperatively, are most grateful to be so immediately relieved of their acute discomfort Their postoperative course is uneventful The convalescence of the one-stage operation is certainly much decreased This procedure has been performed in over 4,000 cases for the most part in Europe, and to a large extent, before the days of antibiotics It has been found to be a safe and effective method

CONCLUSION

We have presented our experiences with 36 cases Small though it may be, it is hoped to stimulate interest in this country, in what we feel is a very rewarding procedure

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LYMPHOSARCOMA OF TONSIL

Discussion and Report of Case with Recovery*†

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Lymphosarcoma of the tonsil is a rare disease,[1] and when a case is seen by the surgeon, it does cause a sense of concern with foreboding of fatality, however, the prognosis is more favorable than is generally realized, and with today's therapy the patient does have a near 40 per cent chance of recovery[3,4] Not to leave too rosy a picture, there are basic facts that must be considered

Lymphosarcoma is a cancer of non-epithelial origin arising in lymphatic tissue Lymphosarcoma may arise in practically any organ or gland, and spread to any area of the body It is notorious for sparing little, and harming much It occurs in all ages, males slightly more than females, and in all races

Just how rare is lymphosarcoma of the tonsil? The incidence is well shown by a summary of cases as reported by the Tumor Registry at the Los Angeles General Hospital[5] During the period 1944-1958, 14 years, there were two cases of lymphosarcoma of the tonsil and one case of reticulum cell sarcoma of the tonsil reported During this same period there were 131 cases of carcinoma of the tonsil reported During this 14-year-period there were 26,731 neoplasms registered at the Los Angeles General Hospital Registry The surgical load at that hospital for this same period, 1944-1958, was 188,758 patients

The annual patient load, both in and out patients, during this past 14 years has been 80,366 patients The total load for the 14-year period makes a grand total of 1,125,124 pa-

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tients seen The rate of incidence, using these statistics, would mean that one patient in 375,000 patients would develop lymphosarcoma of the tonsil, and that about one case of lymphosarcoma of the tonsil would appear for each 62,000 surgical cases operated upon for all conditions

The consideration of clinical findings aids in an evaluation both as to treatment and prognosis A classification as presented by Diamond[6] for lymphosarcoma is as follows

Class 1 Disease limited clinically to a single focus, with no constitutional symptoms, *i e*, fever, weight loss, anemia, fatigue, etc

Class 2 Disease limited regionally, such as homo-lateral cervical adenopathy, with or without constitutional symptoms

Class 3 Generalized disease with constitutional symptoms and signs

Using this classification, the physician is in a better position to consider the form of therapy to be used, whether surgery, X-ray, chemotherapy, or a combination of these three

The pathological classification of lymphosarcoma, by Herbut,[7] designates the types as follows

1 Giant Follicular Lymphoblastoma

There is an increase in the number and size of the lymphoid follicles The follicles are composed of reticulum cells in the center, lymphoblasts beyond these, and lymphocytes at the periphery This is the least malignant of the lymphoblastomas When it terminates in the death of a patient it may still remain in its initial form, or may be transformed into lymphosarcoma, lymphatic leukemia, Hodgkin's disease or reticulum cell sarcoma This shows the close relation among the lymphoblastoma group of diseases In the borderline cases the separation of this group from the less differentiated forms is largely arbitrary Different names have been applied to giant follicular lymphoblastoma including malignant lymph follicle hyperplasia, follicular lymphoblastoma, follicular lymphadenoma, follicular lymphoma, and Brill-Symmer's disease

2 Lymphosarcoma

The more advanced stage with the lymph node diffusely involved This may also represent a local, regional or generalized disease In time these patients may show blood and marrow changes of chronic lymphatic leukemia The cells of the lymph node may be small and mature, (lymphocytic lymphosarcoma), or large (lymphoblastic lymphosarcoma)

3 Hodgkin's Disease

This disease is often divided into a sarcomatous and granulomatous variety The sarcomatous form is less common with the dominant cell identical with reticulum cell sarcoma, and is considered as a granulomatous form which has been transposed into the more malignant type The granulomatous type is the usual form in Hodgkin's disease Histologically, there is complete loss of normal glandular structure, with a diffuse infiltration of a variety of cells including lymphocytes, plasma, monocytes, neutrophiles, and Sternberg-Reed cells

4 Reticulum Cell Sarcoma

Here the node is replaced by reticulum cells, which may also be a local, regional, or a generalized disease In this group the predominant cell type is greater than two to four times the size of the mature lymphocyte This group includes stem cell lymphomas, lymphoblastomas, and lymphoblastic lymphoma It should also be stated that serial sections of a node or of nodes may manifest all stages or types of lymphosarcoma including the above manifestations

The average survival⁻ of patients with giant follicular lymphoblastoma is 4 6 years, *of lymphosarcoma and Hodgkin's disease about two years, and of reticulum cell sarcoma about six months*

The development of lymphosarcoma is insidious There is no discomfort noted during the earliest stages In the tonsil area, obstruction because of size may be the first complaint In the late stages the findings are those of a generalized cancer with anemia, cachexia, weight loss and shortness of breath, etc

The diagnosis is made by examination and confirmed by biopsy The significant finding is the marked enlargement of only one tonsil, which may be three, four or five times the size of the average tonsil In a recent survey[5] of 422 tonsils, the average weight, all ages, was from 4 5 grams to 8 5 grams (Case 1 weighed 22 grams) The involvement of the tonsil is usually so extensive that removal of the tonsil seems indicated, and the more expedient procedure A biopsy, or removal of a small portion of the tonsil may not give the patient relief from obstruction symptoms A biopsy may not present an accurate pathological picture This is due to the nature of the disease where different stages or types of the disease may be present in the larger specimen[1]

The treatment of lymphosarcoma of the tonsil as compared to involvement in other parts of the body, is surgical removal of the involved tonsil as indicated above, however, this represents only the first phase of treatment, for the second, and most important phase, is radiation therapy. The treatment of choice for lymphosarcoma is X-ray Surgery, except for diagnosis, is not advocated

Patients in Class 1 should be treated aggressively by X-ray with potential cure in mind following the removal of the involved tonsil[6] Certainly it seems unwise to do a bilateral tonsillectomy just because you are working in the throat To save the patient as much as possible and maintain his health, is the surgeon's first concern during this preliminary, critical stage

Class 2 patients need much more intensive X-ray therapy with the Roentgen dosage increased, and given in multiple portals to cover the involved area The desired dosage to be designated by the radiologist

Class 3 patients fall into the palliation therapy group The dosage used is mainly to lessen the size of the tumor mass, and decrease symptoms In this group chemotherapy may also be used While chemotherapy has been used with temporary relief, this form of therapy should not be relied upon as curative

Chemicals used include Nitrogen Mustard (HN2) This is of help in Classes 2 and 3 where remissions occur of weeks to months HN2 is given intravenously in a single 0 4 mgm per Kg dose, or divided doses in a period of several days Should this be considered in treatment, Diamond[6] has presented an excellent report on its use

Triethylene Melamine (TEM) is a compound with Nitrogen-Mustard-like action It is given orally It has the ability of acting like radiation therapy in controlling lymphosarcoma but without the same lasting effects of X-ray It has adverse reactions which should be watched for These reactions include a depression of the white blood cells and platelets, and the presence of hyperuricemia

CASE REPORT

M A , white female, six years of age, was first seen October 19, 1956, with a history of having a husky voice of three weeks' duration One week prior to being seen, there was difficulty in swallowing and a definite mouth breathing which had not been present before The patient did not complain of pain, had no fever, and except for her muffled voice, did not appear in discomfort

The past history of the patient was essentially negative, no history of sore throats, no ear trouble, no allergy history

Examination showed a well developed white female Ears, canals clean, TM grey and glistening, nose, normal in appearance, throat, right tonsil presented a very large mass approximately 2x4 cm extending into the right pyriform sinus The mass was not ulcerated, and had a smooth red surface, the left tonsil was moderately enlarged, a mild chronic inflammation present, pharynx showed no acute infection, no edema of the palate, cervical adenopathy, right 2 plus, left, 1 plus, no tenderness in the tissue of the neck Spleen not palpable

The patient was admitted to the hospital the same afternoon she was first seen X-rays of the chest were ordered and also a complete blood count The X-ray report follows

PA and left lateral views were taken The visible bony thorax appears intact The diaphragm is smooth and at normal levels with clear costophrenic sulci The heart is oblique in type and well within normal limits as to size and configuration The great vessels and trachea are within normal limits There is a fullness in both hilar regions In the right hilus, there is a suggestive shadow of a 1 cm lymph node overlying the pulmonary segment In the left hilus, the hilus is slightly enlarged and slightly lumpy suggesting possibility of lymph nodes in this area The remainder of the lung fields are essentially clear The lateral view adds no additional information

Roentgen Conclusion Quite suggestive for early hilar lymph adenopathy This would bring up the possibility of a lymphoma Otherwise negative heart and lungs

The C B C Hemoglobin 13 0 gms , W B C 9,900, Neutrophiles 31%

(Segmented 31%), Eosinophiles 9%, Lymphocytes 52%, Monocytes 8%

Urine specific gravity 1 021 pH 5 0, pus cells 5 6

The patient was taken to surgery the following morning, and a right tonsillectomy was done with the patient in the Rose position The left tonsil was left intact The mass removed dissected freely at the upper pole, but was more adherent at its base The mass extended into the right pyriform sinus and encroached upon the right wall of the epiglottis When the tumor was removed the fossa presented a clean base One tie was placed in the right tonsil fossa The pathology report follows

The specimen consists of 22 gms of tonsil tissue measuring 4x3 5x2 5 cm in its greatest dimensions The cut surface shows a homogeneous pink tan fleshy tissue lacking any gross follicular pattern There are no crypts in the center of the specimen

Microscopic There is almost total disorganization of normal architecture The bulk of the specimen consists of a monotonous growth of large lymphocytes without evidence of significant reticulum or fibrous connective tissue proliferation

Diagnosis Lymphosarcoma right tonsil

The patient was discharged to her home October 23 and was seen on the tenth postoperative day, October 30, at which time the throat was healing nicely She reported to the Roentgenologist for X-ray therapy November 1, 1956 She received a total of 3000 Roentgens from November 1 to November 29, 1956 The patient continued to improve and upon subsequent visits the cervical adenophy disappeared She is now in good health, and at the last examination November 16, 1959, presented no evidence of disease

In checking the hospital records, a second case of lymphosarcoma of the left tonsil in a six-year-old male was found This is the second case located in a ten-year period in a surgical case load of 78,662 patients The case history follows

CASE REPORT No 2

J Y white male age six years was first seen June 10, 1955, with a history of sore throat of approximately two months' duration, dysphagia two plus weeks and dysphonia the past ten days In the ten days prior to being seen there was noted enlargement of the glands in the left cervical area The patient was admitted to the hospital June 14, 1955

Physical examination at the time of admission presented a well developed male child with dysphonia but no respiratory distress Ears, canals clean TM grey and glistening nose normal in appearance, throat right tonsil small in size left tonsil marked hypertrophy approximately 3 5x2 cm with erosion of its medial surface Neck marked adenopathy of left cervical glands adjacent to the sternomastoid muscle Heart and lungs normal, spleen not enlarged

The patient was taken to surgery June 15 1955 and a left tonsillectomy was done The tonsil shelled out of its capsule with no great difficulty and two ties were placed in the left tonsil fossa The right tonsil and the adenoids were not removed

Pathology report The specimen consists of an irregular mass of tonsil tissue measuring 4 cm in its greatest dimensions and weighing 13 gms

Microscopic Shows almost complete obliteration of the follicular pattern Mitotic figures are quite common The bulk of the tumor consists of large hypochromatic cells of the lymphocytic series

Diagnosis Lymphosarcoma of left tonsil

The patient was discharged the following day, and was checked post operatively on June 25 In the meantime, therapy was requested through the City of Hope, and the patient was admitted to this hospital on June 27, 1955, as an out patient He received a total of 4275 Roentgens during the next 30 days The patient gradually became worse, and was admitted to the City of Hope Hospital on August 15, 1955, with an elevation of temperature, and swelling of the left side of the neck He became progressively worse until September 1, 1955, when a tracheotomy was done because of increased dyspnea The patient expired on September 2, 1955 An autopsy was refused by the parents

SUMMARY

A review of the clinical classification, also pathological, findings, and treatment of lymphosarcoma is presented

The treatment of choice is radiation therapy, preferably given early and in adequate dosage

A case presentation of a six-year-old female with recovery, and a six-year-old male who did not recover is given

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PSYCHOGENIC PROBLEMS[*]

C STEWART NASH, M D ,

Rochester, N Y

The motto of the University of Salamanca reads *What nature hath denied this University cannot provide*

What a travesty on the practice of medicine! St Luke, a fellow physician, would have said *be of good comfort, thy faith "hath made thee whole"* *(St Luke 8-48)*

Far be it from me to belittle the values of modern surgery and wonder drugs, but I admonish you to regard with respect the clinical and psychogenic importance of modern *Empiricism*

Empiricism—or "Empirics" as it was called—began in the Second Century B C as one of the post Hippocratic Schools of Medicine In its search for a line of treatment to benefit a particular set of symptoms, this school employed what it called *The Tripod of Empirics* the legs of which were

a One's own observations,

b Learning obtained from contemporaries or predecessors and

c Analogy—that is, in case of new diseases, the forming of conclusions from a study of known maladies which resembled those under consideration

So far as I have been able to ascertain this tripod soon came to include any and all methods of healing (medical, surgical, psychogenic, spiritual, ad infinitum), orthodox and unorthodox

With the advent and growth of this School of Empirics, the old Cock-of-the-roost game began The Cock and his

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[*]Read at the meeting of the Southern Section American Laryngological Rhinological and Otological Society Inc New Orleans La Jan 15 1960

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loyal supporters were orthodox practitioners, those with ideas unacceptable to the group were "quacks" This Cock-of-the-roost situation has come down through the ages and continues unabated, repeatedly, however, there have been changes of actors and scenes

Let us meet the situation head on! The cries of "Quackery" and "Charlatanry" come from doctors—rarely from patients—Why?

1 In time the doctor *should* know whether a given preparation or procedure is valuable or value-less

2 In time the doctor *should* know whether a given preparation or procedure is harmful or harmless

Unfortunately, a doctor may come up with the wrong answers to both these propositions and thereby fail to recognize a medical prize worth striving for

It is within this uncertain Cock-of-the-roost medical environment that *some* of our psychogenic problems arise

Others—as we shall see—are of a more personal nature

First, we shall observe some of the environmental shifts in medicine, all of which have had and are continuing to have psychogenic repercussions in both patients and doctor

a Personally I have read dozens of testimonials concerning the benefits from two of the most remarkable pain relieving remedies this country has ever known Of course, they contained whisky and opium, but no one became an alcoholic or a drug addict, he simply kept on taking such an easily procurable patent medicine and lived happily ever after Peter B Kyne's book "The Clarion" took care of the immediate situation, but the psychogenic problem created by unremitting pain and distress remained Replacing whisky and opium combinations, we now have barbiturates and tranquilizers, but to what avail?

b As a teen-ager I was acquainted with itinerant Medicine Shows I have known of patients having teeth pulled "painlessly" in public and with the percussion of a bass drum as the only anesthesia

Within the last six to seven months the Ritter Company, Inc, of Rochester, New York, received an order for 1,000 newly blue-printed Sono-Anesthesia units to relieve pain and to be used for other symptoms in and about the ears and head Doctor Hallowell Davis, Director of Research at Central Institute for the Deaf in St Louis, informs me that both the apparatus and its usage are safe and effectual means of treatment

Three months ago an English otologist requested a place on the annual program of the Triological Society to read a paper on "Supra-Sonic Sound" as a surgical procedure in the management of Meniere's disease

c As a young aural surgeon I pooh-poohed "Sound Stimulation" and "Otitic Vibration" an effective or ineffective psychobiological agents in the handling of disorders of the ear Today, I am constrained to give thought to "Medical Acoustics" and "Stapes Mobilization" as the legitimate progeny of these psychogenetic grandparents

d Also within the last year one of our own "Triological" members has popularized the use of vinegar and honey and—other home remedies in a book called "Folk Medicine" that has been on the best-seller list of the *New York Times* for about eight months and to date has had sales in excess of 245,000 copies

So far we have brought forward for illustration four environmental shifts in medicine that somewhere in their elucidation have contained real or suspected elements of psychogenesis

May we return to our simile? The doctors are the Cocks-of-the-roost, it is they who attempt to adjudge which factors in modern empiricism are based on psychogenetic phenomenon and which are founded on organic response—but doctors can be wrong This, the patients know, and therein lies an occult and taboo psychogenetic situation

Second, shall we remove ourselves from these environmentally psychological problems to more intimate and personal ones?

PERSONAL PSYCHOGENETIC STOCK-IN-TRADE

A Salesmanship

B The Doctor-Patient Relationship

C Wisdom

D Words

A *Salesmanship*

Never have I experienced an empiric—ancient or modern, false or true—defaulting on salesmanship

For years I listened agog and with vaudeville-like attention to the preachments of Dr Brinkley—and what a power he was! $50,000 a week, I am told, it cost to operate his broadcasting station, first in Medford, Kansas, and later in Del Rio—just across the line People loved his brand of medicine and they almost elected him Governor of the "Sun Flower State"

Of lesser lights there was Dr Locke, the Canadian physician, who never charged more than $1 00 a treatment and who from his rocking chair in an orchard saw several hundred patients a day

Please understand me, I do not recommend showmanship in the practice of medicine, but I do say that medicine is not and perhaps never will be just human engineering and chemistry

B *The Doctor-Patient Relationship*

What is *not* appreciated by some doctors—though it is perceived by their patients—is the power for good or ill that lies in the interplay of doctor-patient personalities

You are familiar with this phase of the subject

C Wisdom

Paracelsus once said "*The greatest and highest of all qualifications which a physician should possess is wisdom—and without this qualification all his learning will amount to little or nothing We cannot find wisdom in books, nor in any external thing We can find it only within ourselves*"

D Words

Of all the technical aids which increase the doctor's power of observation, none comes even close in value to the skillful use of words—the words of the doctor and the words of the patient Throughout all of medicine, use of words is still the main diagnostic technique—and while in therapy many mechanical and chemical aids are truly miraculous in their effectiveness, words continue to play a tremendous role

The aim in talking with a patient is to find out not only about his immediate symptoms but about him—his strength and weaknesses, his experiences throughout life and his reaction to them

In medical school a student spends hours in learning to dissect, to palpate, to percuss, etc—all essential things—but he is presumed to know how to talk This is *not* necessarily true

Success in talking with a patient will certainly be enhanced if the doctor acts like a cheerful human being, sincerely interested in the doings of another human being

Quizzing, grilling, and the third degree have no place at all in talking with a patient Remember the patient cannot talk while the doctor is talking—so often the less said the better Listening is made easier if the doctor realizes that there is no need to make a diagnosis out of every bit of information and does not feel compelled to give premature advice

Be circumspect in dealing with all patients, particularly the difficult or angry patient Help him to talk about his angry feelings Anger often is a defense against anxiety—anxiety from any cause Sometimes the patient makes the doctor angry, sometimes the doctor makes the patient angry

In any case, ideas that float around unspoken are tremendously threatening because they have no boundaries, no limits Once they are put into words they are to a great extent fixed within the limits of those words

If they can be examined as an object, an object that both the doctor and the patient can see, then the threat is largely lost

Dr Brian Bird, Associate Professor of Psychiatry at Western Reserve University, who is responsible for most of these ideas on "words" has said that a common tendency among many doctors is to talk too much to patients about themselves and their families

Matching symptoms with a patient may be fun but it is hardly sporting, and one never knows where it will end

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THE
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THE USE OF HEPARIN AS AN ANTI-INFLAMMATORY AGENT*†‡

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and
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Salt Lake City, Utah

INTRODUCTION

Numerous experiments in our laboratories which have been published in detail elsewhere[1,2] indicate that heparin has a marked capacity to bind endogenously released histamine In addition, its capacity to bind certain other basic substances (histamine releasers) has been well established by both *in vitro* and *in vivo* experimentation[3] Other studies indicate that heparin has a dramatic life-preserving effect in animals given lethal doses of histamine releasers, such as Polymyxin, 48/80, snake venoms, etc - It has been stated that histamine is free or protein bound in cells and that sulfated ground substance binds extracellular histamine The extracellular histamine can be released and when free, exerts its biological effects Heparin acts in the nature of a biological exchange mechanism in which its release from extra or intracellular

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*Read at the Sixty-Third Annual Meeting of the American Laryngological Rhinological and Otological Society Inc Miami Beach Fla March 16 1960

†From the Connective Tissue Study Group Departments of Anatomy and Surgery (Otolaryngology) University of Utah Medical School and the Memorial Medical Center Salt Lake City Utah

‡We credit the Department of the Army (Grant DA-49-007-MD-130) for supporting this work

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sites, results in a greater availability of binding sites for histamine[4] This binding of the histamine prevents its setting off a generalized stress response[1,4] Similarly, serotonin, which is probably the natural edema and inflammation producing agent in the rat and mouse[5] is also bound by sulfated mucopolysaccharides Its participation in the human is, of course, well established[6] Heparin thus has a more, or at least as fundamental a property as a biological ion exchanger as its role in anticoagulation

It seemed important to ascertain whether heparin had any therapeutic effect in the human, particularly in those conditions in which histamine release and the allergic inflammation which results from it, may be the primary cause of the malady Thus, several types of allergic and inflammatory diseases were treated with heparin or heparin combined with either polymyxin, neomycin or streptomycin It had previously been shown that combinations of heparin and these antibiotics did not eliminate the antibiotic capacities of the drugs bound to heparin[7] Lesions that could be observed directly, such as those on the skin, or systemic effects as in asthma and hay fever, were studied to ascertain whether these clinically observed types of allergic inflammations could be blocked by the administration of heparin The cases described here were treated according to the methods outlined below

MATERIALS AND METHODS

Since, of all the allergic states, weeping eczema of the ear could be most easily examined, it was chosen as the first allergic disease to be treated by heparin Clinically, aural weeping eczema may be divided into two types one type will respond to almost any medication and will clear quickly, a second type is very resistant to all treatments Only cases treated unsuccessfully by competent otolaryngologists and/or dermatologists for a period of at least two years were selected for trial by the heparin

Cultures of the existing organisms were taken before the treatment was started Commonly found organisms were hemolytic staphylococcus, proteus vulgaris or pseudomonas aeruginosa. After the identification of the organisms, their sensitivities to the appropriate antibiotic were determined. When possible, the alkaline antibiotics of choice and hydrocortisone were used for the treatment. Prior to this, all the usual treatments such as hot packs, argyrol, etc., had been tried unsuccessfully; therefore, no attempt was made to repeat their use. If the hydrocortisone and antibiotic treatment was successful, the subject was not accepted for trial on heparin.

Hemolytic staphylococci were inoculated on blood agar plates.[8] The blood agar was prepared in three ways: in one, discs of neomycin, polymyxin and streptomycin were placed on a standard blood agar plate, inoculated with the hemolytic staphylococcus aureus. The area of clearing about the discs was measured. In a second plate, the discs were first saturated with heparin and then placed on the agar, and finally, on the third plate discs were placed on the blood agar to which heparin had been added. The clearing in the last two was slightly greater than on the first plate. Heparin either has antibiotic effects of its own or possibly it permitted the diffusion of the antibiotic across the blood agar more easily and, therefore, dissemination was increased and the area of clearing enlarged. Possibly both mechanisms may occur. Further investigation of the antibiotic role of heparin is being pursued. Heparin did not inhibit the action of any of the antibiotics tested.

In order to treat the patients selected, a 3 per cent mixture of the appropriate antibiotic was suspended in 2½ per cent hydrocortisone solution, and 1 per cent heparin.* In the later work, Bio-Parin† and a mixture supplied by the Darwin Laboratories‡ were used. Bio-Parin contains neomycin, polymyxin and heparin. The mixture prepared by Darwin Laboratories for our use contains dihydrostreptomycin 2 per cent, hydrocortisone 25 per cent, neomycin 2 per cent, and heparin 1 per cent. Dihydrostreptomycin was substituted for polymyxin in this preparation because other studies to be published indicate that dihydrostreptomycin had a wider range of antibiotic activity

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*Supplied by Mr. Gil Aaron of Broemmel Pharmaceuticals.

†Trade name for the mixture given here. Broemmel Pharmaceuticals.

‡Supplied by Mr. Leon Freeman, Darwin Laboratories.

RESULTS

Table I shows that 31 cases of infectious external otitis were treated These were divided into two categories the first category had 12 cases which showed infection only, a second category of 13 patients contains those with infection and weeping eczema Bacteriological studies showed organisms sensitive to either neomycin or streptomycin, as well as the other antibiotics These antibiotics were chosen because their alkaline structure allowed a complex with heparin

TABLE I

External Otitis Media

No Cases	Sex M	Sex F	Infection Alone	Infection + Allergy
31	15	16	12	
			Neomycin Sensitivity	Neomycin Sensitivity
			6	12
			Streptomycin Sensitivity	Streptomycin Sensitivity
			6	7

Results

Infection Alone						Infection and Allergy					
Neomycin Sensitivity			Streptomycin Sensitivity			Neomycin			Streptomycin		
1	2	3	1	2	3	1	2	3	1	2	3
0	4	2	0	1	5	2	2	8	0	1	6

Results of the treatment are given in three categories *1* Shows very poor results *2* Indicates moderately successful treatment *3* Excellent curative findings There were only two unsuccessful cases, both treated with the neomycin preparation It was interesting to note that two other patients had only fair results with the neomycin ointment, but following substitution of the streptomycin complex had complete clearing of infection

After being convinced that weeping allergic lesions responded satisfactorily to the heparin antibiotic complexes, it was determined to ascertain whether other allergic states more difficult to observe would similarly show curative effects Heparin,* therefore, was tried in cases of acute pollinosis Amounts varying from 100 mg to 300 mg, depending upon the size of the patient, were injected intravenously into eight patients during acute attacks of hay fever (see Table II)

Table II illustrates ten cases of patients with pollinosis selected for treatment Four of them in addition to the pollinosis, had sinusitis—one with polyp formation The last patient had a catarrhal otitis media Symptoms of nasal discharge, nasal obstruction and headache were relieved within ten minutes after administration of the heparin In the patient with polyps, after acute symptoms had subsided, cultures were taken of the exudate from the sinuses The appropriate antibiotic was selected and given in combination with prednisolone This treatment was ineffective When heparin (100 mgs i m /day) was added and the antibiotic and steroid were continued, the polyps began to shrink, and at the end of a four-week period had substantially disappeared The infection

TABLE II

Hay Fever

No ases	Sex M	Sex F	Polinosis	Polinosis and Sinusitis	Polinosis + Catarrhalotitis Medi
10	4	6	5	4 (1 w/polyps)	1

Results

Nasal Discharge 1	2	3	Nasal Obstruction 1	2	3	Headache 1	2	3	Conjunctivitis 1	2	3	Breathing Eased 1	2	3	Redness an Itching 1	2	3
0	4	3	0	1	8	0	2	2	0	1	5	0	2	4	0	0	7

1 Results graded 1—poor 2—medium 3—excellent responses

2 Sinusitis had failed on antibiotic and steroid With 100/mg heparin added ever ther day sinuses were clear in a week no discharge after four weeks Polps shran n one case Catarrhal otitis media cleared with 200 mg heparin i v for four days hen no longer response of ear

3 Treated with 150-300 mgs heparin i v and given appropriate antibiotic and steroid Two patients received 150 mgs i v one patient 175 mgs i v five patients were give 200 mgs i v one 300 mgs i v and one patient was given 100 mgs i m every da for four weeks

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*Supplied by Dr Samuel Anderson Abbott Laboratories and Mr Leon Freeman of Darwin Laboratories subsided, so that no further pus could be obtained even after shrinking the nose

The patient with catarrhal otitis media was treated with 200 mg of heparin intravenously on four separate occasions In each case the ear would clear, only to refill slowly at first, after several treatments it filled more rapidly After the fifth treatment heparin was no longer successful in eliminating the fluid It was felt that higher doses of heparin might result in too great an anti-coagulant effect and leaving fluid in the ear might result in permanent impairment of hearing The older treatment of mechanical removal was employed alone as it was deemed possible that heparin treatment could increase bleeding at the site of incision

In three of the cases, placebos were given at various times instead of the heparin In no case did the patient's symptoms respond to the placebo, but when followed by heparin at periods of about 15 minutes, relief of symptoms occurred promptly One patient who had a sensitivity to beef protein, developed hives after using the heparin on two occasions She had had relief of her hay fever with the heparin, but we were unable to continue its use because of the allergy, either to the heparin or to the tissue from which it was extracted

Finally, 12 patients with acute recurrent asthmatic attacks were selected for trial In one, 100 mg was given subcutaneously In the remainder, 150 to 300 mg was given intravenously, depending upon the size of the patient There was marked, almost immediate relief of the respiratory distress (see Table III)

Table III is an analysis of the attempts to relieve the attacks of acute asthma Ten patients were included in this group The dosage again varied from 150 to 300 mg of heparin given intravenously at the time of an acute attack of asthma In nine of the patients there was immediate relief from wheezing, cough, and on difficulty in breathing There was a decrease in the panic which affected six patients It is interesting to note that eight of the patients commented spontaneously of a feeling of warmth in their chests The hay fever victims, when given the heparin, commented on the sense of warmth in their heads

In one patient there was a failure of heparin to secure a remission of the acute attack The asthma in this patient is believed to be on a neuropsychiatric basis, as detailed hospital studies revealed no true lung pathology After heparin had been given, wheezing could not be induced by the cough, which usually precipitated an attack If, however, he once precipitated the attack and started coughing and wheezing, the heparin had no effect on his asthma It was felt that the attacks were initiated by mental stimulation, since asking him questions on problems he did not choose to answer were avoided by coughing and wheezing

TABLE III

Acute Asthma

No Cases	Sex M	Sex F	Treatment mgs Heparin IV 150	200	250	300
10	4	6	1	4	3	2

Results

Wheezing 1	2	3	Cough 1	2	3	Breathing Eased 1	2	3	Panic Decrease* 1	2	3	Feeling Warmth† 1	2	3
1	3	6	1	5	4	1	3	6	1	4	2		2	6

*Three patients did not comment
†One patient did not comment

The action of the heparin is felt to be almost immediate As in the cases of experimental animals, heparin counteracted the histamine released in patients having acute asthmatic attacks Other drugs which were inadequate alone were then able to maintain an asymptomatic patient This was especially true of a mixture of antibiotics and steroids in infections, and of the steroids alone in the allergic conditions

One of the authors, being asthmatic, injected himself with 200 mg of heparin intravenously during an acute attack Almost immediately there was a sensation of warmth starting at the site of injection and spreading through the body, but centering chiefly in the chest There was a tremendous feeling of relaxation and in several moments the breathing eased off The sense of panic, which asthma seems always to induce, disappeared and was followed by a markedly relaxed state with normal breathing The sense of warm relaxation without wheezing was the outstanding feeling left by the heparin

Quantitative evidence for the action of heparin on relief of acute asthma has been obtained by Dr Jerome Cohn[15] Preliminary investigation has shown that heparin almost immediately decreases bronchiolar resistance to air flow in acute asthma resulting in increased vital capacity in six patients These investigations are being continued and will be reported in detail later

DISCUSSION

Essentially, the difference between local inflammation and stress is quantitative Within a volume of local inflamed tissue cellular destruction is limited to the area, whereas in stress there is a generalized cell destruction with a great release of the same cytotoxins Injury to one type of cell may affect the function of neighboring cells through the inflammatory response As cells are destroyed, they in turn, release cytotoxins which bring about the destruction of the adjacent cells, so that a chain reaction of cellular destruction occurs[9]

These altered states of the tissues, despite their protective effects, can be termed "intoxication," since it is inimical to the continuance of normal function of the tissue It is well recognized that histamine and serotonin are markedly inflaming substances They are contained both intra-cellularly and extracellularly[10] A hypothesis has been proposed elsewhere[10] that histamine or other amines having similar cytotoxic effects, are released from the cell upon injury They, therefore, accumulate in increased amounts in extracellular tissue As this increase takes place, more toxic amines are available to attack adjacent cells, and a chain reaction of cellular destruction occurs

Whatever the theoretical possibilities may be, it is known that histamine is released in various allergic states, including local and generalized anaphylaxis Thus, endogenous release of histamine occurs following chemical or bacterial poisoning thermal and traumatic wounds, and may be released from cells upon antigen antibody union Despite the arguments and conflicting data concerning the participation of histamine in physiological states, there seems to be relatively little debate that histamine, serotonin and other amines participate and actually initiate inflammation, particularly the inflammation induced in allergy

If the chain reaction of cell destruction was permitted to continue undisturbed, the organism would always die There are three main mechanisms known at present which prevent the lethal accumulation of cytotoxin The first is excretion of histamine from its site of release and from the body[11] The second is the action of the steroids and the third, local detoxification Since the stimulus for histamine release is almost always endogenous, the tissues must be frequently exposed to toxic amines A local mechanism must exist to buffer or destroy the toxic amines interstitially The earliest changes seen in loose connective tissue response to the toxic amines is the destruction of mast cells This is followed by the granules being picked up by the fibroblasts and digested[1] Heparin as well as histamine, is found in these granules[5] The capacity of heparin to complex with histamine and thus inhibit the cytotoxic effect of this substance may be a detoxifying mechanism

It may be seen, then, that any means by which the histamine concentration of the tissue would be reduced would thereby reduce the inflammatory potential, providing the stimulus for the production of the inflammation were held constant The less the histamine available for release in the tissue, the less the degree of inflammation Heparin, in this way, acts as an anti-inflammatory substance It appears that the anti-inflammatory effect of heparin is not so much concerned with the immediate catabolism of histamine, but rather with the capacity of heparin to complex with this base Not only does heparin complex histamine, but probably other amines such as serotonin[12]

It may be that heparin is somehow related to the eventual disposition or catabolism of histamine, but this does not account for its immediate detoxifying effect The anti-inflammatory role of heparin is thus in marked contrast to that of the steroids, in that adrenal cortical steroids apparently do not inhibit the release of histamine upon the initiating stimulus[13] or bring about its eventual catabolism They probably stop the continuous addition of histamine to the extracellular pool by preventing the toxic effect of histamine on the cell and thereby they inhibit the chain reaction of histamine release from destroyed cells In this respect, it is interesting to note that in some allergic states heparin actually possesses a more marked anti-inflammatory effect on certain allergic induced inflammations than that possessed by the adrenal cortical steroids

Heparin provides protection against endogenous histamine released by basic substances such as polymyxin B or other alkaline antibiotics, 48/80, and snake venom If these substances are administered to mice in lethal doses, it has been found that the lethality can be prevented either before or after injection of the histamine releaser by administration of heparin [11] A movie has been made in our laboratory which illustrates these actions of heparin [11] Heparin acts in respect to inhibiting the lethal consequences of endogenously released histamine in a linear dose response fashion[2], thus, there is a stoichiometric relationship existing between the concentration of the histamine releaser and the protective agent heparin

Studies performed in our laboratories indicate that the protective role of heparin is primarily due to the sulphuric acid substitution of this polymer [1] Other sulphated carbohydrate polymers such as chondroitin sulphate, agar agar and various synthetic suphated polysaccharides, will also provide protective effect against histamine release [15] It is thus apparent that heparin and other sulphated polysaccharides act as biological ion exchangers and essentially follow the law of mass action

It is of significance that although there may be a relation between the anti-inflammatory effects of the steroid hormones and heparin, it is very apparent that the mechanisms of anti-inflammatory action are entirely different

CONCLUSIONS

1 Laboratory work showed that heparin and other sulphuric acid substituted polysaccharides possess the ability to neutralize histamine by acting as ion exchangers following the law of mass action, by the formation of a heparin histamine complex *2* Studies of the effect of heparin added to previously unsuccessfully tried steroids and antibiotics helped clear infected external otitis, with and without weeping eczema *3* Selected cases of acute hay fever and asthma were helped by the intravenous administration of heparin As the acute attack subsided, more permanent relief was established by the classical methods of treatment *4* There was sufficient clinical improvement to warrant further studies to check on the hypothesis that sulfated polysaccharides, especially heparin, can provide protection against endogenous histamine release, suggesting a basic protection at least as important as that of anti-coagulation *5*. Combinations of alkaline antibiotics and heparin are valuable agents since the complex possesses the antibiotic activity and as antibiotic is released from the complex, heparin, in turn, is available to complex histamine and thereby prevents the noxious effects of this substance

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GILL MEMORIAL EYE, EAR AND THROAT HOSPITAL

The Gill Memorial Eye, Ear and Throat Hospital has just completed its Thirty-Third Annual Spring Congress in Ophthalmology and Otolaryngology and allied specialties The attendance was one of the largest in the history of the school with an attendance of 300 doctors and their wives There were 40 states, including Canada, represented There was a total of 18 guest speakers and there were 50 lectures during the five-and-a-half days of the Spring Congress In 1961, the Thirty-Fourth Annual Spring Congress will be held from April 9 through April 14

VASODILATOR THERAPY IN SENSORY-NEURAL HEARING LOSS*†‡

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Individuals developing an obscure sudden unilateral or bilateral sensory-neural (perceptive) hearing loss have been the object of much medical treatment for a number of years. This treatment has been based on the assumption that the loss of hearing was due to a vascular accident in the inner ear. Very little information has appeared in the literature as to the results of this treatment. One purpose of this paper is to present the findings in a group of patients with sudden hearing loss of the sensory-neural type and to show their response to an intensive vasodilator regime.

Some otologists[1,2,3a,3b] have stated that treatment is usually ineffective in this group of patients. Others[3c] feel that treatment, to be effective, *must* be initiated within a few hours of the onset of the hearing loss. A second purpose of this paper is to point out that treatment *does* seem to be effective in a significant percentage of these individuals. Although early treatment is undoubtedly more effective, treatment instituted after five or six weeks may still result in the return of serviceable hearing.

MECHANISM OF HEARING LOSS

Many theories have been advanced as to the cause of sudden neural or end organ deafness of obscure origin. Some otologists[4-6] have felt that the etiology was a viral infection or a neuritis. The majority,[3c,7,8,9,10,11] however, believe that sudden loss is due to a disturbance of the vascular bed in the

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†From the Otologic Medical Group and the Department of Otolaryngology, University of Southern California School of Medicine.

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inner ear Such disturbances include sludging of blood, hemorrhage, thrombosis, arteritis, embolism, spasm, edema, and combinations of these If due to hemorrhage or thrombosis, then very little can be expected in the way of recovery If due to spasm and edema, however, early treatment should be effective Unfortunately, a differential diagnosis between these two types cannot be made at present Treatment is, therefore, advocated on the assumption that the sudden loss is due to vascular spasm

The mechanism of spasm appears to be autonomic dysfunction as described by Williams[10,12] and Hilger[11] There is a localized area of arteriolar spasm associated with dilatation of the capillaries and venules Anoxia of the capillary loop then develops This anoxia results in injury to the endothelium, sludging of blood, release of toxic substances and increased permeability of the capillary loop A local disturbance of the electrolyte and fluid balance develops, resulting in endolymphatic (and perilymphatic?) hydrops or hypertension The autonomic nerve supply of the end-organ is not clearly understood, but clinical experience indicates that in most cases the spasm seems to be of cholinergic origin[13]

THERAPY

Treatment is based on the assumption that autonomic dysfunction and the resultant vascular bed disturbance is the cause of the hearing loss and is reversible There are three factors that must be considered in each case the underlying or precipitating factor, the neurovascular dysfunction, and the edema

Underlying or Precipitating Factors Stress, fatigue, emotional disturbances, allergy, endocrine imbalance—one or all of these have been considered to be of importance as underlying or as precipitating factors[8,9,10,11] Recently Shambaugh[14] reported a number of cases that had been unresponsive to vasodilator therapy, but that responded well to thyroid medication In the author's experience there has often been an episode of severe fatigue or emotional disturbance just prior to the onset of a sudden hearing loss If the sudden loss is of a fluctuating character, this fluctuation can frequently be related to repeated emotional episodes

Neurovascular Dysfunction In sudden hearing loss, specific drug therapy is directed primarily towards the neurovascular problem Hilger,[11] in an excellent paper on inner ear therapy, divided drugs affecting the neurovascular tone into two groups the autonomic blocking agents and the smooth muscle dilators The autonomic blocking agents that have been used are banthine, procaine, atropine, priscoline and ergots Smooth muscle dilators include nicotinic acid, histamine, nitrites and papaverine Various combinations of these drugs have been used by most otologists Some[15 16] have induced stellate ganglion block with procaine and, if favorable response is obtained, have recommended upper dorsal sympathectomy as definitive treatment

Edema To relieve the edema due to vasospasm, a low salt diet, at times with a diuretic, has been recommended Immediate fenestration of the horizontal semicircular canal to decompress the labyrinth has been advocated by some [17]

VASODILATOR REGIME

A standard vasodilator therapy has been used in all of the 247 patients with a sensory-neural hearing loss in this series The regime is also used in cases of Ménière's disease and recurrent spontaneous or postural vertigo

Histamine Intravenous histamine is given on three consecutive days This solution* contains 2 75 mgs of histamine phosphate in 250 cc of normal saline The solution is administered at an initial rate of 20 to 30 drops per minute, and if well tolerated, this may be increased to 50 to 60 drops per minute after five minutes The time of administration is 90 minutes in the average case The patient is made aware that a mild flushing, headache, and increased head noise may develop Following completion of this iv series, 0 1 cc of 1 100,000 dilution of histamine phosphate is administered subcutaneously twice weekly The patient is instructed to take two drops of 1 10,000 histamine phosphate sublingually twice daily

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*Obtained from Don Baxter Incorporated Glendale Calif

Nicotinic Acid Nicotinic acid tablets (50 mgs) are prescribed 30 to 45 minutes before breakfast and before dinner The dose is adjusted by the patient up to 200 or 300 mgs until a definite flush is obtained Frequently a flush is obtained by a smaller dose before breakfast than that required before dinner It is important that this be administered on an empty stomach to insure rapid absorption and maximum effect If tolerance develops, the dose may be increased, or Roniacol may be substituted Arlidin or Vasodilin has also been substituted on occasions

Diet A 2 Gm salt diet of 1,800 calories is recommended Coffee is not allowed because of the vasoconstrictor action of caffeine

Sedation An "autonomic stabilizer" such as Donnatol, Probanthine with Dartal, or Bellergal is frequently recommended A tranquilizer or ataraxic may also be indicated

Miscellaneous Vitamin C, usually in the form of C V P , is prescribed in doses of 300 mgs daily Merazine is advised for vertigo if indicated Cytamel or thyroid is administered if hypothyroidism is present

SUDDEN SENSORY-NEURAL HEARING LOSSES

In this report sudden sensory-neural hearing losses have included all cases in which a hearing impairment of an inner ear or neural type has developed in a period of one week or less in an otherwise healthy individual who has not received ototoxic drugs recently The majority of losses in this series developed in a matter of hours The only cases excluded are those that have fluctuated following the development of the sudden loss These cases will be considered later

This group comprises 223 patients, the majority of whom were seen in the last five years In the last two years, approximately 2 per cent of our new patients have had this diagnosis made Thirteen patients developed a loss in both ears Nine of these had a simultaneous bilateral loss and four developed a loss in the second ear from three months to 20 years following the first episode

Age and Sex Distribution The youngest patient in this series was 15 years old and the oldest was 84 Table I shows that there was a fairly even distribution of patients in the fourth, fifth, sixth, and seventh decades Seventy-five per cent of the patients were over the age of 40 There were 35 per cent more females than males

Type and Extent of Hearing Loss Hearing losses were classified into four groups low tone, flat, high tone, and

TABLE I

Sudden Sensory Neural Hearing Loss

Age and Sex Distribution (N-223)

Age	Number	Per Cent		
10 19	5	2		
20 29	13	6		
30 39	39	17		
40 49	58	26	52 Per Cent	75 Per Cent over 40
50 59	57	26		
60 69	41	18		
70—	10	5		
Sex—				
Female	129	58		
Male	94	42		

total hearing loss A hearing loss was considered to be a *low tone* if normal hearing was maintained at 4,000 or 8,000 c/s, or if there was a low tone tilt to the audiogram with a difference of 25 decibels between the frequencies 500 and 4,000 c/s The hearing loss was considered to be low tone even if there was involvement of the high tones, if this high tone loss was also present in the other (normal) ear The hearing loss was classified as *high tone* if normal hearing was maintained at 250 or 500 c/s or if there was a high tone tilt to the audiogram with a difference of 25 decibels between the frequencies 500 and 4,000 c/s Any loss not falling into one of these two categories was considered to be a *flat loss* as long as the average loss in the speech hearing frequencies (500 through 2,000 c/s) did not exceed 90 to 95 decibels If the loss exceeded this, it was considered to be a *total loss* of hearing

A flat type hearing loss occurred in 41 per cent of the 236 ears (see Table II) The next most common type was the high tone loss Over 70 per cent of the group had an average hearing level or 50 db or more in the three speech hearing frequencies

TABLE II

Sudden Sensory Neural Hearing Loss

Type and Extent of Loss (N 236 Ears)

			Hearing Level (db) (500 2,000 c/s)			
Type Loss	Number	Per Cent	15-25 Per Cent	30-45 Per Cent	50-75 Per Cent	80+ Per Cent
Low Tone	39	17	13	41	46	
Flat	98	41	6	32	45	17
High Tone	69	29	1	17	65	17
Total	30	13				100

TABLE III

Sudden Sensory Neural Hearing Loss

Tinnitus (N 223)

Present	86 Per Cent			
Type Specified—78 Cases				
Buzz		26	33 Per Cent	90 Per Cent Low Tone
Roar		23	29 Per Cent	
Low (Below 512 c/s)		16	21 Per Cent	
Rumble		3		
Hum		2		
Ring Steam Hiss		8		

Tinnitus Eighty-six per cent of the patients complained of tinnitus (see Table III) This was usually a low tone tinnitus, the two most common types being buzzing and roaring This may be compared to a study[15] of normal and hard-of-hearing individuals where low tone tinnitus was present in less than 40 per cent of those complaining of tinnitus

Vertigo Vertigo was present in 37 per cent of this group (see Table IV), and was most frequently of the induced or postural type It occurred least frequently in those individuals with a low tone type of impairment and most frequently in the group with flat and total hearing losses Vertigo was more than twice as common in the females

When spontaneous vertigo was present, it invariably occurred at the onset of the sudden hearing loss In no case did the spontaneous vertigo have its onset after 48 hours and only in an occasional individual was there spontaneous vertigo prior to the onset of the loss Although some had only a brief episode of spontaneous vertigo, the dizziness usually lasted from a few hours to three days, often requiring confinement to bed

TABLE IV

Sudden Sensory-Neural Hearing Loss

Vertigo (N-223)

Present	83	37 Per Cent
Type—		
Spontaneous		45 Per Cent
Induced		61 Per Cent
Occurrence of Vertigo Re Hearing Loss—		
Low Tone		20 Per Cent
Flat		44 Per Cent
High Tone		32 Per Cent
Total		47 Per Cent

Induced vertigo, when present, was not usually associated with an initial episode of spontaneous vertigo As opposed to the spontaneous vertigo, the postural vertigo at times had its onset three or four days after the onset of hearing loss and often persisted for periods of weeks or months In many cases it was the persistence of this postural vertigo that led the patient to seek medical advice five or six months following the onset of the hearing loss

Vestibular function tests were performed on 20 patients Thirteen of these had normal responses and of this group, 11 had losses greater than 50 decibels Of the four who had no vestibular response, all had losses in excess of 75 decibels

Radiographic Findings Approximately one-third of the patients had radiographic examinations of the temporal bone Of 92 patients seen in 1958 and 1959, 17 were X-rayed by Eugene Compere, M D One of these (a case of clinical otosclerosis) had diffuse otic capsule otosclerosis. Nine patients had petrosal sclerosis, and seven showed a normal radiographic picture. A large percentage of individuals with progressive *hereditary* types of sensory-neural hearing loss show petrosal sclerosis, and this may also be true in the sudden losses. At the present time, X-rays are being obtained in most cases of hereditary and sudden sensory-neural hearing losses to investigate this finding further.

Summary. Sudden non-fluctuating sensory-neural hearing losses in otherwise healthy individuals usually occur over the age of 40, and are more common in the female. The loss often involves all of the frequencies resulting in a flat or high tone type of curve with an average loss in the speech hearing frequencies of 50 decibels or more. A low tone tinnitus is the rule, and vertigo is present in about one-third of the cases.

TABLE V

Sudden Sensory Neural Hearing Loss

Treated	Onset of Treatment Re Recovery Number	Recovered	Per Cent
Total	70	24	34
Within Four Days	21	13	62
Within Two Weeks	38	20	53
Within Six Weeks	49	23	47
After Six Weeks	21	1	5

TREATMENT RESULTS—SUDDEN LOSSES

A vasodilator regime was prescribed for 66 patients (70 ears). Seven were treated one year or more following the onset of a sudden loss. Five of these were treated because of persistent postural vertigo and annoying tinnitus. In all cases the response to treatment was satisfactory although there was no change in the hearing level. In the other two cases (15 and 20 years following the hearing loss) treatment was instituted in connection with a second ear that had become involved by a sudden hearing loss recently.

In the following paragraphs and accompanying tables an ear was considered to have recovered if the hearing returned to a 15 decibel level or better, if the hearing returned to the level of the opposite ear, or (in a few cases with severe hearing loss) if the hearing returned to a serviceable level

Twenty-four of the 70 treated cases recovered (see Table V) Of those treated within four days of the onset of loss, 62 per cent recovered Forty-seven per cent of those treated within six weeks recovered, but only one case treated after six weeks returned to normal Details of the recovered cases are shown in Table VI

Age and Sex Distribution Table VII shows the age and sex distribution of individuals treated within six weeks who recovered Fifty-five per cent of those between the age of 30 and 59 recovered Under 30 and over 60 the results were poor There was no significant difference between the recovery rate in females and males

Type and Extent of Hearing Loss (See Table VIII) In the group of cases treated within six weeks, the distribution relative to type and extent of loss is approximately the same as that in the 223 cases There was no apparent selective factor from this standpoint Of 12 individuals with a low tone loss, ten recovered The flat and high tone losses showed a much lower recovery rate, and the total loss cases showed the poorest results Eleven of 16 cases having a loss of 45 decibels or less recovered Favorable factors appear to be the presence of a tow tone loss and an impairment of 45 db or less

Tinnitus There was no apparent relationship between the presence or character of the tinnitus and the incidence of recovery

Vertigo Vertigo was present in a third of the 223 patients This same incidence was present in the treated group and in the group of cases treated within six weeks of the onset of their hearing loss There was no apparent selective factor from the standpoint of vertigo Despite this lack of selection, only two cases of 24 (8 per cent) that recovered had vertigo at any time during the course of their illness (see Table IX) The absence of vertigo appears to be favorable for recovery

Recovery Time Eighteen cases were treated within one

TABLE VI

No Case	Age	Sex	Hearing Loss				Tinnitus	Vertigo	Therapy		
			Curve	Level (Avg 500-2 000)	Onset	Fluctuation			Onset	Type	Response
2	32	F	LT	25	S	—	+	—	1 day	+	Normal hearing 3 weeks, maintained 3 years
45	47	F	F	60	S	—	+	I	1 week	+	Level of other ear (20 db) 1 month, maintained at 1 year
46	43	F	LT	40	S	—	—	—	1 week	+	15 db at 2 months, 5 db at 2 years
47	51	F	LT	35	W	—	+	—	1 month	M	13 db at 2 months, 25 db at 1 year No change 3 years
71	64	F	LT	35	S	—	+	—	6 weeks	+	Normal at 4 months Maintained at 1 year
88	34	M	F	65	S	—	Buzz	—	2 days	+	7 db at 9 days
89	49	M	HT	75	S	—	Buzz	—	1 week	+	Normal at 2 weeks
99	38	M	LT	70	S	—	Hum	—	3 days	+	Normal 1 week, maintained 1 year
110	41	M	F	60	S	—	Buzz	—	1 day	+	Normal 4 days maintained 1½ years
122	44	F	F	80	S	—	—	—	4 days	+	Level of other ear (10 db) at 1 month
129	69	M	F	90	S	—	Roar	—	5 days	+	12 db at 7 days maintained 1 year

134	35	M	LT	15	S	—	+	—	3 days	+	Minus 5 db in 1 week
140	50	M	F	45	S	—	Roar and Buzz		2 weeks	+	Normal 10 days, maintained 1 year
164	46	M	HT	30	S	—	Steam	S	2 weeks	+	15 db at 1 month, 8 db at 4 months
169	35	F	HT	75	S	—	—	—	2 days	+	Normal at 1 month
175	31	M	F	60	S	—	+	—	6 months	+	35 db at 1½ months, normal 4 months
183	47	M	T	—	S	—	+	—	20 years	+	No response
			F	45	S	—	+	—	5 weeks	+	27 db at 4 months, maintained 2 years
193	59	F	F	70	S	—	+	—	1 week	M	Normal at 2 weeks
203	43	M	LT	70	S	—	Steam	—	2 days	+	22 db at 6 weeks, 7 db at 6 months, maintained 2 years
213	49	M	LT	70	D	—	+	—	4 days	+	Normal at 2 weeks
219	40	F	HT	10	S	—	Ring	—	1 day	+	"Normal" at 1 week
221	53	F	LT	30	S	—	Low	—	1 day	+	Normal at 1 month
224	57	M	T	—	S	—	Buzz	—	3 days	+	28 db at 2 weeks (On Dicumerol and Priscoline at time of loss)
228	54	F	LT	30	S	—	—	—	1 day	+	Normal 1 day Maintained 3 months
			F	30	S	—	—	—	4 years	+	No change

Case No	Age	Sex	Hearing Loss				Tinnitus	Vertigo	Therapy		
			Curve	Level (Avg 500-2000)	Onset	Fluctuation			Onset	Type	Response
1	39	M	LT	40	G	—	Buzz	—	6 months	+	12 db at 1 month, maintained
			LT	40	G	—	Buzz	—	6 months	+	22 db at 1 month, then fluctuated
9	55	M	LT	40	G	+	Low	—	8 months	+	Normal at 2 months, then fluctuation
			LT	45	D	—	Low	I	1 week	+	Normal at 2 weeks, maintained 1 year
15	57	M	FT	20	D	+	+	—	5 weeks	+	Normal at 3 days, fluctuated for months
16	58	M	FT	45	S	+	Low	—	2 weeks	+	Fluctuated to normal with treatment
18	51	F	LT	30	G	—	Buzz	—	1 year	+	Normal at 2 months, maintained at 8 months
19	54	M	LT	45	S	+	+	—	2 months	+	11 db at 1 month
20	71	F	LT+HT	15	G	+	Roar	—	3 months	M	Normal at 3 weeks
26	13	F	T	—	S	+	Buzz	—	1 week	+	Normal after treatment See Summary

Curve LT—Low Tone FT—Flat Loss HT—High Tone T—Total Loss
Onset S—Sudden D—1 day W—1 week or less G—Gradual
Vertigo I—Induced S—Spontaneous
Therapy +—Full Vasodilator Regime M—Modified Regime (Minus IV Histamine)

week of the onset of a sudden hearing loss. Six of these cases recovered within a week, and these were the only cases that recovered within one week of onset of therapy. Other than this, there seems to be no definite correlation between the time of onset of therapy and the speed of recovery. Eighty-five per cent of the group that did recover their hearing, did so within a period of six weeks from the onset of therapy (see Table X). Those who had not recovered by six weeks had shown evidence of response to treatment: partial hearing improvement, fluctuation in hearing, or marked fluctuation in tinnitus.

TABLE VII

Sudden Sensory-Neural Hearing Loss
Cases Treated Within Six Weeks (N-49)

Age and Sex Distribution	Recovered	Per Cent	
Age—			
Under 30	0(3)	0	
30-39	5(10)	50	21(38)—55 Per Cent
40-49	10(15)	67	
50-59	6(13)	46	
Over 60	2(8)	20	
Sex—			
Female	11(24)	46	
Male	13(25)	52	

TABLE VIII

Sudden Sensory Neural Hearing Loss
Cases Treated Within Six Weeks (N 49)

Type and Extent of Loss	Recovered	Per Cent
Type Loss—		
Low Tone	10 (12)	83
Flat Loss	8 (19)	42
High Tone	4 (10)	40
Total Loss	1 (8)	12
Extent Loss (500-2,000 c/s)—		
15-45 db	11 (16)	69
50-75 db	9 (18)	50
80 db—	3 (15)	20

Treatment After Six Weeks. Cases treated six weeks or more following the onset of a sudden hearing loss, but within one year of the loss, were analyzed to see if there was some factor other than time that could be related to the lack of response Analyzing this group from the standpoint of age, type and extent of loss and incidence of vertigo revealed no significant difference from the group of cases treated under six weeks or from the overall group of treated cases It would seem that time was the major factor in the failure of these cases to respond to therapy

Summary Intensive vasodilator therapy resulted in recovery of hearing in approximately 50 per cent of the cases of sudden non-fluctuating sensory-neural hearing losses treated within six weeks of the onset of the loss It appears that time is a major factor in the failure of cases treated later than six weeks to respond to treatment Low tone impairments respond more favorably than others, as do impairments of less than 50 decibels The presence of vertigo appears to be a poor prognostic sign

TABLE IX

Sudden Sensory Neural Hearing Loss

Response to Treatment Re Vertigo

Total Cases Treated Within Six Weeks	49	
With Vertigo	15	(31 Per Cent)
Number of Cases That Recovered	24	
With Vertigo	2	(8 Per Cent)

TABLE X

Sudden Sensory-Neural Hearing Loss
Recovered Cases (N 24)

Recovery Time	No Cases	
Less Than Two Weeks	11	85 Per Cent Within Six Weeks
Two to Four Weeks	7	
Four to Six Weeks	2	
Six Weeks to Four Months	4	

FLUCTUATING AND GRADUAL SENSORY-NEURAL HEARING LOSSES

This group consists of 24 patients, three of whom had bilateral hearing loss, two of these occurring simultaneously This group includes fluctuating sensory-neural hearing losses of sudden or gradual onset, and gradual non-fluctuating losses in which vasodilator therapy was used Most of these patients were treated within the last two years

Age and Sex Distribution Table XI demonstrates that the distribution of cases is similar to that seen in the sudden loss group Seventy-nine per cent of the individuals were over the age of 40 There was an equal distribution between the sexes

TABLE XI

Fluctuating and Gradual Sensory-Neural Hearing Losses

Age and Sex Distribution (N-24)

	No Cases	Per Cent	
Age—			
Under 30	1	4	
30-39	4	17	
40-49	2	8	79 Per Cent Over 40
50-59	10	42	
60 69	6	25	
70—	1	4	
Sex—			
Equal Distribution			

TABLE XII

Fluctuating and Gradual Sensory Neural Hearing Losses

Type and Extent of Loss (N-27 Ears)

Type Loss	No Cases	Per Cent	Hearing Level (db) (500-2 000 c/s) 15-25	30-45	50-75	80 —
Low Tone	23	85	9	11	3	–
Flat	2	7 5	–	–	2	–
High Tone	0		–	–	–	–
Total	2	7 5	–	–	–	2

Type and Extent of Loss Cases were grouped into four categories (low tone, flat, high tone total hearing loss), using the same criteria as for the sudden loss group Eighty-five per cent showed a low-tone type of hearing impairment (see Table XII) No high tone losses were observed The majority of cases had less than a 50 decibel hearing loss

Tinnitus Tinnitus was present in all but one case (see Table XIII), and was of the low tone type

Vertigo Seven patients (30 per cent) had had vertigo at one year of the loss, were analyzed to see if there was some factor other than time that could be related to the lack of response Analyzing this group from the standpoint of age, type and extent of loss and incidence of vertigo revealed no significant difference from the group of cases treated under six weeks or from the overall group of treated cases It would seem that time was the major factor in the failure of these cases to respond to therapy

Summary Intensive vasodilator therapy resulted in recovery of hearing in approximately 50 per cent of the cases

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of sudden non-fluctuating sensory-neural hearing losses treated within six weeks of the onset of the loss It appears that time is a major factor in the failure of cases treated later than six weeks to respond to treatment Low tone impairments respond more favorably than others, as do impairments of less than 50 decibels The presence of vertigo appears to be a poor prognostic sign

FLUCTUATING AND GRADUAL SENSORY-NEURAL HEARING LOSSES

This group consists of 24 patients, three of whom had bilateral hearing loss, two of these occurring simultaneously This group includes fluctuating sensory-neural hearing losses important factor Nine of 13 cases with low tone hearing loss recovered Details of the recovered cases are shown in Table VI

Age and Sex Distribution There are too few cases in this series to draw any conclusions, but it is interesting that five of six cases in the age group 50 to 59 recovered There were nine males, of whom five responded, and four females, of whom three responded

Vertigo Vertigo was present in two of the 13 individuals treated Of the eight who recovered, one had vertigo

TABLE XV

Fluctuating and Gradual Sensory Neural Hearing Losses
Recovered Cases (N-10)

Recovery Time and Duration of Loss	No	Per Cent
Recovery Time—		
Within Two Weeks	5	50
Within Four Weeks	9	90
Duration of Loss Before Treatment—		
One Year	1	
Eight Months	1	
Six Months	1	

Recovery Time Five of the ten ears that recovered did so within two weeks (see Table XV) Four more recovered during the following two weeks, and the final case had shown maximum response at two months It is interesting, particularly in comparison with the findings in cases of a sudden *non-fluctuating* loss, that one case responded after a loss had been present one year, one after eight months, and one after six months It appears that the time factor in treatment of fluctuating losses is not critical as long as the hearing continues to fluctuate

Summary Intensive vasodilator treatment resulted in recovery of hearing in approximately two-thirds of the cases of fluctuating and gradual sensory-neural hearing losses The length of time between onset of loss and onset of treatment was not a factor in this recovery some time during their illness In only one of these patients was the vertigo spontaneous, and this for a period of approximately five minutes at the onset of a sudden low tone hearing loss

Radiographic Findings Eight patients had X-ray examinations by Dr Compere One of these showed diffuse otic capsule otosclerosis Four patients had petrosal sclerosis, and in three the X-ray showed normal findings

TABLE XIII

Fluctuating and Gradual Sensory Neural Hearing Losses

Tinnitus (N 24)	
Present	23—95 Per Cent
Type Specified—16 Cases	
Buzz	6
Roar	5
Low Tone (Below 512 c/s)	5

Buzz, Roar and Low Tone (6, 5, 5): 100 Per Cent Low Tone

TABLE XIV

Fluctuating and Gradual Sensory Neural Hearing Losses
Treated Cases (N 15 Ears)

Recovery Rate and Type of Loss			
Recovered		No	Per Cent
Total		10 (15)	67
Treated Within Six Weeks		4 (7)	57
Type Loss	No	Recovered	Per Cent
Low Tone	13	9	69
Total Loss	2	1	50

Summary Fluctuating and gradual sensory-neural hearing losses occur most commonly over the age of 40 The loss usually involved the low tones more than the high tones, with an average loss in the speech hearing frequencies of less than 50 decibels A low tone tinnitus is the rule Spontaneous vertigo is uncommon

TREATMENT RESULTS—FLUCTUATING AND GRADUAL LOSSES

There were 13 patients (15 ears) who were placed on a vasodilator regime Of this number, ten ears recovered (see Table XIV) Time of onset of therapy did not seem to be an made three weeks after the initial examination demonstrated a 12 decibel hearing level (see Fig 2) Normal hearing was maintained during a one-year follow-up period

Case 20 A high and low tone loss developed simultaneously in this elderly patient Minimal therapy resulted in recovery of hearing and relief of tinnitus despite the fact that the symptoms had been present for three months prior to treatment

A 71-year-old woman was examined because of a roaring tinnitus in the left ear The tinnitus had developed following eye surgery some three months prior to this examination The tinnitus and stuffiness in her left ear fluctuated There was no vertigo

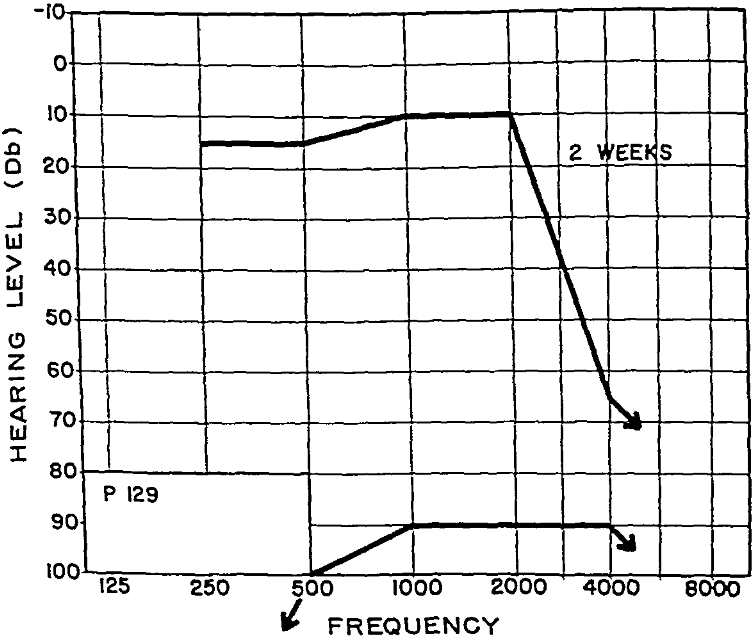

Fig 2 Case 129 Sudden Loss

Examination revealed a combined high and low tone hearing loss with normal hearing at 1 000 c/s Sublingual histamine, nicotinic acid, and Vitamin C were recommended Three weeks later examination revealed marked improvement in hearing and a remission of all symptoms (see Fig 3)

Case 210 A high tone dip may be reversible as demonstrated in this patient upon whom we had audiograms prior to the hearing loss

This 40 year old woman was first examined because of a seven year history of a gradually progressive hearing loss associated with tinnitus Examination revealed a conductive hearing impairment of 48 decibels in the right ear and 23 decibels in the left ear Right stapes mobilization improved the hearing to a 25 decibel level

CASE REPORTS

A summary of each of the 37 recovered cases is presented in Table VI. The following cases are presented in more detail to demonstrate certain interesting aspects of the history or treatment. The case numbers correspond to those in Table VI.

Case 110. This demonstrates optimum response in an individual treated within a few hours of onset of a sudden sensory-neural hearing loss.

A 41-year old physician developed buzzing in his right ear while performing emergency surgery one evening. The following morning he awakened with a severe hearing loss in that ear. There was no vertigo. Examination a few hours after the onset of the hearing loss revealed a 60 decibel level in the right ear and normal hearing in the left ear. A vasodilator regime was instituted and an examination four days later demonstrated normal hearing (see Fig 1). A year and one half later hearing tests showed normal hearing.

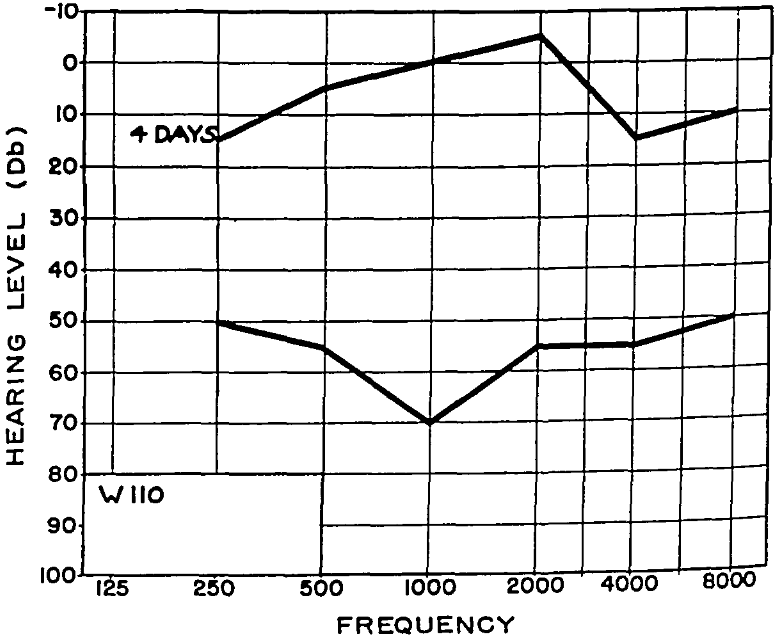

Fig 1. Case 110. Sudden Loss

Case 129. This demonstrates an excellent response in an elderly individual with a profound hearing loss of a flat type. Statistically he should have had very little chance of recovery.

A 69 year old man developed a full feeling in his left ear associated with roaring tinnitus and marked hearing loss five days prior to examination. There was no vertigo. Examination revealed normal hearing in the right ear and a 90 decibel hearing level in the left ear. A vasodilator regime was instituted. Within seven days the roaring noise had subsided, and he said his hearing had returned to normal. An audiogram made three weeks after the initial examination demonstrated a 12 decibel hearing level (see Fig 2) Normal hearing was maintained during a one-year follow up period

Case 20 A high and low tone loss developed simultaneously in this elderly patient Minimal therapy resulted in recovery of hearing and relief of tinnitus despite the fact that the symptoms had been present for three months prior to treatment

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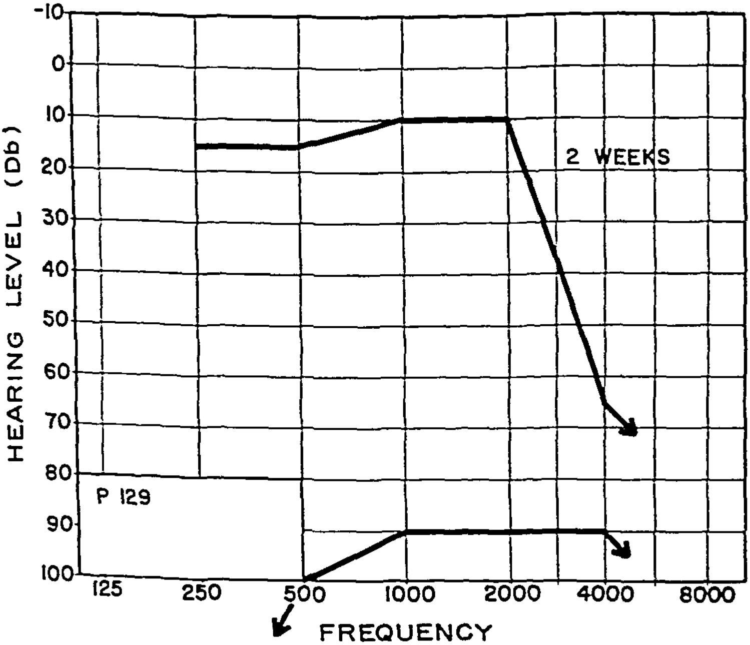

Fig 2 Case 129 Sudden Loss

Examination revealed a combined high and low tone hearing loss with normal hearing at 1,000 c/s Sublingual histamine, nicotinic acid, and Vitamin C were recommended Three weeks later examination revealed marked improvement in hearing and a remission of all symptoms (see Fig 3)

Case 219 A high tone dip may be reversible as demonstrated in this patient upon whom we had audiograms prior to the hearing loss

This 40 year-old woman was first examined because of a seven year history of a gradually progressive hearing loss associated with tinnitus Examination revealed a conductive hearing impairment of 48 decibels in the right ear and 23 decibels in the left ear Right stapes mobilization improved the hearing to a 25 decibel level

Two months following mobilization she developed a sudden ringing tinnitus and stuffiness in the *unoperated* ear An audiogram made at that time demonstrated a sharp tonal dip at 2,000 and 4,000 c/s in the left ear A vasodilator regime was instituted and a partial hearing improvement was noted immediately following the first intravenous histamine Her hearing had returned in one week (see Fig 4)

Case 1 A gradually progressive bilateral nonfluctuating hearing loss was present for six months The only key to the possibility that this might be of vascular origin and possibly reversible was the presence of a low pitched buzzing tinnitus

A 39 year old man stated that he had noticed a buzzing tinnitus in his left ear for six months before being seen and that his right ear became involved one month later During these six months his hearing had gradually decreased to a point where he had lost his job and purchased a hearing aid There was no history of ototoxic drug therapy and no vertigo

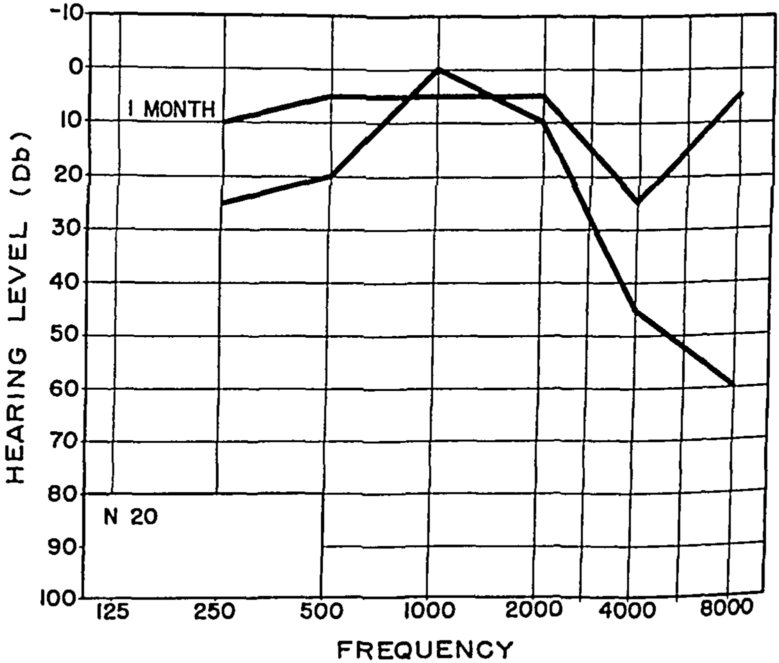

Fig 3 Case 20 Fluctuating High and Low Tone Loss

An audiogram demonstrated a 40 decibel hearing level in each ear A vasodilator regime was instituted During the next three or four weeks his hearing gradually returned Fluctuating tinnitus was noticed Audiometric examination after one month of therapy revealed a 12 decibel level in the right ear (see Fig 5) and a 22 decibel level in the left ear (see Fig 6) Therapy was continued and two months later the hearing level in the right ear was unchanged, the left ear fluctuated

Case 9 This patient had a bilateral hearing impairment gradual in one ear (eight months duration) and a recent sudden loss in the other ear The ear with the sudden loss responded immediately to treatment Because of the tinnitus and hearing level fluctuation in the other ear, treatment was continued with gradual improvement to a serviceable level

A 55 year-old man was seen regarding a hearing aid Eight months previously he had noted a gradual onset of fluctuating hearing loss in his left ear associated with low pitched tinnitus and stuffiness in the ear His hearing level had stabilized during the previous three or four months One week before examination he had noticed a stuffy sensation with roaring tinnitus in his right ear He had noticed mild unsteadiness for one day prior to his first visit Examination revealed a bilateral sensory neural hearing impairment of 40 decibels in the right ear and 45 decibels in the left ear

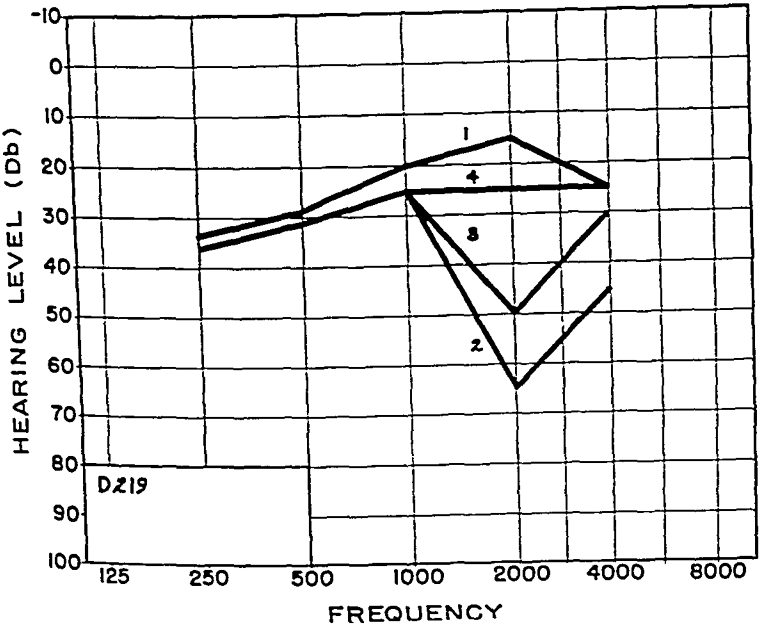

Fig 4 Case 219 Sudden Loss 1 Two months prior to loss, 2 one day after onset of loss 3 after first iv histamine 4 after one week of treatment.

A vasodilator regime was instituted The hearing in the most recently affected ear returned to a normal level within 12 days and this has been maintained for one year (see Fig 7) Hearing in his left ear improved only slightly and then regressed Because both the hearing level and the tinnitus in the left ear were fluctuating, treatment was continued An audiogram at two months revealed an 8 decibel level (see Fig 8) The hearing in this ear continues to fluctuate particularly if histamine therapy is omitted

Case 26. This is the youngest patient in the series. The case is presented because it shows repeated dramatic response to intravenous histamine therapy.

This girl was first seen in 1955 at the age of nine. A hearing loss had been discovered at the age of four and there had been no change since that time. An audiogram revealed residual hearing, only, at 256 and 512 c/s in the right ear. The left ear showed a mixed loss of 70 decibels, predominantly sensory-neural except in the low frequencies. A hearing aid was advised and accepted. She was seen intermittently during the following four years.

In April, 1959, (at the age of 13) she developed a sudden buzzing and hearing loss in her left ear. There was no dizziness. Examination one week later revealed an average hearing level of 100 decibels in the left ear.

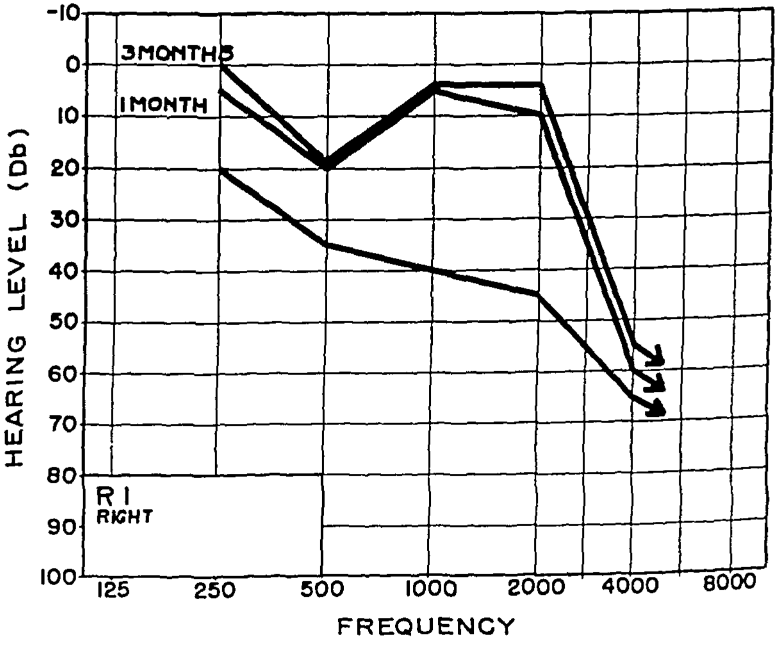

Fig. 5. Case 1. Gradual Loss Right Ear.

She was immediately given intravenous histamine. During the course of the first day her hearing returned to the point where she could use her hearing aid again. For the next two days, her hearing fluctuated slightly. Three weeks later an audiogram revealed a 55 decibel hearing level (see Fig. 9). Vasodilator therapy was continued.

During the following three months she had two further episodes of sudden total hearing loss. Each attack responded immediately to intravenous histamine. Her hearing has stabilized at a satisfactory level up to the present time (about five months).

DISCUSSION

Controls

One of the major difficulties in evaluating the results of treatment in this group of cases is the lack of a control series There is a natural hesitancy to withhold treatment when by so doing the patient may be left with a permanent severe sensory-neural hearing loss Spontaneous recovery has been

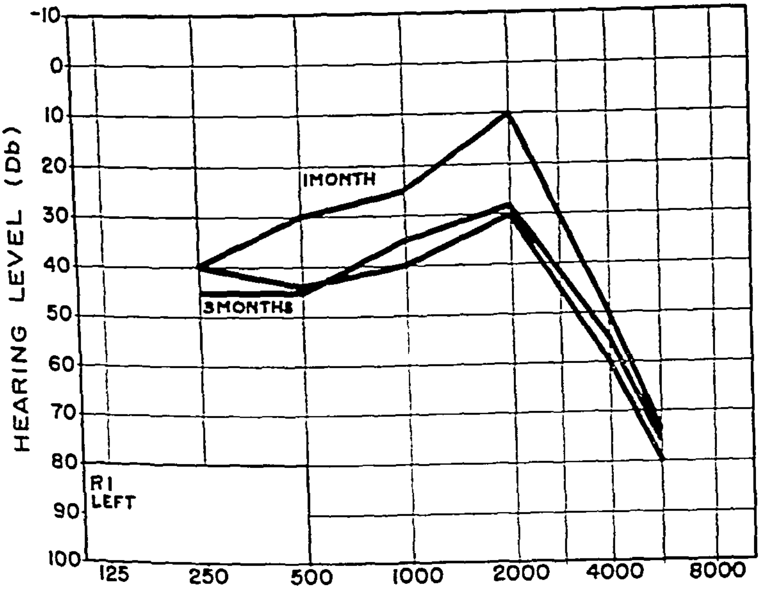

Fig 6 Case 1 Gradual Loss Left Ear

noted by some[3a 3c 9 10 19] and perhaps a small percentage of the individuals in this series would have recovered without treatment Certainly the pessimistic view taken by many otologists[1 2 3a-b] would indicate relatively little likelihood of recovery in the majority of cases, with or without treatment

A few reports have been found in which treatment was not instituted and adequate follow-up was obtained Perhaps these reports could serve as a control for this series Svane-Knudsen[4] reported 21 cases of sudden sensory-neural hearing loss Although a partial hearing improvement occurred in some cases, none showed return to normal hearing All of his cases had a total or high tone type loss, and the majority had vertigo, both of which factors are unfavorable for recovery according to the results in the present series He believed that a toxic or infectious neuronitis was the most likely cause in his cases

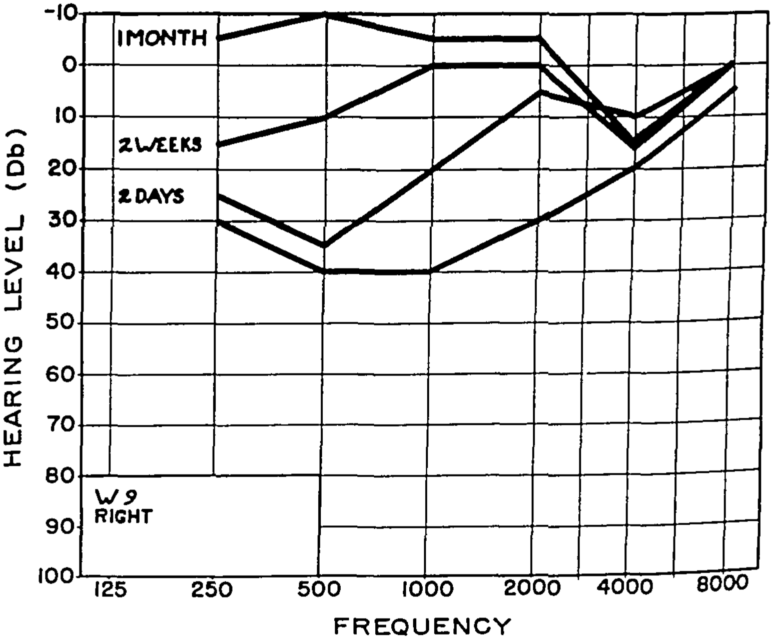

Fig 7 Sudden Fluctuating Loss Right Ear

Rasmussen stated that suddenly occurring unilateral deafness in otherwise normal individuals is a very rare phenomenon He observed 18 cases in 15 years Eleven of these had a profound deafness, and 15 had vertigo The hearing level in only one patient returned to normal

Error in Diagnosis

The majority of patients observed were not seen within six weeks of the onset of their hearing loss Many of these came for an examination a year or so later hoping that they were candidates for stapes mobilization Others were pri-mainly concerned about the persistence of tinnitus or postural vertigo and had waited three or four months following a sudden hearing loss before consulting an otologist A number of patients, however, were seen late because of an error in diagnosis This is unfortunate because it is realized that up to 50 per cent of these patients might have had normal hearing had a correct diagnosis been made and vasodilator treatment instituted early A mistaken diagnosis of a middle

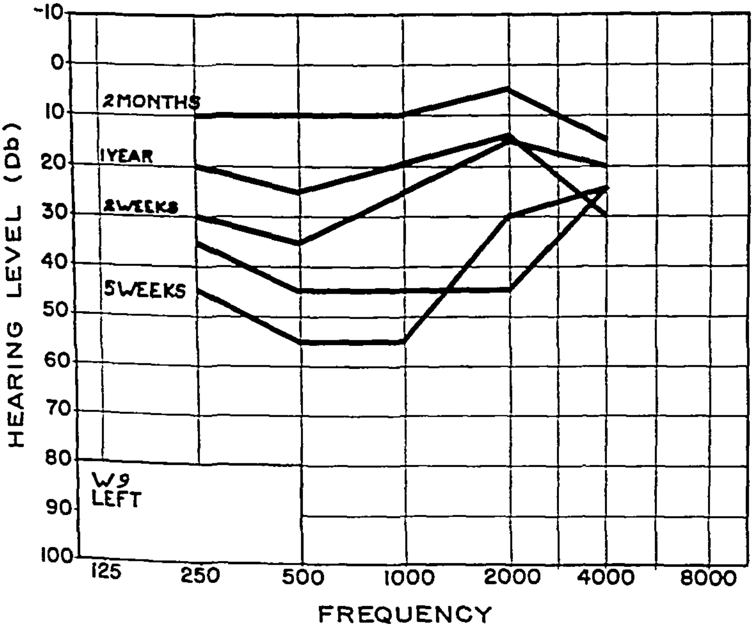

Fig 8 Case 9 Gradual Loss Left Ear

ear impairment may have been made on a basis of history alone, inasmuch as the subjective complaints are quite similar to those of Eustachian tube blockage There is a feeling of stuffiness, as though there were water in the ear, and often the patient will volunteer that this is "just like what happens when I come down from the mountains" The initial impression may be strengthened by the fact that a chronic vasomotor rhinitis may become more symptomatic at the time of the sudden sensory-neural hearing loss due to the fact that both conditions are probably manifestations of autonomic dysfunction Careful examination of the eardrum membrane, including use of the pneumatic otoscope, should suggest that the problem is not due to tubal blockage In those cases of serous otitis media where the membrane appears almost normal, there is usually marked limitation of motion as demonstrated by the Valsalva maneuver and the pneumatic otoscope An incorrect diagnosis of conductive hearing impairment may also result from an audiogram in which the opposite (normal) ear has been inadequately *masked*, resulting in a shadow bone curve on the affected side

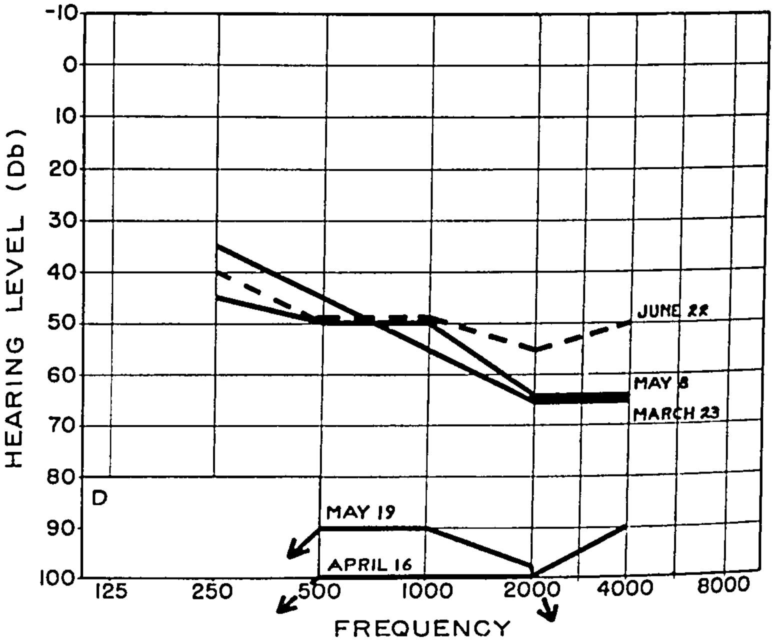

Fig 9 Case 26 Fluctuating Loss

Central Nervous System Disease

When a diagnosis of a sudden unilateral sensory-neural hearing loss has been made, it is important to rule out central nervous system disease At the initial visit the cranial nerves are carefully examined, paying particular attention to corneal and nasal vestibular sensation At the present time X-rays of the petrous pyramid are obtained routinely in all these cases If neurological and radiological examinations are negative, a vestibular test is deferred pending the outcome of vasodilator treatment If there is no response to treatment within six weeks, a vestibular test is then carried out Should this test reveal a dead labyrinth, a spinal fluid protein examination is obtained, even though neurological examination is negative If the patient is first examined two months or more after the onset of a sudden non-fluctuating loss, a vestibular test is performed at the initial examination, inasmuch as vasodilator therapy will in all probability not be instituted

Three cases of posterior fossa pathology presented as sudden sensory-neural hearing loss Two of these had acoustic neuromata, and in each case the neuroma was over 50 per cent cystic, apparently the result of a hemorrhage The other case had a thrombosis of a vessel supplying the brain stem with mild sensory and motor signs when first examined

Vasodilator Therapy

Vasodilator therapy should be advised for any individual with a sudden sensory-neural hearing loss of obscure origin seen within six to eight weeks of onset If seen after two months treatment is probably not indicated unless there has been fluctuation in the hearing There is no time limit however for institution of therapy in *fluctuating* sensory-neural hearing impairments A trial of vasodilator therapy is indicated in gradually progressive non-fluctuating sensory-neural hearing losses if a low pitched buzzing or roaring tinnitus is prominent Two such cases are included one treated after one year (Case 18, see Table VI) and one treated after six months (summary Case 1)

Once vasodilator therapy has been initiated how long should it be continued? Eighty-five per cent of the sensory-neural losses that responded did so within six weeks of onset of treatment Those that had not recovered by this time had shown some evidence of recovery as manifested by partial improvement in hearing fluctuation of hearing or marked fluctuation of tinnitus Based on these findings it would seem that there is little point in continued therapy past six weeks unless some encouraging signs are shown

When a non-fluctuating hearing loss has shown an immediate response to treatment (recovery within one to two weeks) medication may be gradually eliminated over a two-week period If response to treatment has been gradual, and in the case of fluctuating and gradual losses, some form of vasodilator therapy (usually nicotinic acid and sublingual histamine) should be continued for a period of from one to two months after the hearing has returned

CONCLUSIONS

1 A sudden sensory-neural hearing loss is a medical emergency Intensive vasodilator therapy should be initiated immediately, and has resulted in recovery of hearing in 47 per cent of the cases in this series

2 Favorable prognostic signs in sudden sensory-neural hearing losses are the following

a Low tone type of impairment

b Forty-five decibel or less hearing level in the speech hearing frequencies

c Absence of vertigo

If an individual with the above listed findings is treated within six weeks of onset of a sudden hearing loss, he should have better than a 50 per cent chance of recovery of serviceable hearing

3 Fluctuating sensory-neural hearing losses are predominantly of the low tone type associated with a low pitched tinnitus Intensive vasodilator therapy has resulted in recovery of hearing in two-thirds of the cases in this series

4 Gradually progressive non-fluctuating sensory-neural hearing losses associated with a low pitched buzzing or roaring tinnitus, should be given the benefit of intensive vasodilator therapy An occasional case may respond to this treatment

SUMMARY

The clinical findings in 247 patients with sudden and fluctuating sensory-neural hearing losses are presented A vasodilator regime is outlined and was instituted in 79 of these patients The results of this treatment are presented

Although no conclusions have been drawn as to the etiology in these cases, treatment based on the assumption that vascular spasm is fundamental in their development has resulted in recovery of hearing in approximately 50 per cent of the cases treated within six weeks of onset of the loss

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VARIATIONS OF RESULTS IN TREATMENT OF POSTOPERATIVE EAR INFECTIONS[*]

NEIL CALLAHAN, M D ,
(By Invitation),
MARSHALL H HOOD, M D ,
and
CHARLES S SALE M D ,
(By Invitation),
Portsmouth, Va

This report concerns mixed infections of the postoperative ear and has been adapted from the original and as yet unconfirmed clinical observations of one of us (M H H)

The incidence of mixed infection, which includes pseudomonas during or at the end of the postoperative phase of an otherwise faultlessly managed otologic operation, is one of the most exasperating and challenging facets in otolaryngologic practice

When an otologic procedure is planned, the surgeon must assume that he can control all the important variables from the necessarily rigid asepsis, to the successful psychic guidance of the patient through preoperative, operative, postoperative and the initial part of the after care phase, however, as the postoperative stage passes, and the packing is gradually removed, the healing process brings back the perception of sensation, including that of itching, and the danger of conscious or unconscious finger exploration by the patient becomes apparent

The impressive ceremony of sterile dressings, and the litany mass reiteration by the physician and his aides to avoid injury to the ear may persuade most patients to cooperate until final healing is completed, but there are some who do

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[*] Read at the meeting o the Southern Section American Laryngological Rhinological and Otological Society Inc New Orleans La Jan 15 1960

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not cooperate These are the patients with a long-standing history of intermittent drainage, who have been fingering their ears unconsciously, as long This patient may take a perverse joy in converting the initial period of the after care phase into an exercise in the management of external otitis

In the occasional case, completely innocent professional cleansing of an old radical cavity will stir up old infection When this type of disease is complicated by the introduction of fungus spores, the resulting growth in the depth of the cavity will defy successful cure It was the reference of just such a case of aspergillus niger, which led me to the treatment advocated by Marshall Hood, who has been successfully relieving such cases for over 20 years with 6 per cent sulphurous acid

Sulphorus acid, H_2SO_3, is made by combining sulphorous anhydride with water It is volatile, unstable, has a rather unpleasant and distinctive smell, and is said by our patients to cause a burning sensation to an inflamed otic skin, as does vinegar or alcohol A chemical firm in our locality, the Virginia Smelting Company, graciously furnishes us with our material By keeping the stock bottle under refrigeration and taking out only 5-10 cc at a time, we are able to retain the virility for approximately three months

Currently the listed authors are engaged in a cooperative project, whose purpose is to confirm by a controlled scientific approach, the clinical observations of our senior member The cases listed below have been drawn from my personal series and are presented to show essentially safe, effective control experienced in relieving the occasional local secondary infection which jeopardizes plastic procedures of the ear In the recalcitrant patient, the discovery of the characteristic smell found on the offending finger, after the initial treatment, emphasizes the importance of avoidance, and the implied threat of more pain if further irritation from self-trauma occurs, insures continued cooperation of the patient

Case 1 Mrs D N, 25 year old, white, female, choir director who was referred after exhaustion of the treatment resources of the physician, who had innocently irrigated a three year old radical cavity, was first seen on November 13, 1958, and last seen on February 24, 1959 During weekly

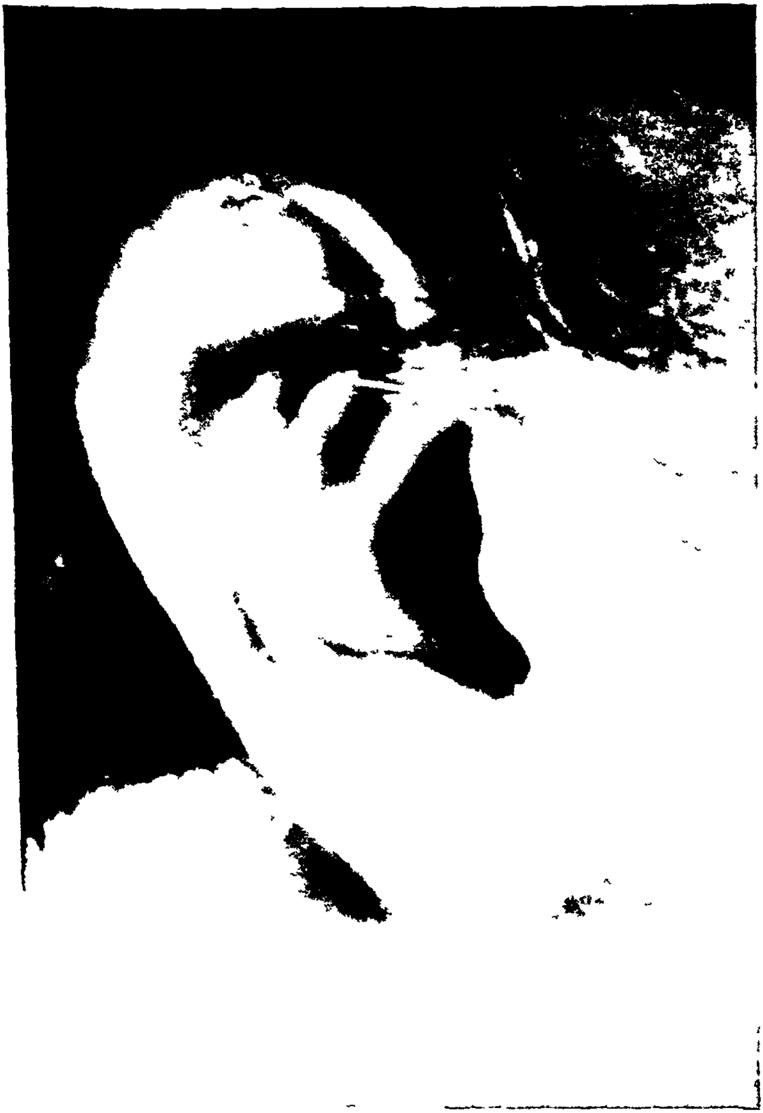

Fig 1

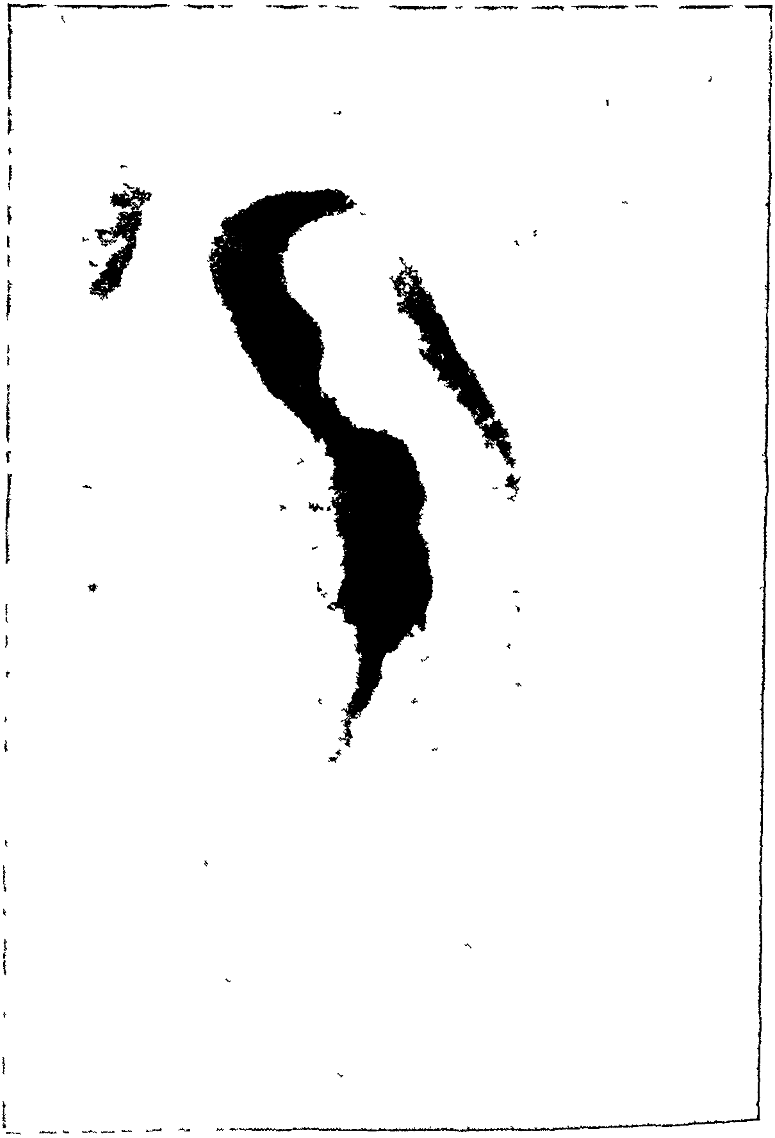

Fig 2

visits in November and December, the mixed infection was attacked with most of the standard varieties of treatment, with riddance of the staph albus and a prompt increase of the aspergillus niger Consultation with Dr Hood was sought early in January, and his method of meticulous cleansing, followed by thorough scrubbing of every nook and cranny with acid soaked swabs, was done on January 10, 1959 The immediate subjective relief and the improved appearance on the visit of January 13, gave us new hope and after three such visits it became apparent that while the sinodural angle and the mastoid tip undercuts were still growing fungi, we were ahead In our elation the patient permitted me to fill the cavity with a newly prepared solution and demonstrated the courage to permit it to remain for four minutes This was extended to ten minutes one week later, and the patient was discharged on February 24, 1959

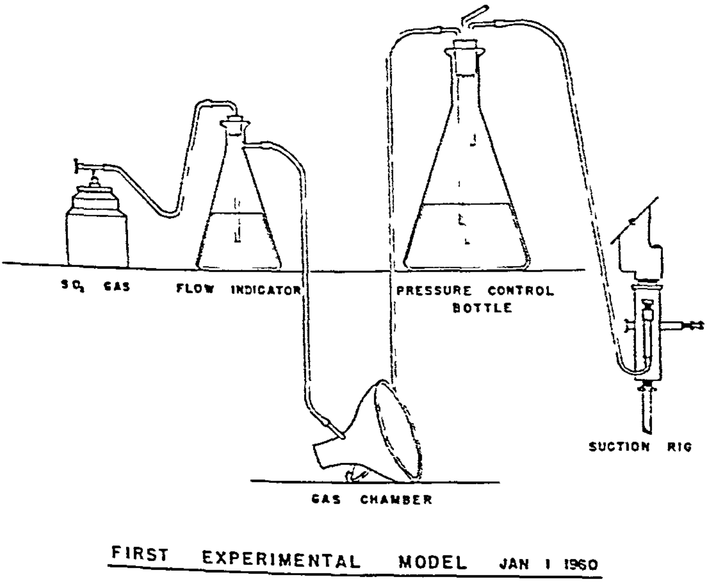

Fig 3

Case 2 Mrs G J white housewife had a modified radical mastoidectomy with skin graft on October 12 1959 Three days before her discharge from the hospital on October 20, she got her hands under the bandage and scratched the donor site and the ear canal vigorously The packing still present in the ear protected the ear drum area but the self inflicted trauma continued to appear in spite of the exhortations of the physician and her family

The case had been designated as a control, was followed carefully, with scrupulous cleanliness neomycin ointment and powder to the plastic areas and office visits every four to seven days The depths of the cavity were carefully walled off until culture taken from the tip area indicated a mixed infection of staphylococcus aureus, B coli and pseudomonas on November 20 The routine was changed to the acid pattern on December 1, 1959, and on December 12, 1959, all the surface granulation tissue had disappeared, and by Decemeber 24, the ear was dry There were breaks in the skin, and the acid drops routine was continued, dispensing a new dose each time On January 5, 1960, the skin appeared normal, and the culture was negative

This report should be closed by the statement that infections which arise at operation are evident at the first dressing These will respond satisfactorily to this treatment or to exposure of the ear to the SO_2 gas in a chamber which will cover the ear It is anticipated that either method will clean up resistant infections in those cases in which tympanoplasties are planned

THE DE ROALDES AWARD

At the recent annual meeting of the American Laryngological Association, the DeRoaldes Award and gold medal were presented to Dr Dean M Lierle as a great physician, surgeon, educator and humanitarian and in recognition of his services to otolaryngology and to the American Laryngological Association

METHOD FOR THE IDENTIFICATION AND LOCALIZATION OF CEREBROSPINAL FLUID, RHINORRHEA AND OTORRHEA [*][†]

F R Kirchner, M D ,
(By Invitation),
and
G O Proud, M D ,

Kansas City, Kans

This paper describes the experimental background as well as the practicality of a new method that can be used for the identification and localization of cerebrospinal fluid fistulae into the different cavities located within the base of the skull

Several authors who studied the late complications of head trauma found that about one per cent of the patients presented this particular condition Although injury is by far the main causative factor, ear, nasal and cranial surgery, infections, hydrocephalus and tumor can also bring about this problem Surgery is frequently indicated to effect permanent cessation of the leakage, but the neurosurgeon is often hard put to determine the site of surgical attack The otolaryngologist has been of modest help to the neurosurgeon in his difficulty

The identification and localization of CSF leakage has depended almost entirely upon the clinical history and radiological findings Various chemical tests have been advocated for the differentiation between spinal fluid and other secretions, but these sources of information are often insufficient and sometimes equivocal The medical literature contains many reports attesting to this fact For example, Lewin[1] reported about a patient with vasomotor rhinitis who was

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[*] Read at the Sixty-Third Annual Meeting of the American Laryngological Rhinological and Otological Society Inc Miami Beach Fla March 16 1960

[†] From the Department of Otorhinolaryngology of the University of Kansas School of Medicine

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submitted to craniotomy because of the incorrect diagnosis of CSF rhinorrhea Ridley[2] in 1930 demonstrated that the lacrimal secretions contain as much reducing substances as the spinal fluid, therefore, the presence of this substance in any nasal secretion is inconclusive

Concerning the localization of the site of leakage, Dandy[3] wrote "Drainage of fluid from one nostril is fair evidence that the fistula is on the corresponding side of the anterior fossa, but that is by no means dependable proof There are times when only an exploratory operation will determine the site of the opening, and there may be bilateral fistulae" He also stated that[3] "Drainage of CSF from the nose is not pathognomonic of a fistula into the frontal or ethmoid sinus, but may occur through the mastoid bone into the middle ear and Eustachian tube"

Roentgenological studies are of some aid in the localization of the general area of leakage, but as was pointed out separately by Morley and Hetherington,[4] Ecker,[5] Adson and Vihlein,[6] they are of little assistance in pointing out the exact site and of no assistance in the localization of CSF fistulae involving the ethmoid and sphenoidal sinuses

Noting these problems several authors have tried different methods in an attempt to make the site of leakage objective Fox[7] injected indigo carmine dye into the cisterna magna, but due to the poor contrast obtained, the usefulness of the method was limited to instances in which the rhinorrhea was copious Crow and Keogh[8] injected radioactive sodium (Na24) into the cisterna in two cases Cotton pledgets were conveniently distributed in the walls and roof of the nose and nasopharynx and upon the openings of the frontal sinuses and Eustachian tubes After their removal the pledgets were examined under a Geiger Mueller counter and the site of leakage was determined by the one showing the highest radioactive count This technique, however, required specialized instruments for its performance and was an indirect method

MATERIALS AND TECHNIQUE

While doing studies on the inner ear with photoluminescent substances the possibility of using fluorescein to detect the site of spinal fluid leakage became apparent The advantages of this substance lie in its physical chemical and pharmacological properties For example, the substance is photoluminescent, and under ultraviolet light its yellowish green glare makes it easily detectable from the surrounding environment This is true even when it is mixed with other colored material such as hemoglobin, thus it is superior to formerly used dyes The amount required for its detection is minimal

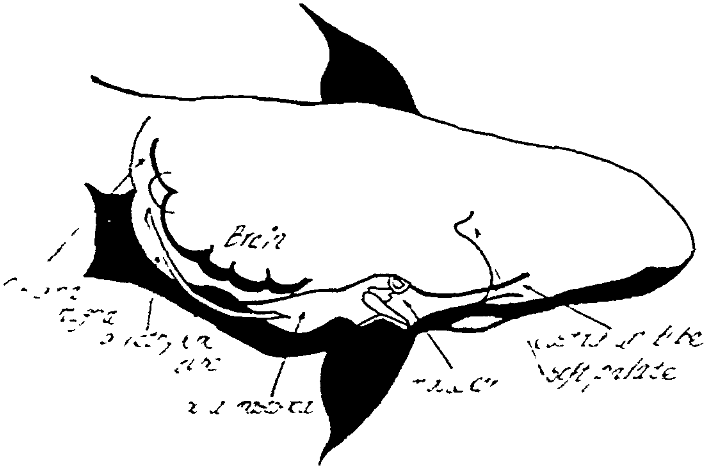

Fig 1 Method of creating a cerebrospinal fluid fistula between the cisterna magna and bulla mastoidea in the experimental dog

(1-1,600,000 dil), and fluorescein's minute molecular structure makes for its ready diffusion in other fluids Its chemical structure is stable and its mild alkalinity is not incompatible with body fluids It is excreted by becoming bound temporarily to some of the plasma proteins Its toxic effects are nil, Strauss[9] administered it orally in doses as large as 6 gms with no outward effects, and Lange[10] recommended intravenous injection of 5 cc of 5 per cent solution for determination of circulation time

The dog was elected as the experimental animal, for its anatomy was familiar and the available facilities for its care were superior In three animals anesthetized by sodium nembutal, a fistula was created by inserting one end of a No 18 polyethylene tube into the cisterna magna and the other as shown in Fig 1, into the bulla mastoidea This allowed the spinal fluid to flow into the respective Eustachian tube Antibiotic therapy was administered for one week of convalescence Direct visualization of the nasopharynx then revealed no evidence of fluid as shown in Fig 2, but when 1 cc of 5 per cent fluorescein was injected into the spinal canal and the re-examination done under ultraviolet light the leakage was apparent in all three animals Fig 3 demonstrates fluorescein coming through left Eustachian tube

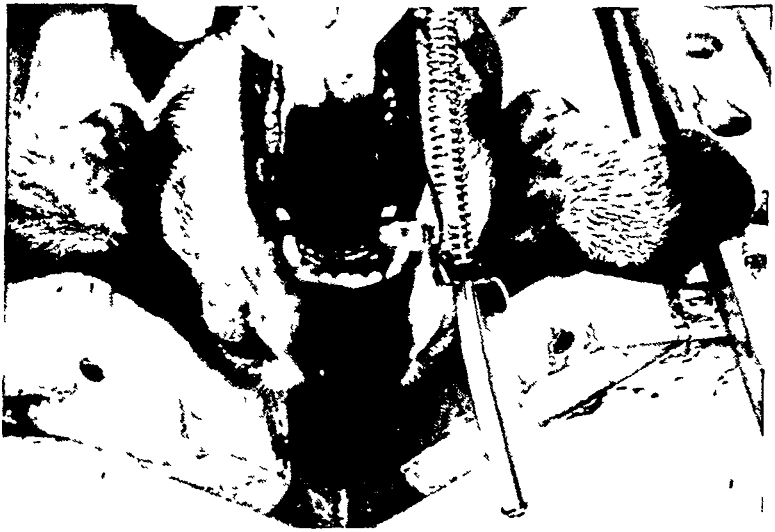

Fig 2 Anesthetized dog

In a fourth dog, under general anesthesia, after opening the bulla mastoidea a perforating burr was used to create a fistula between the middle ear cavity and the cranial fossa via the tegmen tympany Fig 4 shows the place of the fistula (marked by an arrow) just above the round window One cc of fluorescein was injected into the spinal canal in the lumbar area and ultraviolet examination disclosed, as shown by Fig 5, the fluorescent materials both on observation through the bullar opening as well as through the tympanic membrane

The final steps of the experiment were performed upon the human on demand by the Department of Neurosurgery which had heard of the animal experiments

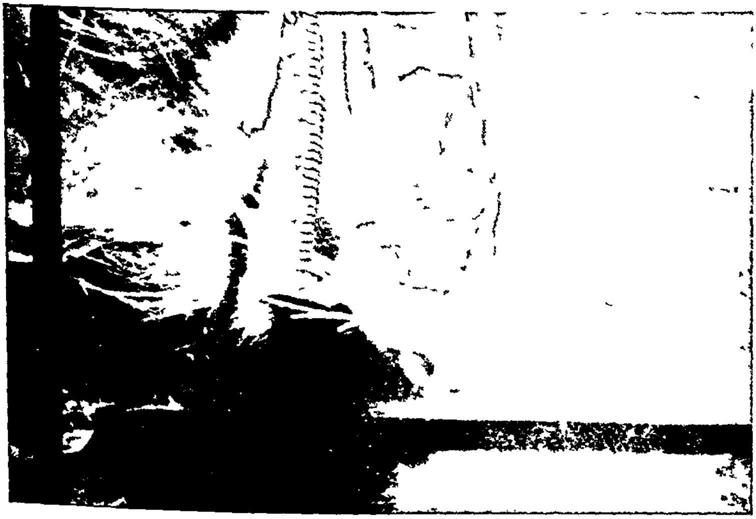

Fig 3 Anesthetized dog under ultraviolet light showing the glare of fluorescein in the opening of the left Eustachian tube

To meet these clinical problems a technique of examination was developed The initial step was the cocanization of the nose and nasopharynx followed by the insertion of a cotton pack midway in the nasal chambers (see Fig 6) Divided in this way it was possible to detect where the leakage was located For example, fluorescein detected under the middle turbinate anterior to the pack was necessarily coming from the frontal sinuses (see Fig 7) If the fistula was located in the cribiform plate the fluorescein was detected upon the midline in the nasal septum (see Fig 8), leakages coming through the sphenoidal sinus could have been easily discovered

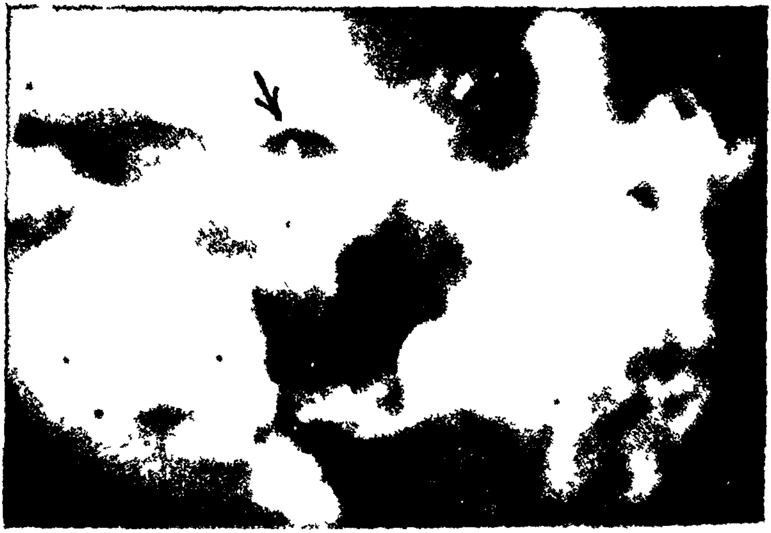

Fig 4 Photograph of middle ear, note the round window and arrow showing the site where the fistula was created (10X)

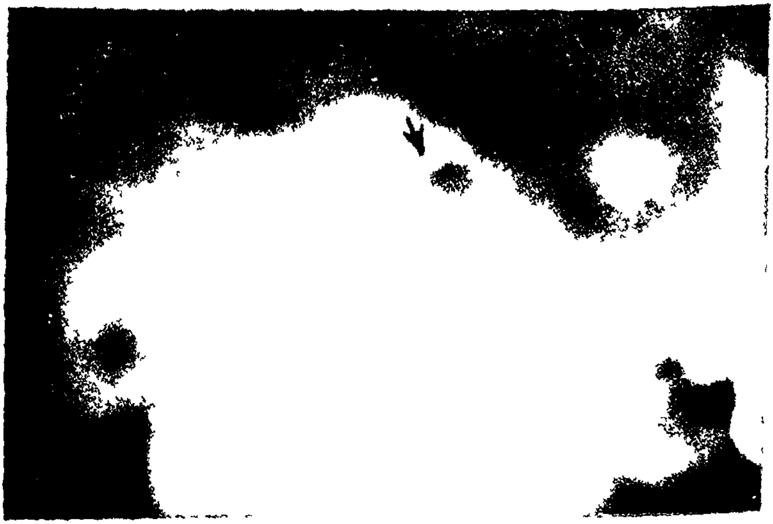

Fig 5 Photograph of middle ear of the dog under ultraviolet light arrow pointing to the glare of fluorescein leaking through the created fistula (10X)

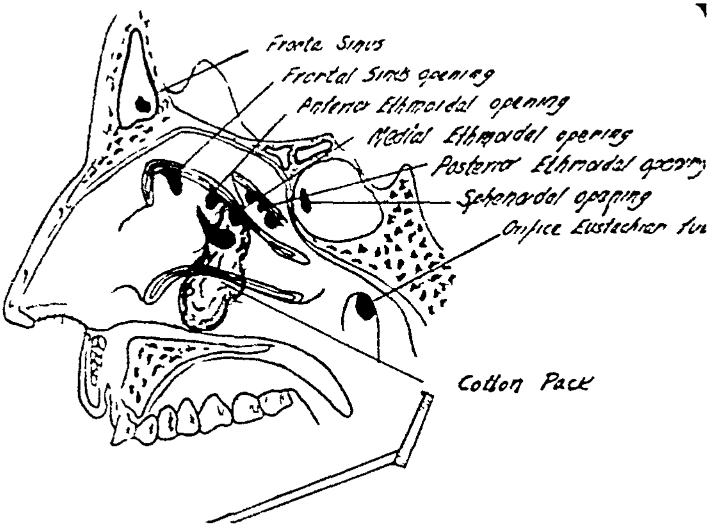

Fig 6 Location of cotton pack in the nose

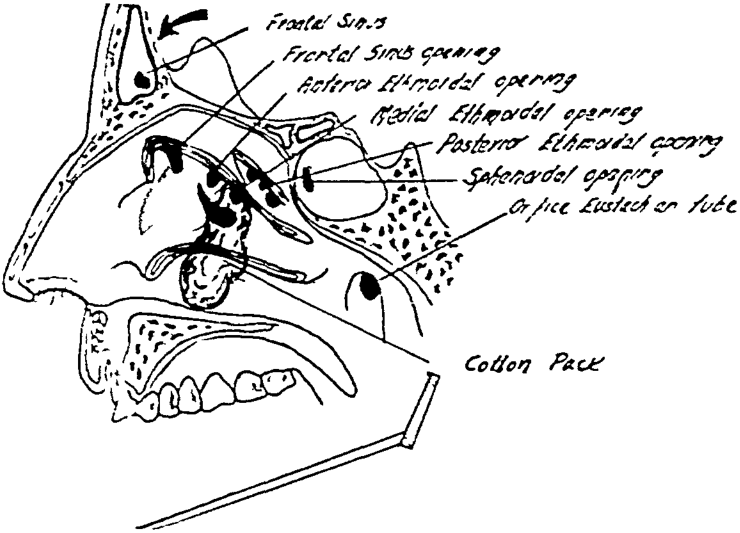

Fig 7 Leakages through the frontal sinus can easily be discovered in the opening of the nasofrontal duct in the nose

by indirect nasopharyngoscopy under ultraviolet light Fistulas from the middle cerebral fossa involving the petro-tubal area, as shown in Fig 9, would cause fluorescence upon the respective Eustachian tube openings In cases of leakage through the middle ear or tegmen mastoidea the fluorescence would become apparent in the respective Eustachian tube opening as well as behind the respective tympanic membrane (see Fig 10)

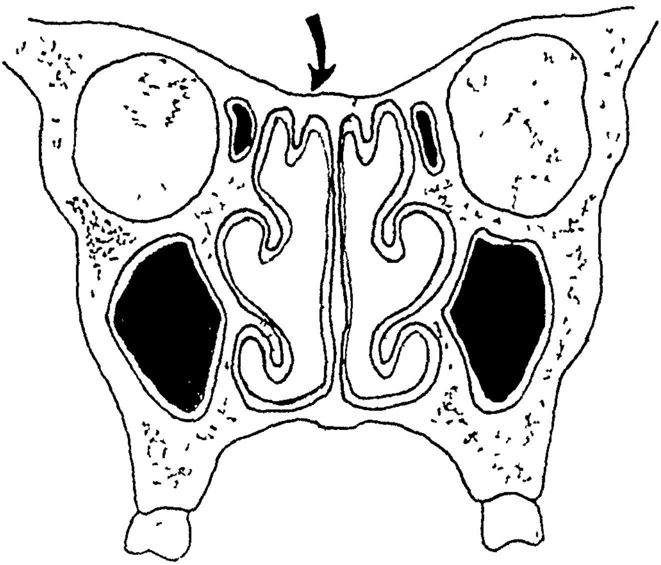

Fig 8 Leakages through the cribriform plate can be discovered on either side of the nasal septum

The first case investigated by this method was that of an adult female complaining of hearing loss in the right ear following an automobile accident X-ray studies of the skull were negative Examination revealed a hemotympanum One cc of 5 per cent fluorescein was injected into the lumbar spine, and the patient was placed in a prone position with the head slightly dependent for 15 minutes Ultraviolet examination of the ears was then carried out and fluorescence was observed in the escaping fluid following myringotomy The

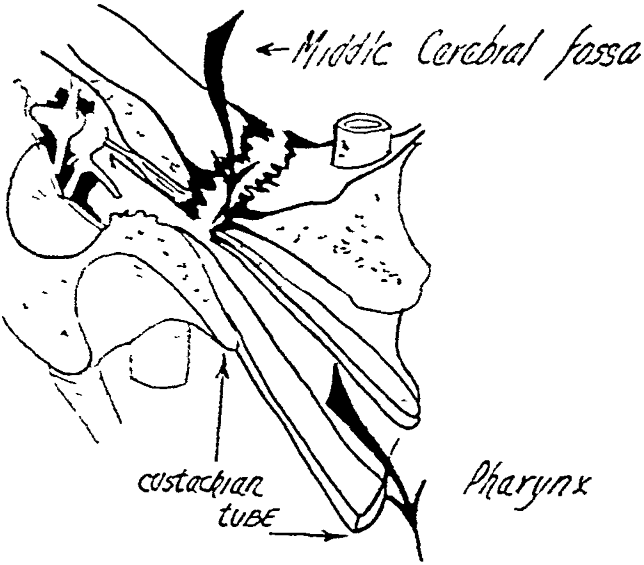

Fig 9 Fistula involving the petro-tubal area

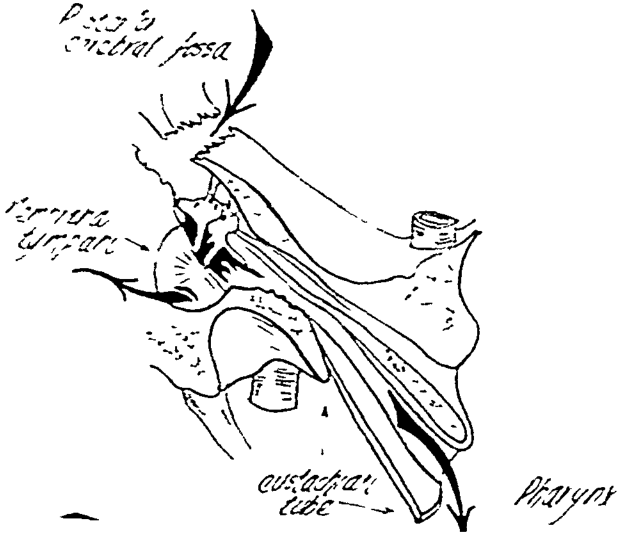

Fig 10 Fistula of the tegmen mastoidea fluorescein present at the Eustachian tube opening and tympanic membrane

same material was discovered in the nasopharynx in the area corresponding to the Eustachian tube opening The patient did well under conservative treatment It was three months later, and after two X-ray examinations of the skull were performed that a line of fracture was discovered upon the middle cerebral fossa

A second patient, a 24-year-old male was involved in an automobile accident at the age of 18 He had experienced six attacks of meningitis and had undergone surgery seven times for closure of dural tear evidenced by intermittent rhinorrhea After the intraspinal injection of 1 cc of 5 per cent fluorescein an ultraviolet examination was carried out with the patient in the sitting up position and in a darkened room The fluorescent material was found coming anterior to the previously inserted nasal pack near the midline The assumption was that the fracture was located in the cribiform plate Surgical exploration found the tear at the predicted site, and it was repaired with fascia lata Four months later meningitis recurred and an identical leak was found by the same method though rhinorrhea was not apparent without fluorescein study Surgical repair was again performed, this time with success

CONCLUSIONS

1 Fluorescein injected intraspinally and searched for under UV light is of practical value in the identification and localization of CSF leakages

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VIIeme INTERNATIONAL CONGRESS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY

The Seventh International Congress of Otology, Rhinology and Laryngology will be held in Paris, July 23-29, 1961, at the New Faculte of Medicine, Rue des Saints-Peres The program includes *1* The Recent Acquisitions, Pathology and Therapeutics of Otosclerosis by Profs Sourdille, Sercer and Krmpotic, Weber, Ardouin, Larroude, Ferreri and Shambaugh *2* Indications and Five-Year Results in Surgery and Radium in the Treatment of Cancers of the Larynx and Hypopharynx—Profs Pietrantoni and Agazzi Alonzo, Casadesus, C L Jackson, Leicher, Leronx-Robert and Ormerod *3* A Report on Allergy and Infections of the Nose and Bronchus—Profs Van Dishoeck, Clerici, Hlavacek, Voorhorst, Mayer, Laskiewicz and Terracol

Tours and entertainment for guests and their ladies have been arranged President Dr Maurice Aubry, Secretary General Henry Guillon For further details write Dr. Guillon, 6 Avenue McMahon, Paris, France

ISOLATED LARYNGEAL KERATOSIS

Its Relation to Carcinoma of the Larynx Based on a Clinicopathologic Study of 87 Consecutive Cases With Long-Term Follow-Up[*][†][‡]

MALCOLM H McGAVRAN, M D ,
(By Invitation),
WALTER C BAUER, M D ,
(By Invitation),
and
JOSEPH H OGURA, M D ,

St Louis, Mo

INTRODUCTION

The relationship of keratosis of the larynx (variously termed hyperkeratosis, leukoplakia and pachydermia laryngis), to cancer, has been the subject of a number of case studies[1-9] All generally support the view that metaplasia and keratinization of the laryngeal mucosa are associated with the subsequent appearance of invasive epidermoid carcinoma The implication is that isolated keratosis can unexpectedly and unpredictably undergo "transformation" or "metamorphosis" to cancer[1,4,6,10-15] The concept of the "malignant potential" of laryngeal keratosis has become so widely accepted that few individuals have asked critical questions or have been skeptical of the prevalent view[16,17]

With the recent interest in cancer prevention by treating "precancerous" lesions, the conservative therapy of the past for the management of keratosis is yielding to change Some laryngologists now advocate treating this lesion in remark-

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[*] Read at the Sixty-Third Annual Meeting of the American Laryngological Rhinological and Otological Society Inc Miami Beach Fla, March 17 1960

[†] From the Department of Surgery Division of Surgical Pathology Washington University School of Medicine McMillan Barnes, and Barnard Free Skin and Cancer Hospitals St Louis, Mo

[‡] This work was partially supported by a Grant-in-Aid from the United States Public Health Service [Grant No CS-9108 (C9)]

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ably radical ways, *e g* , laryngofissure,[8,15] laryngectomy[13,15] and irradiation with cancerocidal doses[13,15,18]—ways usually reserved for treatment of cancer itself The preoccupation with a chance for successful cancer prevention and cure has tended to hide the hard fact that the magnitude of the risk of cancer in keratosis patients is unknown as yet and that therapeutic procedures for cancer of the larynx are known to be associated with appreciable risks of morbidity and laryngeal dysfunction[19,20,21] Before permitting keratosis patients to undertake such risks, either of developing cancer or of morbidity and laryngeal dysfunction from cancer prophylaxis, it is imperative that the relationship between keratosis and cancer be more closely investigated

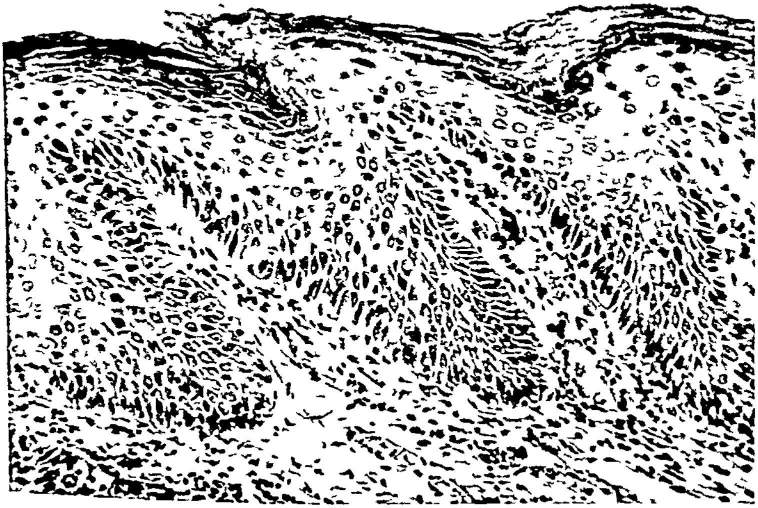


Fig 1 This area shows hyperkeratosis with minimal epithelial hyperplasia There is acanthosis and a regular maturation of the spinous layer to form a well-developed granular layer A thin layer of acellular keratin is formed on the surface No atypia is seen This is classified as HK. Case 1 W U III 59-6936A (X275)

It would seem, therefore, that a careful clinical and pathological analysis of a group of patients with isolated laryngeal keratosis correlated with the results of long term follow-up *might clarify the situation and supply prognostically significant information.* The work of Putney and O'Keefe[18] represents the only other study with a somewhat similar approach to the problem. Their conclusions, which support the concept of the precancerous nature of keratosis, are at variance with the results of this study and will be discussed.

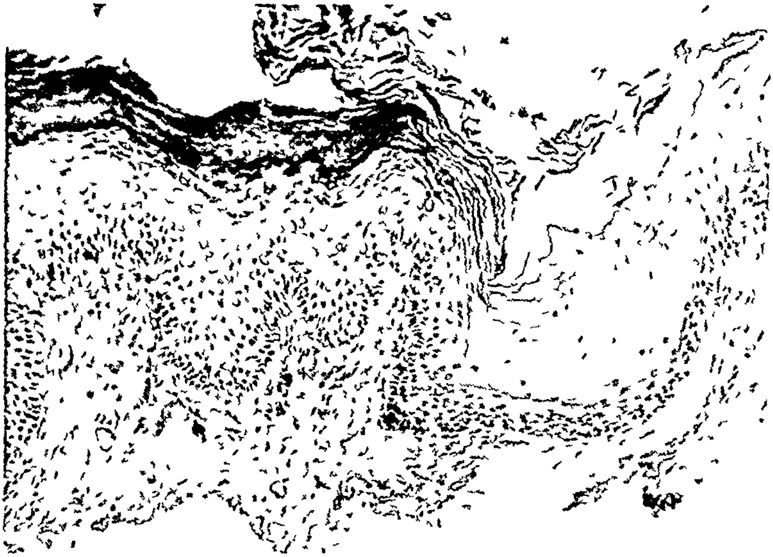


Fig. 2 Note the epithelial alterations of hyperkeratosis on the left with orderly maturation and granular layer formation topped by a thick layer of acellular keratin in continuity with an area of parakeratosis showing surface accumulation of keratinized cells with retention of nuclei. No atypia is present. For classification purposes this is HK. Case 2. W U III 59-3518A (X150.)

MATERIAL AND METHODS

All of the laryngoscopic biopsies accumulated during the years 1945 through 1954 were reviewed by two of us independently and without knowledge of the clinical findings or subsequent course. The histologic alterations were classified (see below). On the basis of these studies and analysis of the histories, clinical findings and diagnoses all cases of squamous carcinoma, squamous papillomas, laryngeal vocal nodules, contact granulomas and nonspecific inflammatory lesions associated with minimal epithelial alterations were

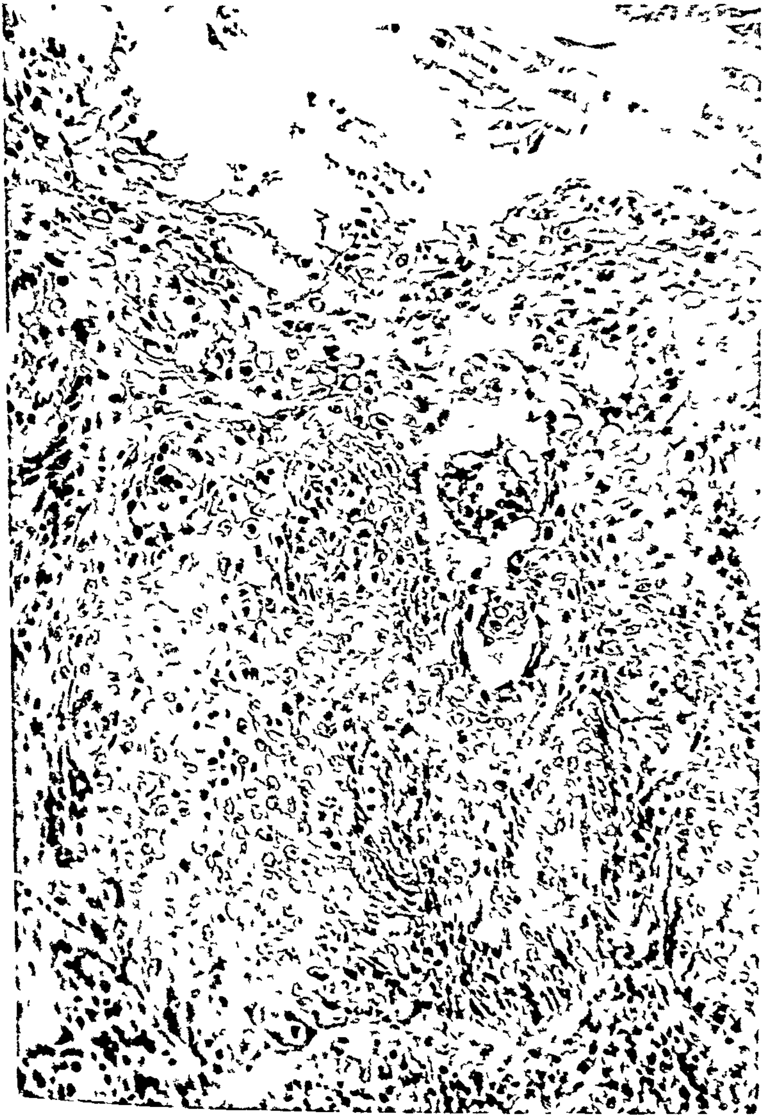
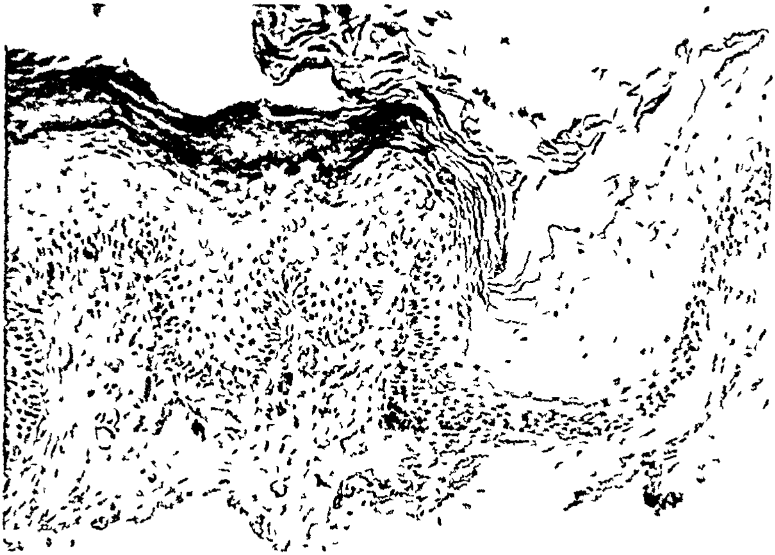


Fig [illegible] A parakeratotic area from a subsequent biopsy showing disturbed maturation in deeply acanthotic epithelial folds The nuclei present considerable variation in form and chromatin distribution This is classified as PKA Case 2 W U III 59-5519A (X250) might clarify the situation and supply prognostically significant information The work of Putney and O'Keefe[18] represents the only other study with a somewhat similar approach to the problem Their conclusions, which support the concept of the precancerous nature of keratosis, are at variance with the results of this study and will be discussed



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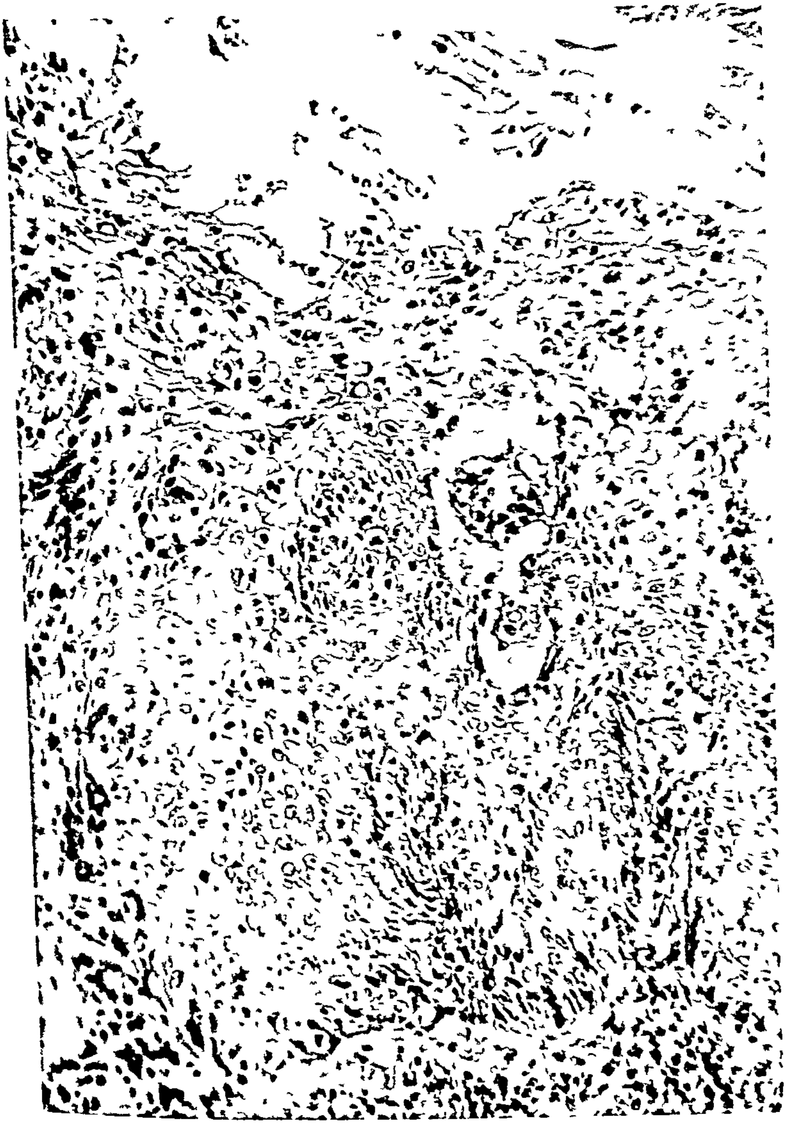

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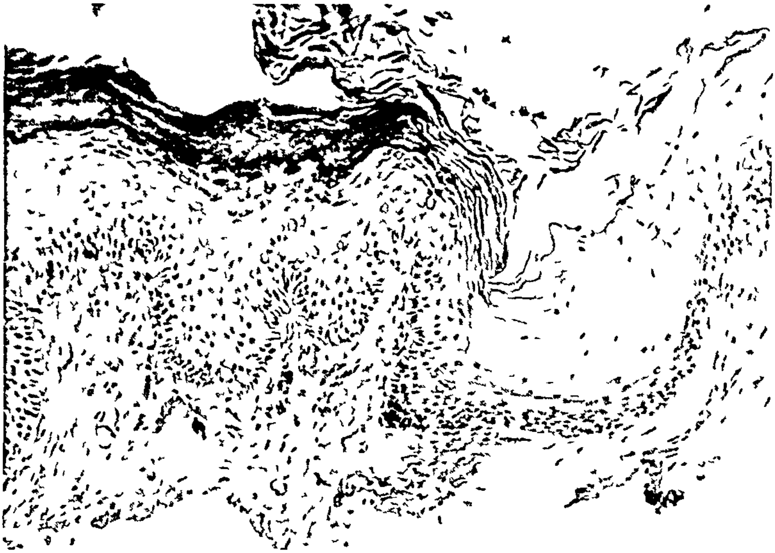

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RESULTS

On subsequent biopsy, at intervals of 2 5, one and one months respectively, three of these 87 cases were found to have squamous carcinoma These cases are not interpreted as "quick metamorphosis" of keratosis into carcinoma as suggested by Priest,[9] but simply as the association of keratotic changes and invasive cancer—a very frequent finding[22]—in which the first biopsy was taken from an unrepresentative area This is a remarkably low incidence for this course of events and speaks highly of the accuracy of the laryngologists' biopsy samples and the thoroughness of the pathological examination, for 470 cases of carcinoma of the larynx were

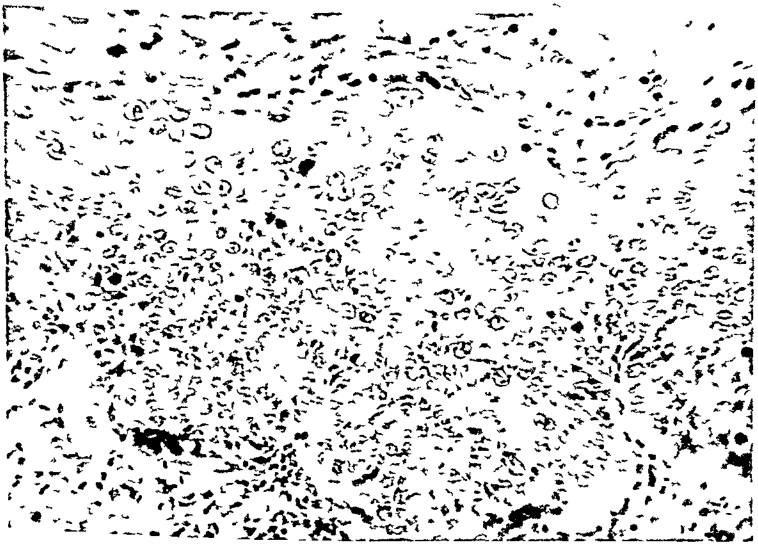

Fig 5 Another area in the same biopsy showing disorderly maturation of cells most apparent in the deeper zones but present to a degree even in the top zone Such areas as depicted here often fail to completely keratinize and are parakeratotic This is PKA Case 4 W U III 59-3517A (X310)

excluded There remained 87 consecutive cases of isolated laryngeal keratosis All of these patients have been followed to date, December, 1959, or to death, a minimum of five and a maximum of 15 years, and all subsequent pathological specimens have been studied and clinical observations recorded

The normal epithelial covering of the larynx is nonkeratinized squamous epithelium, except for the laryngeal ventricle and subglottic area where pseudostratified, ciliated, columnar epithelium exists *Epithelial hyperplasia,* defined as thickening with or without acanthosis, *parakeratosis,* defined as the surface accumulation of incompletely keratinized cells retaining their nuclei, *keratosis* and *hyperkeratosis,* defined as the formation of a granular layer with surface accumulation of completely keratinized cells, *epithelial atypia,* defined as abnormal maturation of the epithelial cells with nuclear abberation, but of insufficient degree to be classified as carcinoma-*in situ,* are the morphologic alterations that were studied On

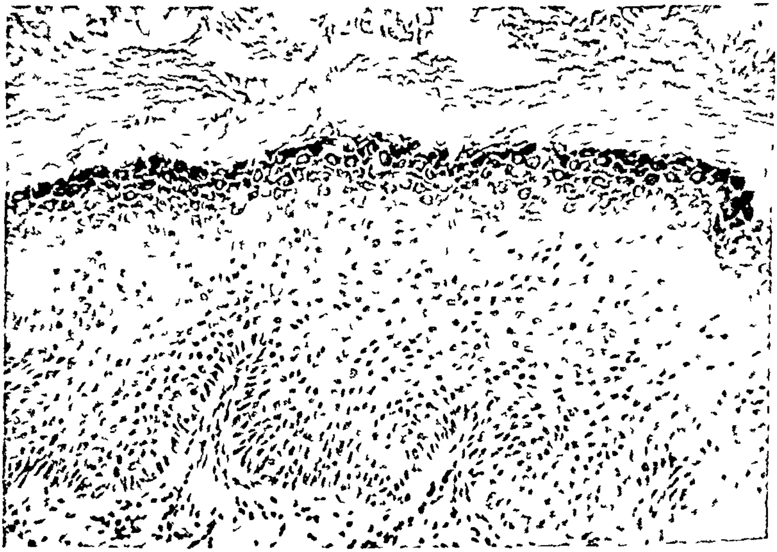

Fig 4 A typical area of hyperkeratosis showing a thick hyperplastic epithelium with a uniform and orderly maturation of cells to form a thick keratohyalin granular layer A layer of desquamating keratin tops the lesion Typical HK Case 4 W U Ill 59-3516A (X250)

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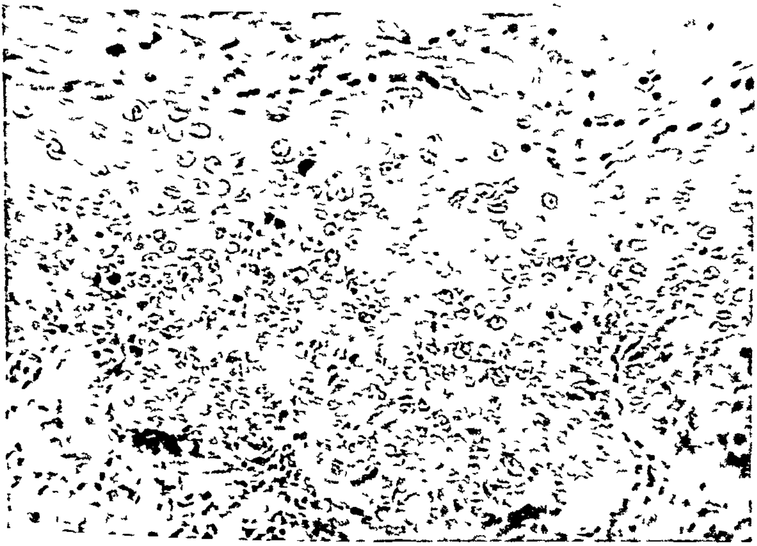

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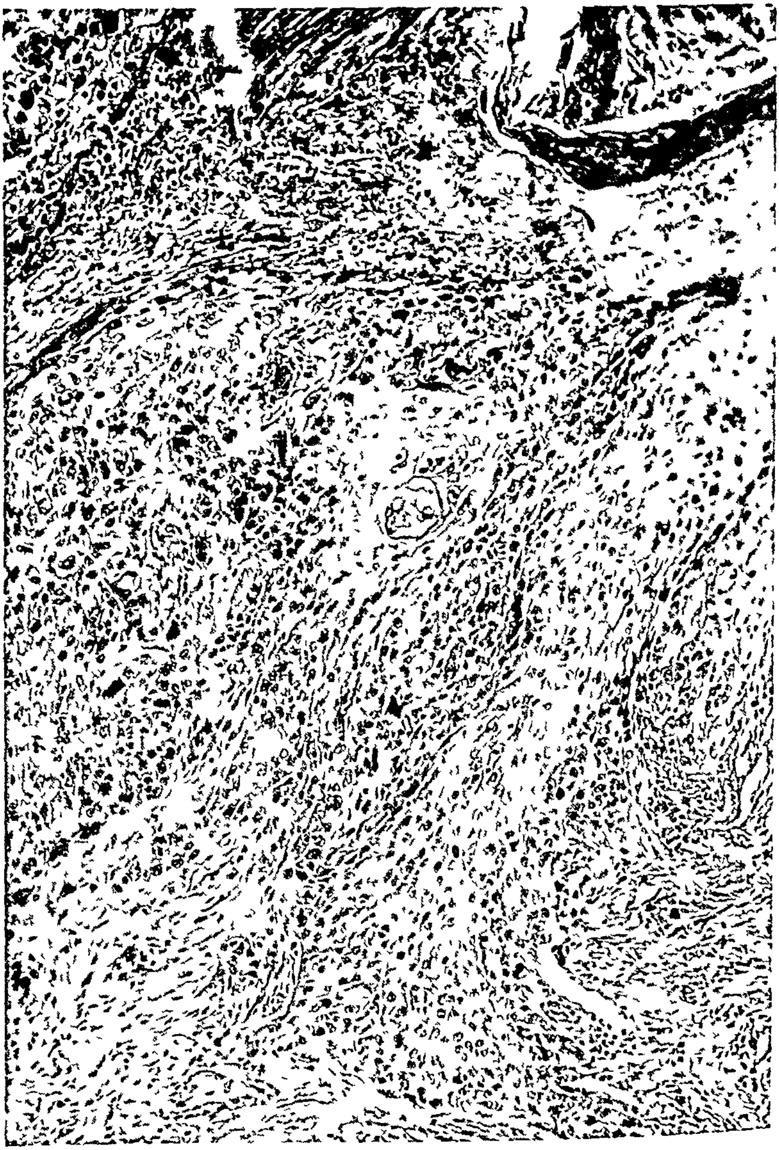

Fig 6 Same case as Figs 4 and 5 This biopsy taken six years later shows an invasive epidermoid carcinoma W U Ill 59-3515A (X130)

diagnosed by biopsy during this same period An incidence of 0 2 per cent for this "error" lays to rest, we hope, the debate concerning the accuracy of biopsy and/or histologic examination These three cases, all in males, are considered squamous carcinoma from the onset

Of the 84 remaining cases, 74 were male and ten female The mean age for the males is 51 7 years and for the females 50 5 years The distribution of cases by decades of age is shown in Table I The dominant symptom was hoarseness, which was present for varying lengths of time as shown in Table II The areas of the larynx involved by keratosis are shown in Table III No correlation is found between site of involvement and duration of symptoms or histologic changes The nine cases with involvement of the interarytenoid area and the posterior portions of both vocal cords correspond to what has been called pachydermia laryngis[4] None of these nine cases developed invasive carcinoma

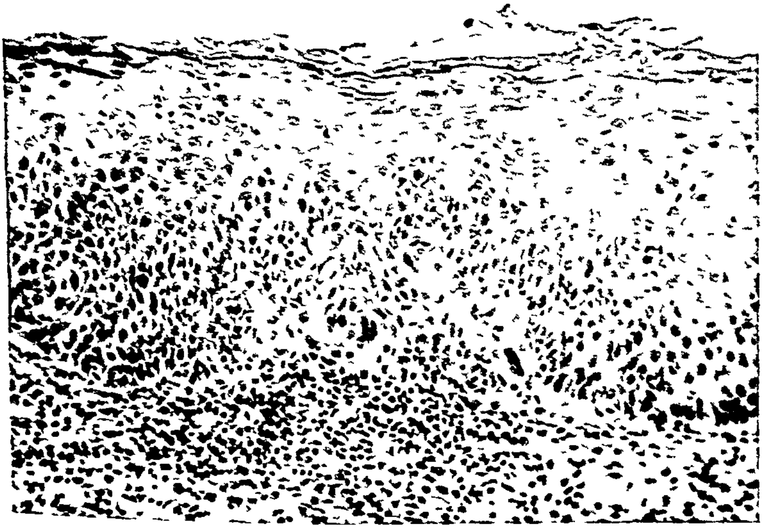

Fig 7 An area from a biopsy taken in 1951 showing epithelial hyperplasia, atypia and parakeratosis Other areas were interpreted as HKA Case 5 W U III 59-5523A (X275)

TABLE I

Distribution by Decades of 84 Patients with Laryngeal Keratosis

	Decades					
	20-29	30-39	40-49	50-59	60-69	70-79
Males	2	10	20	18	20	4
Females	0	3	2	3	1	1
Total	2	13	22	21	21	5

TABLE II

Duration of Hoarseness Prior to Biopsy in 84 Patients with Laryngeal Keratosis

	Months						
	0-6	7-12	13-18	19-24	25-30	30+	Unknown
Number of Patients	44	7	8	0	6	17	2

TABLE III

Sites of Laryngeal Involvement by Keratosis, 84 Patients

	Number of Cases	Per Cent
Both True Vocal Cords	28	33
Left True Vocal Cord	16	19
Right True Vocal Cord	13	15
Both Vocal Cords and Interarytenoid Area	9	11
Interarytenoid Area Only	8	10
Miscellaneous	10	12
	84	100

TABLE IV

Histologic Classification, Mean Ages, Years Followed
84 Cases of Laryngeal Keratosis

Histologic Subgroup*	No of Cases	Mean Age Years	Mean Years Followed
HK	35	51.8	7.0
PK	31	47.3	8.0
HKA	13	61.5	7.2
PKA	5	52.0	6.4

*See text for definition of these groups

We have no conclusive evidence regarding inciting agents or causal factors. Symptomatically a few individuals improved after the cessation of excessive smoking or the institution of voice therapy, but the significance of this is uncertain.

The histologic classification of the keratotic lesions in these patients is found in Table IV. Included are the mean age of the patients in each sub-group and the average number of years the patients were followed As previously mentioned all of these patients have been followed to date or death Those alive have been questioned as to persistence of symptoms, further therapy and, where possible, current laryngoscopic observations recorded The cause of death is known in all patients dead

The number of cases with recurrence or persistence of hyperkeratosis is estimated in two ways (see Table V) one, those cases proven by subsequent biopsies and two those cases with persistent or recurrent hoarseness Twenty-five of the 84 cases (30 per cent) had recurrence proven by biopsy Close to half of the cases had persistence of hoarseness and presumably of hyperkeratosis The true recurrence or persistence rate lies somewhere between the two estimates A high percentage (73 per cent) of patients with epithelial atypia in addition to keratosis had persistence of symptoms and multiple biopsies Table IV shows the distribution of the lesions in the 25 cases with multiple biopsies

TABLE V

Recurrence of Hyperkeratosis in 84 Cases as Determined by (a) Multiple Biopsies and (b) Peristence of Symptoms (Hoarseness)

Histologic Subgroup*	(a) Multiple Biopsies	(b)		
		Symptomatic	Asymptomatic	Unknown
HK	10	12	20	3
PK	4	13	13	5
HKA	10	10	1	2
PKA	1	1	4	0
Total	25	36	38	10

*See text for definition of these groups

Laryngeal keratosis may occasionally involve large areas, appear quite aggressive and narrow the glottis enough to necessitate tracheotomy for respiratory distress, two such cases were found Unless the repeated endoscopic excisions in such cases are done with care a useless larynx may result Two instructive cases in this series with extensive and refractory lesions are reported herein

Case 1 G B , a 60 year old white insulation service manager, was first seen in December, 1946, having been intermittently hoarse for ten years He abused his voice, shouting at work, and subsequently was found to have a positive serologic test for syphilis Endoscopic examination showed thickened red gray vocal bands with involvement of the left aryepiglottic fold Biopsies were taken that showed hyperkeratosis Over the ensuing 12 years he was admitted to the hospital 44 times and 46 biopsies and strippings of both true and false cords and extensive areas of the supraglottic mucosa performed All of these showed hyperkeratosis, epithelial hyperplasia and occasionally slight epithelial atypia The last biopsy was taken in December 1957 Carcinoma was never found His voice improved somewhat after each excision, and though he decreased smoking he did not stop He died in May, 1958, of heart failure without evidence of laryngeal cancer Due to the care taken by his laryngologist his larynx remained functional, though his voice was hoarse (see Fig 1)

TABLE VI

Distribution of Recurrent Isolated Keratosis in 25 Cases with Multiple Biopsies

Site	No of Cases	Proposed Therapy*
One Cord	5	Laryngofissure
Both Cords	11	Irradiation Therapy
Entire Larynx	3	Laryngectomy
Both Cords and Subglottic Area	1	Laryngectomy
"Pachydermia Laryngis"	3	Conservative Measures
One Cord and Interarytenoid Area	1	Conservative Measures
Interarytenoid Area Only	1	Conservative Measures

*See text

Case 2 I L , a 73 year old white male merchant was seen in June, 1948, with a history of hoarseness for two years and a white plaque on the anterior third of both cords with involvement of the anterior commissure The first and eight subsequent biopsies, over the ensuing nine years, showed extreme hyperkeratosis with moderate atypia Each stripping was associated with marked improvement in the quality of his voice He was last seen in March, 1959, at the age of 84, with a small quiescent white patch on the left cord Keratotic involvement in this case has been limited to the true cords and anterior commissure Carcinoma has not presented itself, and this man still has a functional larynx (see Figs 2 and 3)

The results of the follow-up study on the 84 determinate cases are shown in Table VII Three of these 84 patients developed carcinoma of the larynx one, nine months, one, six years, and the other eight years after the initial biopsy demonstrating keratosis

Case 3 M K a 50 year old policeman was seen in January, 1954, complaining of intermittent hoarseness of one year's duration and a 30 pound weight loss *Laryngoscopy showed a rough white lesion involving both true cords* Biopsy showed hyperkeratosis Nine months later the patient was seen at another hospital by another physician for persistent symp toms Another biopsy showed invasive epidermoid carcinoma and he was treated by irradiation therapy Persistent cancer was found three months later and a laryngectomy demonstrated a 4x3 cm poorly differentiated carcinoma with blood vessel invasion Tumor was present at the surgical margins Stomal recurrence was noted a few months later and he died 19 months after his first biopsy

Case 4 H J a 64 year-old negro laborer was first seen in September 1952 complaining of painless hoarseness of one year's duration Laryngoscopic examination showed leukoplakia of the anterior third of the left vocal cord without change in its mobility A biopsy and stripping demonstrated hyperkeratosis and atypia (see Figs 4 and 5) He was not seen again until August 1955, when he returned complaining of hoarseness A small white plaque on the anterior third of the right cord was biopsied but no histological abnormalities were seen In September 1958 he was admitted to St Louis Veterans Hospital a lesion of the left vocal cord was biopsied diagnosed as squamous carcinoma and a laryngectomy done (see Fig 6) He is alive without evidence of metastases to date

TABLE VII

Survival of Patients with Isolated Keratosis Followed for Five to 15 Years (1945 1955)

87 Cases—Total Series
3 Cases— Missed Carcinoma "*
84 Cases—Determinate Series
Three Cases Developed Invasive Epidermoid Carcinoma—
  1 case 9 months post biopsy died of cancer 10 months later
  1 case 6 years post biopsy, alive and well
  1 case 8 years post biopsy alive and well
81 Cases Did Not Develop Epidermoid Carcinoma—
  55 cases alive and well
  26 cases died of unrelated disease
    12 cardiovascular
    7 cancer
      2 each lung and liver
      1 each breast stomach and soft tissue (sarcoma)
    7 miscellaneous (cirrhosis tuberculosis asthma etc)

*See text.

Case 5 S W a 57 year old white male was first seen in May 1951 having been hoarse for six to eight years Laryngoscopic examination showed thickening keratosis of the posterior third of each cord more marked on the left Biopsies done in May and December of 1951 and November of 1953 showed epithelial hyperplasia minimal hyperkeratosis and moderate atypia (see Fig 7) No therapy was given In April 1959 he was seen with an exophytic mass on the posterior half of the left vocal cord This was biopsied and diagnosed as invasive squamous carcinoma (see Fig 8) He received irradiation therapy and has a functional larynx to date

Case 3 is different from Cases 4 and 5 Although the development of carcinoma is beyond the three-month time limit for cases designated as a "missed carcinoma" its size, extent

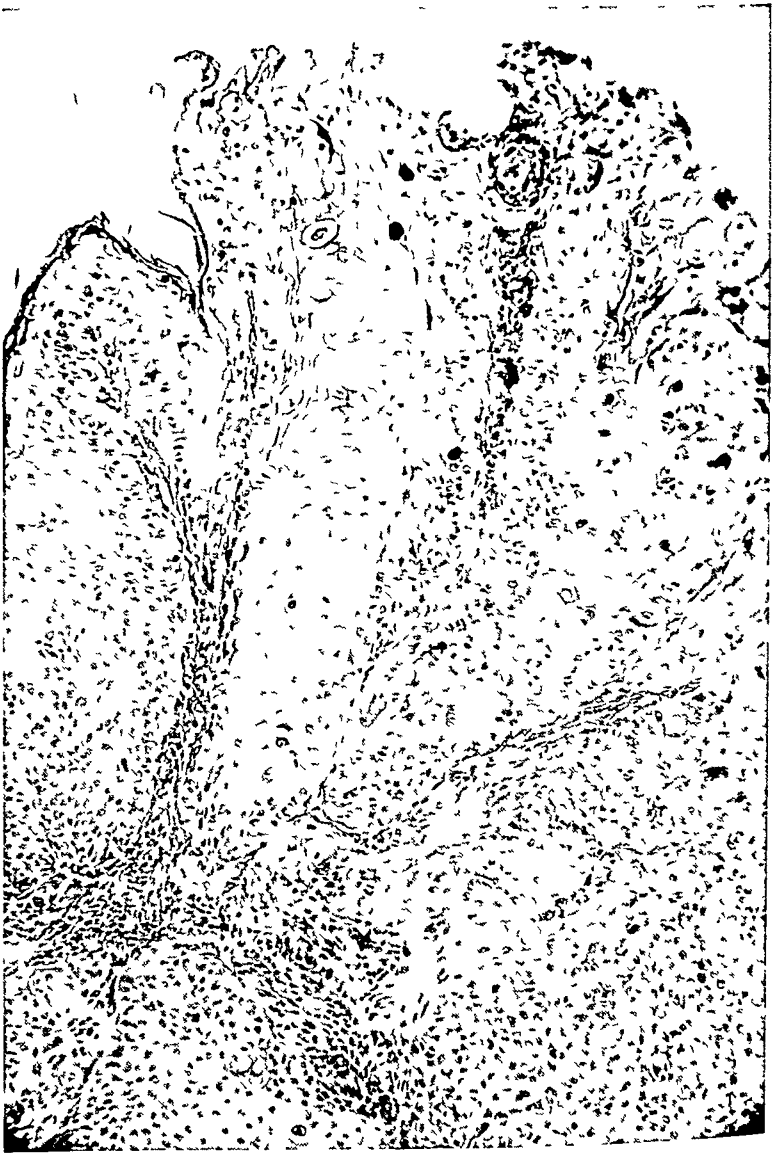

Fig 8 Same case as Fig 7 This invasive well differentiated keratinizing carcinoma was found on biopsy eight years later W U Ill 59-3521A (X115)

and rapid evolution to death in spite of therapy are in favor of its having been cancer from the onset In addition, the size of the initial biopsies was not what we consider adequate at the present time for a laryngeal lesion of the extent initially described This is the only case of this nature in this series and emphasizes the need for adequate sampling, however, we prefer to classify this case as one of carcinoma developing in a larynx with hyperkeratosis

Cases 4 and 5, both in the HKA histologic group, may be considered in three possible ways The first is that they also represent carcinoma present from the onset but overlooked for a period of six to eight years This is most unlikely in view of the repeated examinations and biopsies and the natural history of laryngeal cancer A second possibility is that the sequential appearance of these lesions is purely coincidental Finally, there is the possibility that isolated keratosis is truly precancerous and that in some way as yet unknown, carcinoma arises from the keratotic lesion In order to support this last possibility it would be necessary to demonstrate that the number of cases of carcinoma of the larynx encountered in a group of patients with laryngeal keratosis represents a significantly increased incidence when compared to a suitable control group

Attempts to analyze our data statistically yielded no conclusive information in that we were unable to prove that the patients with keratosis, as a group had the same laryngeal cancer incidence as the general population Nor could we prove that the incidences differed significantly

The analysis failed because the minimum requisite statistical data were not available *i e* we do not know the true incidence of keratosis in the general population or at autopsy, and we have too few cases of keratosis followed by cancer The statistical relationship between keratosis and carcinoma of the larynx will not be known until considerably larger numbers of patients with keratosis are studied with long-term follow-up and until accurate data related to the incidence of laryngeal carcinoma in a suitable control population become available

DISCUSSION AND COMMENT

The recent attention given to the treatment of premalignant lesions and early cancer by ablative surgery or irradiation therapy[8,13,15,18] as well as the popular notion of the precancerous nature of laryngeal keratosis[1,4,6,10-11] should at least be supported by the demonstration of an appreciable risk of malignancy attendant upon conservative management, further, it should be shown that the risk of mortality, morbidity and loss of function attending any proposed therapy be less than the risk of associated or subsequent malignancy. Ultimately it would be desirable if some rational cause and effect relationship could be established between the proposed "premalignant state" and invasive cancer. In our present state of ignorance regarding the pathogenesis of cancer this last requirement, at least for keratosis and epidermoid carcinoma of the larynx, is impossible to establish. The influence of occupational irritants, chronic laryngeal inflammation, voice abuse, smoking, alcohol intake, vitamin deficiencies and structural laryngeal abnormalities, all factors considered of importance in relation to keratosis provide, as yet, no rational causal explanation for the development of carcinoma.

In our opinion the premalignant nature of keratosis and the metamorphosis of keratosis to cancer are based in the main upon repetition of previously reported case material with an increment each time of a few more clinical cases. These studies certainly raise the question of a premalignant state, but they hardly serve to provide the requisite clinical proof.

In the absence of statistical evidence of the malignant potential of laryngeal keratosis, clinical judgment must rest upon the results of series of consecutive cases treated conservatively. The observations of Putney and O'Keefe[18] on 68 cases of keratosis are most pertinent. Of their 68 patients, 27 later developed carcinoma. Of these 27, six were found within six months. They consider these six cases as carcinoma from the start and these cases are comparable to the three in our group in which cancer was evident within 2.5 months. Having excluded these six, 62 cases treated conservatively remained and of these 21 developed carcinoma over a period ranging up to ten years, ten within two years and 11 thereafter

The ratio of cases eventually developing cancer to total cases (21 of 62) in the Putney and O'Keefe series does not compare favorably with the results of the present series (three of 84) We have not found any important differences between these two groups of patients with keratosis to explain this difference The age range, mean age, sex ratio and mean duration of laryngeal symptoms are approximately the same Perhaps some unknown patient selection factors and differing pathologic criteria are responsible for the observed variance

In our experience conservative therapy has yielded most gratifying results With careful cooperation between the laryngologist and pathologist the risk of missing cancer in any of its stages is very small Our evaluation of these results is that the risk of subsequent carcinoma in carefully followed patients with keratosis is so small as to obviate the necessity of prophylactic anticancer treatment This evaluation finds additional support from the following considerations Suppose the most advanced and recent thinking on the therapy of keratosis[15] were utilized in our series of cases This would call for laryngofissure for recurrent keratosis limited to one vocal cord, irradiation therapy for recurrences involving both vocal cords and laryngectomy for irradiation failure and recurrences with extensive papillomatous involvement of the larynx Pachydermia laryngis and posterior commissure lesions would be treated conservatively Of the 25 cases in our series with proven recurrent keratosis (see Table VI), five would have had laryngofissure, 11, irradiation therapy and four would be considered candidates for laryngectomy We are unable to say that this treatment plan would have prevented a single case of laryngeal cancer None of the cases that would have had surgical therapy subsequently developed carcinoma and ten of the 11 candidates for X-ray therapy are also free of cancer One case (Case 5) might have benefited from X-ray therapy in that his recurrent keratosis might have disappeared, however, irradiation therapy does not help all such cases[13, 14, 15] and there is no evidence that such therapy actually prevents the subsequent appearance of malignancy These views are made even more compelling when one realizes that the X-ray therapy actually given for the true cord carcinoma that appeared in this case (Case 5) would have been an impossible therapeutic consideration had prior irradiation been given for keratosis, and the surgical risk *of morbidity in operating in an irradiated field* is also increased Finally, emphasis should be directed to the fact that the proposed therapy would not have prevented two of three cases of carcinoma (Cases 3 and 4) in this series because these two patients did not have a recurrence of keratosis after nine months and eight years respectively, but rather the reappearance of hoarseness heralded the presence of invasive carcinoma

Conservative therapy, in the form of investigation and exclusion of irritant factors known to be associated with keratosis, adequate laryngeal examination at properly timed intervals together with careful biopsy and stripping of the involved mucosa, has proven adequate in all cases The appearance of cancer in patients of this series with atypical epithelial alterations is some indication that these patients should be followed with greater care

Several well defined lesions and some less well categorized conditions are associated with hyperplasia and keratinization of the larynx, particularly the vocal cords Of these chronic hypertrophic laryngitis, squamous papillomas, laryngeal vocal nodules and squamous carcinoma are more frequent than isolated keratosis The gross differentiation of these lesions is not difficult for the experienced laryngologist, and the microscopical appearance is characteristic The exception is the gross differentiation of isolated keratosis from well differentiated keratinizing squamous carcinoma Here the laryngologist must rely on the pathologist's interpretation of the biopsy and the pathologist must accept the responsibility of his diagnosis Equivocal diagnoses, such as "almost a keratinizing squamous cell carcinoma," "epithelial papillosis, perhaps malignant" and "very like cancer,"[13] are of no help to the laryngologist They serve only to create a climate of confusion in which every pathologic diagnosis of keratosis is viewed with suspicion and apprehension Even worse, they may force him to the use of radical therapeutic procedures for a manifestly benign condition When close cooperation exists between a laryngologist who takes accurate biopsies and an experienced pathologist who makes a careful study of the material, the error in assessing the true nature of the laryngeal lesion is sufficiently small to place confidence in the use of conservative measures in the management of isolated keratosis

SUMMARY

Eighty-seven individuals with clinical and histological findings of laryngeal keratosis have been followed for a minimum of five and a maximum of 15 years The age, sex, anatomical and pathological characteristics of the group are defined

Three individuals were found on subsequent biopsy, within three months, to have invasive carcinoma These are considered to have had carcinoma from the onset that was missed by the first biopsy They represent an incidence of less than 0 5 per cent for this "error" among the 470 cases of laryngeal carcinoma diagnosed during the time under study

Of the remaining 84 cases, three developed laryngeal carcinoma at intervals of nine months, six years and eight years respectively after the diagnosis of keratosis One of these three patients died of laryngeal carcinoma Two are alive and well without evidence of persistent disease one following laryngectomy and one following irradiation therapy

Eighty-one patients did not develop laryngeal carcinoma Of these 55 are alive and well to date while 26 died of unrelated diseases Of the 25 cases with biopsy proven persistence of keratosis and the 36 with symptomatic persistence, one developed laryngeal carcinoma

These findings do not support the prevalent concept that laryngeal keratosis is a distinct precancerous condition Analysis of the data does not support the institution of prophylactic anticancer therapy, *e g* , laryngofissure, irradiation and laryn-

does not help all such cases[13,1 18] and there is no evidence that such therapy actually prevents the subsequent appearance of malignancy These views are made even more compelling when one realizes that the X-ray therapy actually given for the true cord carcinoma that appeared in this case (Case 5) would have been an impossible therapeutic consideration had prior irradiation been given for keratosis, and the surgical risk of morbidity in operating in an irradiated field is also increased Finally, emphasis should be directed to the fact that the proposed therapy would not have prevented two of three cases of carcinoma (Cases 3 and 4) in this series because these two patients did not have a recurrence of keratosis after nine months and eight years respectively, but rather the reappearance of hoarseness heralded the presence of invasive carcinoma

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THE JAMES E NEWCOMB AWARD

Dr Joel J Pressman, of Los Angeles was chosen by the members of the American Laryngological Association at its annual meeting recently to be the recipient of the James E Newcomb Award for his outstanding contribution to research in otolaryngology.

gectomy for patients with laryngeal keratosis The theoretical application of these measures to our series shows that not a single case of cancer would definitely have been prevented and emphasizes the risk of morbidity and laryngeal dysfunction attending such procedures

Conservative therapeutic measures, careful follow-up laryngeal examinations with biopsy as indicated have proven adequate in the patients studied

The morphologic alterations are classified, defined and illustrated Individuals with atypia as well as keratosis have a higher incidence of persistence and should be observed more closely

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or mold. Others have followed, using various materials as molds or stents to preserve the continuity of the pharynx with the esophagus until the graft was secure. Pressman,[5] experimentally in animals, has used the lyophilized aorta as a homograft to close this defect with very interesting results. Som[6] has used a portion of the upper trachea and larynx with partial skin grafting to close the defect produced by surgery when the pharynx and a portion of the cervical esophagus was removed. Any lesion involving the pharynx and requiring the removal of more than 70 per cent of its circumference is more than likely to end with a stricture or incomplete healing.

The original tube stent was first made by Negus,[7] of polyethylene tubing, and later the irregular shaped tube conforming more to the shape of the pharynx and laryngopharynx was made of latex rubber and used by Shaw and Ormerod[8] rather extensively. Conley[9, 10] has used both the tantalum wire mesh, tubal plastic stent and also woven nylon stent in a number of cases reported with success. Much the same results have been obtained by others who have attempted to reconstruct the pharynx and cervical esophagus when either the complete circumference was removed or a sufficient amount so that primary closure was thought impossible.

Briefly before reviewing the cases operated, I will discuss the technique used. In all cases the skin was removed from the anterior surface of the thigh, where the least hair bearing surface was found. This was taken with the electric dermatome. An attempt was made to cut the graft 18/1000 of an inch in thickness in all cases. Then the skin was trimmed to fill the gap between the pharynx and esophagus. In most of the cases an Edwards Tapp arterial graft was used as the stent. The skin graft was sutured first to the remaining strip of mucosa which was nearest the mid-line in the partial pharyngectomized patients, and then to the entire circumference of the pharynx above and the esophagus below. In case of the completely pharyngectomized patients it was placed on the tissue over the anterior longitudinal ligament, sutured above to the complete circumference of the pharynx and below to the esophagus. The Edwards Tapp tube was

SKIN GRAFTING IN PARTIAL AND COMPLETE LARYNGO-PHARYNGECTOMY AND CERVICAL-ESOPHAGECTOMY *†

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Houston, Tex

When dealing with large advanced carcinomatous lesions of the laryngopharynx and adjacent cervical esophagus, one must assume the attitude of an optimist, for at best the five year survival rate is poor with either surgery, or irradiation If one can count either as definitive, surgery would certainly be the choice The extensive surgery that must be done is not without its complications and hazards As to life of the patient, the ultimate poor results are not gratifying

When the continuity of the pharynx and esophagus is interrupted in order to remove all the cancerous tissue, some type of reconstruction must be done in order to form a new gullet for the passage of food and saliva Prior to the antibiotic days this was almost an impossibility, though Trotter[1] mentioned it with much enthusiasm in 1913 Esser,[2] in 1917, first described the principle of inlay grafting of split thickness skin supported by a solid mold or stent for mouth lesions The use in the laryngopharynx had to await the advent of chemotherapy and antibiotics for its success Wookey[3] established the really first successful method in his two stage procedure in 1948 By the use of skin flaps and reconstructive procedures he was able to form a new pharynx This method was often fraught with complications, temporary pharyngostomes and sometimes weeks of hospitalization In 1950 Owen and Negus[4] were the first to reconstruct the pharynx and cervical esophagus in a one-stage procedure by means of a free split thickness graft held in place by a stent

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*Read at the meeting of the Southern Section American Laryngological Rhinological and Otological Society, Inc New Orleans, La Jan 15 1960

†From the Department of Otolaryngology Baylor University College of Medicine and Affiliated Hospitals

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good health by eating well masticated and pureed foods The opening remains at about 16 to 18 F

In the other case, a fistula developed the tenth day, but, because of the increasing size of the fistula the mold was removed in four weeks The mold did not slip, and the fistula closed one month after the mold was removed This patient expired six months after surgery, of cardiac decompensation No clinical evidence of carcinoma could be seen at the time of death, although a post-mortem was not obtained

The third case was an extremely extensive lesion involving the larynx, laryngopharynx and the cervical esophagus The esophagus was removed down to the level of the suprasternal notch and the trachea at approximately the same level In addition, a bilateral thyroidectomy was done *en bloc* with the laryngopharynx and cervical esophagus This patient expired on the seventh day as a result of aspiration of vomitis A post-mortem examination revealed that only about 30 per cent of the graft had taken This patient was extremely emaciated and in very poor condition for surgery.

The five cases of laryngectomy and partial pharyngectomy, some with radical neck dissection, will be taken up in two groups those without irradiation, and those with irradiation prior to surgery

There were three cases in the first group all with large pyriform sinus lesions and direct extension beyond the cartilaginous confines of the larynx In only one was there a metastatic gland in the upper cervical chain All the cases were operated by total wide field laryngectomy, removal of the lobe of the thyroid gland and radical neck dissection incontinuity on the side of the lesion In all, the lesion involved the aryepiglottic fold and extended well down to the lower limits of the pyriform sinus, so that the lower margin of excision was at the level of the lower border of the cricopharynges, thus including a very small portion of the cervical esophagus The major part of the defect in all of these cases was approximately 8-9 cm, with only a small strip of mucosa one to one and one-half centimeters remaining connecting the esophagus with the pharynx This was not considered adeleveled below by removing a wedge so that it would fit better into the esophagus After the stent has been placed so that it was well above the upper suture line, it should also extend at least one to two centimeters below The complete closure of the graft was accomplished by suturing it to the opposite side of the mucosa in the partially pharyngectomized patients and suturing the edges of the free graft together to complete the closure in the completely pharyngectomized patients In all instances 3-0 catgut was used The sutures were placed as close together as was necessary to close any defect and to obtain a water tight closure The over-lying skin was then closed in the usual manner with two rows of sutures A small drain was placed away from the graft on each side and a light pressure dressing applied A feeding tube had already been introduced through the stent into the stomach Eight cases were managed and repaired by the afore-mentioned technique

The three cases of laryngo-pharyngectomy and cervical-esophagectomy will be discussed as a group The first case was in very good condition and presented an almost complete annular lesion in the region of the cricopharyngus or slightly above The other two cases had rather large lesions of the laryngopharynx, the pyriform sinus and extending into the esophageal area Both were poor surgical risks having lost considerable weight as a result of dysphagia and also presented the problem of laryngeal obstruction due to the extensive encroachment of the lesion into the supraglottic larynx In the first case and in one of the more advanced cases a tubular plastic stent was used approximately 2 cm in diameter Both cases developed a fistula One had the stent removed at 14 days and the other at one month

In the first case, the smaller cricopharyngeal lesion, the fistula closed in approximately two weeks The patient has remained well for five years and ten months The stent was ejected partially into the oropharynx the seventh day but was not removed until the fourteenth day as mentioned above Though she has a small stricture which has been dilated several times (none recently), she has been able to maintain good health by eating well masticated and pureed foods The opening remains at about 16 to 18 F.

In the other case, a fistula developed the tenth day, but, because of the increasing size of the fistula the mold was removed in four weeks The mold did not slip, and the fistula closed one month after the mold was removed This patient expired six months after surgery, of cardiac decompensation No clinical evidence of carcinoma could be seen at the time of death, although a post-mortem was not obtained

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There were three cases in the first group all with large pyriform sinus lesions and direct extension beyond the cartilaginous confines of the larynx In only one was there a metastatic gland in the upper cervical chain All the cases were operated by total wide field laryngectomy, removal of the lobe of the thyroid gland and radical neck dissection incontinuity on the side of the lesion In all, the lesion involved the aryepiglottic fold and extended well down to the lower limits of the pyriform sinus, so that the lower margin of excision was at the level of the lower border of the cricopharynges, thus including a very small portion of the cervical esophagus The major part of the defect in all of these cases was approximately 8-9 cm , with only a small strip of mucosa one to one and one-half centimeters remaining connecting the esophagus with the pharynx This was not considered adequate for closure because of the great length of the defect beyond this strip of mucosa In all of these cases a split thickness graft was draped over a three-fourth inch Edwards Tapp tube and sutured in place as described in the technique Two of these cases recovered without surgical complications In one a fistula developed the sixth day, when the patient drank a full glass of water Some of it leaked around the tube and extravasated beneath the skin over the side of the neck where the radical neck procedure had been performed It was necessary to remove the stent on the seventh day because of infection, and a fistula developed just above the tracheal stoma

In the other two cases, without complications, the stent was allowed to remain in place for 21 days In the case where the fistula had developed, a secondary closure was accomplished at the end of two months All of these cases have had local recurrence one at the end of six months, one at the end of ten months, and one at the end of one year Swallowing was normal in all cases after removal of the stent, or when the fistula was closed in the one case The swallowing remained good even after evidence of recurrence Two of the cases had recurrence from involved glands in the recurrent laryngeal nerve chain or from lymphatic extension into the esophageal or tracheal area

In one the recurrence was higher up in the pharynx All were treated with X-rays following evidence of recurrence Two have died of hemorrhage six months and one year after irradiation, and one is still living and free of clinical evidence of disease six months after irradiation

In the latter group the two cases were treated with X-rays prior to surgery at the request of the patient Both of these patients had large pyriform sinus lesions and should have been treated initially with surgery Both had evidence of irradiation necrosis with extension of the tumor into cartilage In both, a small strip of mucosa was left remaining, approximately one to one and one-half centimeters in width, with the greater defect approximately 8-9 cm between the pharynx and the cricopharyngeal area In one, a laryngectomy was done, with a partial skin graft of the area Infection supervened, and a large fistula developed The stent was removed two weeks later Pathological study of the specimen showed no evidence of tumor The large defect after laryngectomy was necessary because of the tremendous fibrosis and necrosis which had occurred due to irradiation This patient died seven months after surgery, due to direct extension from a metastatic gland beneath the clavicle on the right side This became ulcerative and infiltrative, and the patient expired of hemorrhage The pharyngostome which had developed did not close Post-mortem examination showed no evidence of metastatic carcinoma in other portions of the body

In the other case, a laryngectomy, removal of the lobe of the thyroid and radical neck dissection on the side of the lesion was done The defect was larger than in the previous case, and skin grafting was done over a three-quarter inch Edwards Tapp tube, as in the previous case A complete take of the graft occurred, it was removed in three weeks, and swallowing was normal Two months after surgery a small fistula appeared on the side of the radical neck dissection, and a hemorrhage from the carotid artery occurred Ligation of the artery was done above and below the rupture, and three days later this patient developed hemiplegia with marked cerebral damage A large pharyngostome developed following surgery At surgery it was found that the graft had taken amply but had been placed over the carotid artery, and no doubt infection in this area had supervened causing the hemorrhage of the carotid artery The patient expired of hypostatic pneumonia several months later Post-mortem examination showed no evidence of recurrence of carcinoma locally nor was there evidence of distant metastasis

ANALYSIS OF CASES AND CONCLUSIONS

A review is given of eight cases, where skin graft to the pharynx and cervical esophagus has been made either because of a complete interruptation of the pharynx with the esophagus or because of insufficient remaining strip of mucosa for adequate closure

It is apparent that in view of the high rate of recurrence, whether it be from insufficient excision of mucosa where lymphatic spread may have taken place, or from minimal metastasis into the recurrent laryngeal chain of glands and lymphatics, a wider excision is to be considered in all future cases Due to the fact that recent recognition of spread along nerve sheaths is a distinct possibility, the recurrent laryngeal nerve should be removed as far down as possible

In all cases the use of 18/1000, thickness of skin was used This is of sufficient thickness that it does not tear easily when sutured and yet is thin enough that minimum infection does not destroy the graft A three-fourth thickness graft has been recommended by some, but it is likely that minimal infection would destroy part of the graft and contracture would be practically as much as with the thinner graft

A good take of the graft apparently was achieved in all but two cases, one a result of extreme malnutrition and infection, although about 30 per cent of the graft did take well by the end of seven days, in the other, there apparently was no take due to irradiation necrosis and infection Apparently the time of sojourn of the stent in the pharyngeal area does not affect the take of the graft, as those that remained only one or two weeks did as well as those which remained longer Some have recommended that the stent remain in place six months This hardly seems practical to me The greatest length of time that the stent was left in place was four weeks In two cases of complete pharyngectomy, a stricture occurred, one in which the patient lived to eat well masticated foods at the end of five years, ten months, in the other, three dilations were sufficient to maintain a good lumen until death occurred six months after surgery

A rather rigid tube was used in the first three cases, and in one, the stent was ejected against the soft palate about 14 days Of the latter five cases the Edwards Tapp arterial graft was used as a stent This is completely elastic and is not rigid The lower end must be tapered so as to fit in the cervical esophagus below the lower circumferential suture line The Edwards Tapp tube is thought to be superior to any other stent because of its elasticity and pliability and does not slip as easily as the smoother plastic tubes It may be made more rigid by dipping it in Vinyl plastic My experience with the Negus stent is limited to one case which was terminated by death, and no conclusion can be drawn

Only four cases operated upon were good surgical risks, yet the operative mortality rate was only 15 per cent While this is relatively high it does not seem extremely so when considering the condition of these patients at the time of surgery

Finally it must be admitted that skin grafting for the preservation of an ample tube for swallowing in cases of partial or complete laryngo-pharyngectomy and cervical-esophagectomy is not without its complications and hazards It seems quite apparent that there was less hazard and fewer complications when the general condition of the patient was good and little or no evidence of stricture when a small strip of mucosa was left, supplemented by the skin graft

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907 Hermann Professional Building

THE PROCUREMENT AND PROCESSING OF ENDOSCOPIC BIOPSIES*

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KENNETH C JOHNSTON, M D ,†
and
EDWIN F HIRSCH, M D ,‡
(By Invitation),

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The closest cooperation between endoscopist and pathologist is essential if maximum information is to be obtained from endoscopic examinations The endoscopist in the practice of his specialty requires, probably more than any other specialist, the technical services and the professional opinion of the pathologist The purpose of the endoscopic examination is largely for diagnosis This means that all secretions and biopsy tissues taken from the air or food passages during a laryngoscopy, bronchoscopy or esophagoscopy must be processed for microscopic examination by the pathologist The small volume of the secretions and the limited size and total amounts of the biopsy tissues establish restrictions in the processing techniques Stated in another way, the processing procedure must conserve all of the secretions or tissues obtained by the surgeon, and safeguard them against loss, especially of a solitary particle that may have diagnostic value Limitations as to the size and amount of biopsy material obtainable are due to several factors The anatomical limits of the surgical field available to the peroral endoscopist necessitates employing small instruments which often can, at best, procure only fragments of tissue or a few cubic centimeters of significant

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*Read at the Sixty-Third Annual Meeting of the American Laryngological Rhinological and Otological Society Inc Miami Beach Fla March 16 1960

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secretions, furthermore the lesion itself may be small in total volume Its complete removal with a surrounding area of apparently normal tissue, favorable for histologic diagnostic studies, may permanently impair function should the lesion be benign This is particularly true of laryngeal biopsies in the so-called border-line lesions of keratosis or leukoplakia when an adequate biopsy following a previous procedure and a report of "insufficient tissue," may result in permanent hoarseness, nevertheless, satisfactory results are realized when endoscopic techniques are closely integrated with the processing procedures required for the preparation of the secretions The following work arrangements between the bronchologist and the pathologist have been used with advantage for many years

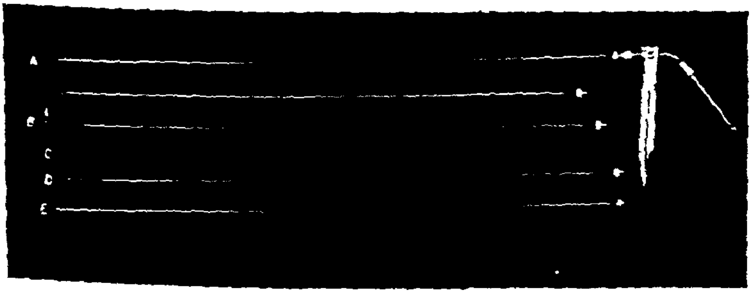

Fig 1 Bronchoscopic aspirator with centrifuge collecting tube Straight or curved flexible aspirators and straight open-end tips are interchangeable

Secretions

Secretions are aspirated directly into an ordinary, tapered 15 cc glass centrifuge tube The aspirators are so constructed that the material can be drawn into the centrifuge tube through a two-hole rubber stopper onto which the tube is fastened with a clamp (see Fig 1) A thumb valve on the aspirator permits intermittent suction and allows release of suction should the tube suddenly fill with secretions, thus avoiding loss of material into the suction apparatus Straight or curved rigid or flexible aspirating tubes of appropriate size may be attached to the collecting tube This collecting system is simple, efficient and sufficiently variable in size to permit its use in any of the peroral endoscopic examinations, whether the patients are adults or children

Generally, the first specimen obtained is sent to the laboratory for bacteriologic study The centrifuge tube is removed, and a second tube is fixed to the two-hole stopper of the aspirator and another specimen is collected which will be processed for histologic examination A saline wash of the bronchus may be necessary if there is insufficient secretion for the second collecting tube On completion of the endoscopic examination, Zenker's solution* (or other fixative solution) is added to the second tube, the solution being drawn through the aspirator in order to include any residue in the

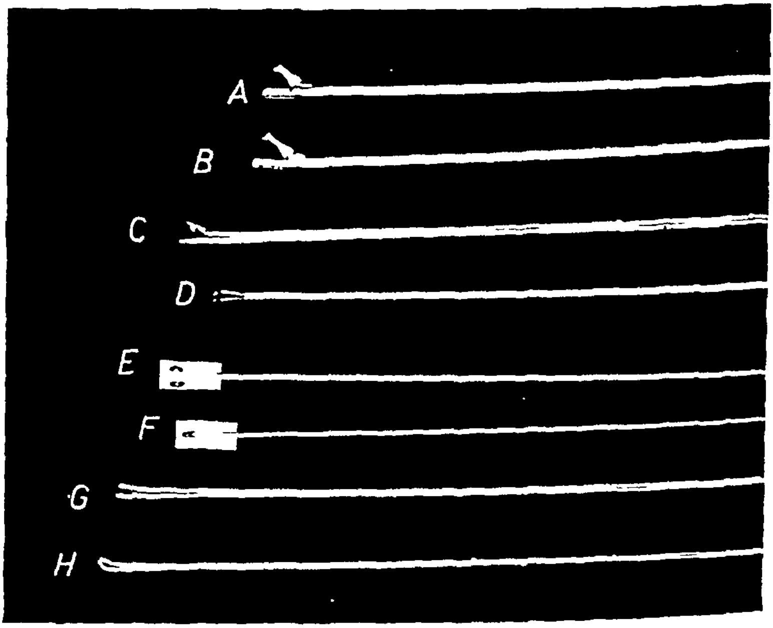

Fig 2 Endoscopic biopsy forceps The small size of the biopsy tissues requires special processing in order to preserve every fragment for histologic analysis

laboratory specimen Both tubes are closed with rubber stoppers, and a strip of surgical tape with the name of the patient is attached to each tube and the tubes with the requisitions are sent to the laboratory

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*Zenker's solution (without acetic acid) Distilled water 1 000 cc mercuric chloride, 50 grams potassium dichromate 25 grams sodium sulfate, 10 grams

The handling of the specimen for bacteriologic examination is rather standard, with smears, cultures, sensitivity determinations and animal innoculation or special fungus studies requested according to the clinical needs of each patient The contents of the second aspiration collecting tube are processed as biopsy material in the manner described below, for cell-block study

Biopsies

Each endoscopist has his own preference in regard to biopsy forceps (see Fig 2) For verrucous tumors almost any forceps will give adequate, representative tissue The straight and angulated cup forceps of appropriate length (see Fig 2 G, H) and the globular-objects foreign-body forceps (see Fig 2, D, E, F) are most commonly used Cutting biopsy forceps require frequent adjustment and sharpening, and these must be used in tougher, fibrous tissues (see Fig 2, A, B, C) Their construction should be such that the cutting blades continue into the angle of the jaws of the forceps to prevent leaving a strip of uncut tissue in the closed jaws Basket types of forceps have the advantage of reducing the tissue crushing action of forceps with solid blades The small telescopically guided forceps are still not entirely satisfactory, but positive biopsies can be obtained if all of the fragments of tissue collected in these tiny blades are preserved

Routinely, particles of biopsy tissues are placed at once in a tapered 15 cc centrifuge tube (see Fig 3) A cubic centimeter of saline solution can be placed in the tip of the tube to aid in releasing the tissues from the instrument Zenker's solution (or other fixative solution) is added in sufficient amounts, the tube is stoppered, labeled properly and sent to the laboratory Zenker's solution precipitates the formed and dissolved tissue elements in the secretions and properly fixes the biopsy tissue particles system is simple, efficient and sufficiently variable in size to permit its use in any of the peroral endoscopic examinations, whether the patients are adults or children

Generally, the first specimen obtained is sent to the laboratory for bacteriologic study The centrifuge tube is removed, *and a second tube* is *fixed* to the two-hole stopper of the aspirator and another specimen is collected which will be *processed for histologic* examination A saline wash of the bronchus may be necessary if there is insufficient secretion for the second collecting tube On completion of the endoscopic examination, Zenker's solution* (or other fixative solution) is added to the second tube, the solution being drawn through the aspirator in order to include any residue in the

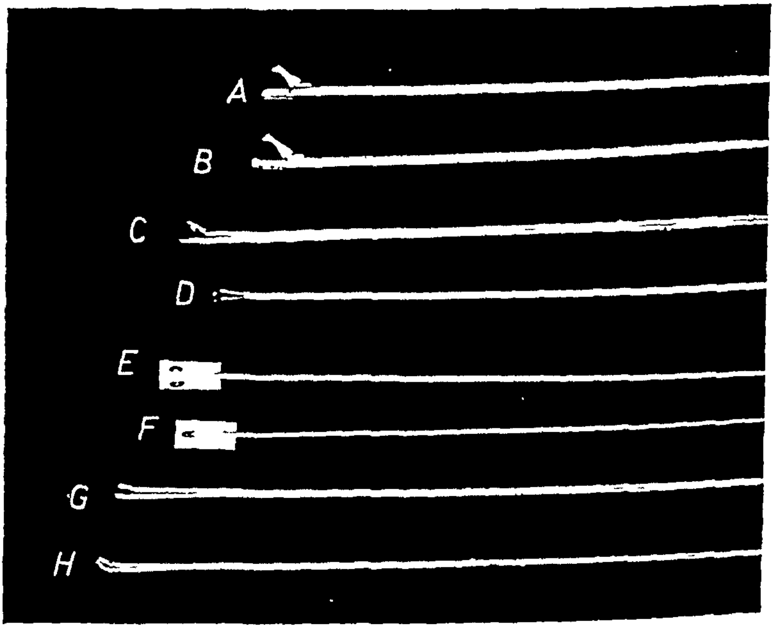

Fig 2 Endoscopic biopsy forceps The small size of the biopsy tissues requires special processing in order to preserve every fragment for histologic analysis

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*Zenker's solution (without acetic acid) Distilled water 1 000 cc mercuric chloride, 50 grams potassium dichromate 25 grams sodium sulfate 10 grams

After two or three hours' fixation, these precipitates or tissues are collected by centrifugation in the tip portion of the tube and the fixative solution is decanted. The entire process of dehydration, clearing and paraffin embedding is carried out in the original centrifuge tube. After each change of water (for washing), 70 per cent alcohol, 80 per cent alcohol, 95 per cent alcohol, two changes of absolute alcohol, two changes of xylol or toluol, the secretion precipitates or tissues are collected in the tip portion of the tube by centrifugation, and the solutions are decanted. Finally, melted paraffin is added to the tube and the infiltration of the precipitates and tissues with paraffin is carried out by placing the tube and its contents in a paraffin oven. Two changes of paraffin suffice. A short piece of flexible wire (copper) with a loop at the end reaching into the melted paraffin is hung over the lip of the centrifuge tube, and the tube is cooled to harden the paraffin. Then the tube is dipped quickly into warm water and by means of the wire, the column of paraffin is

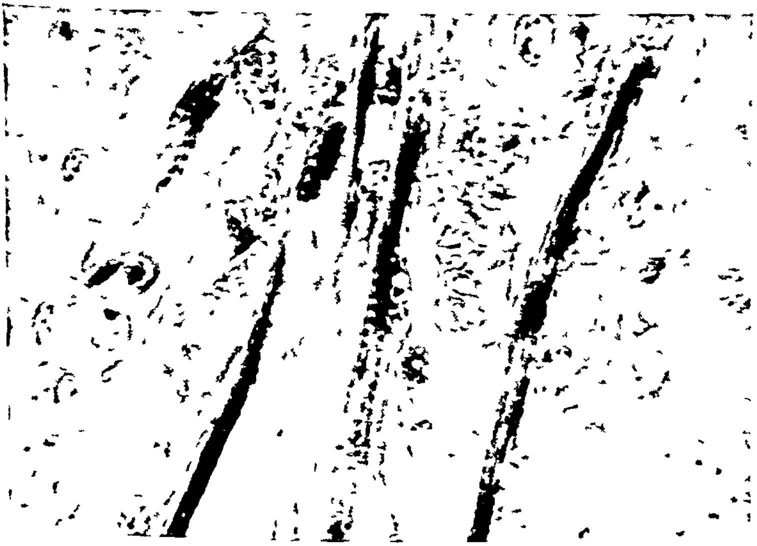

Fig. 4. Rhabdomyosarcoma of the larynx of a five-year-old boy. The advantage of Zenker's fixation over formalin is demonstrated in this photomicrograph of Zenker's fixed tissue.

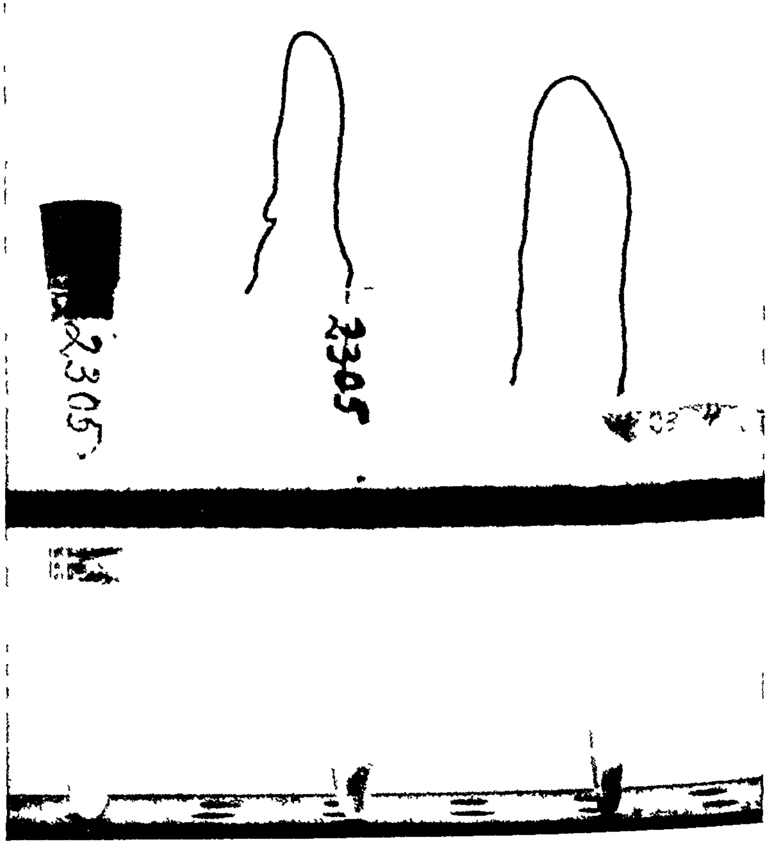

Fig 3 Three steps in processing small endoscopic biopsies Left—The biopsy in Zenker's solution in the centrifuge tube Center—The specimen centrifuged covered with paraffin into which copper wire had been inserted to facilitate handling Right—Button of biopsy material at bottom of paraffin column which will be embedded in block for cutting All tissue fragments obtained are included in the button

Zenker's solution is preferred to other fixatives such as 10 per cent formalin for two reasons first, the formalin has a tendency to shrink tissues, and a significant loss of detail may be associated with its use in handling the small bits of endoscopic biopsy material, second, in unusual tumors requiring further histologic staining and study (see Fig 4), tissues fixed in Zenker's solution permit more favorably such further investigation, whereas those fixed in formalin may no longer be suitable for this further processing

This gives a morphologic pattern often of more diagnostic significance than cytologic smears and improves the percentage of positive biopsies in lesions in which the endoscopic findings are equivocal

SUMMARY

A simple, practical method of collecting and processing endoscopic secretions and biopsies is described All diagnostical material is aspirated or placed directly into centrifuge tubes that are sent to the laboratory without transfer Bacteriologic studies are made from the contents of the first tube Histologic studies are made from the centrifuged contents of the second The preservation in this manner of even the smallest particle of tissue which may be of diagnostic significance is possible The advantages of various types of biopsy forceps in securing tissue and Zenker's solution as a fixative are discussed

700 N Michigan Ave

HUMBOLDT UNIVERSITY OF BERLIN (CHARITE)

The celebration of the 250th Anniversary of the Charité will be held in Berlin from November 6-19, 1960, in connection with the 150th Anniversary of the Humboldt University.

Applications for participation are to be directed to the Committee for the Preparation of the 250th Anniversary of the Charité, Berlin N 4, Schumannstrabe 20-21, c/o Dozent Dr med habil Dagobert Muller, secretary of the committee drawn out of the tube The short cell-block button of infiltrated tissues at the tip is cut off, is embedded in melted paraffin, cooled and blocked for sectioning The secretions are cut serially, enough to cover at least five slides, stained with hematoxylin and eosin and mounted for microscopic examination

In using this collecting and processing technique described, all of the secretion precipitates and the biopsy tissues are

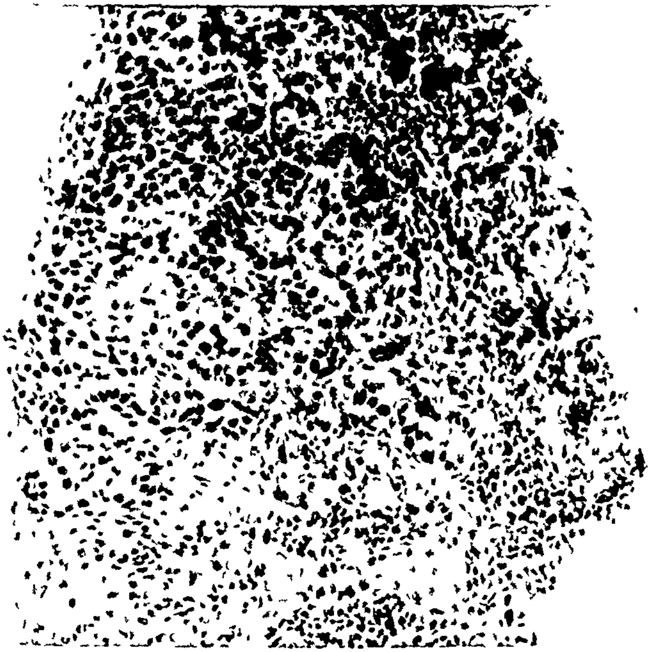

Fig 5 Photomicrograph of bronchogenic carcinoma tissues obtained by aspiration technique There was no visible tumor tissue obtainable by forceps biopsy

conserved without loss, even to the smallest particle which alone may be the important tissue for diagnosis (see Fig 5) The pathologist or his technician does not have to strain out small strands of tissue from the comparatively large volume container often supplied by the laboratory to the operating room Loss during transfer is eliminated Most important, tissues in aggregates of cells are collected by the aspirator cerumen In selection of submarine personnel, it is quite important that body odors be controlled and it is known certain types of cerumen emit unpleasant odors This may be due to certain types of bacteria acting upon the cerumen There is a racial and individual difference in this respect The function of cerumen has many theories as a coating, trapping foreign matter that might enter the canal, also carrying with it epithelial scales and hairs out of the meatus It may prevent dessication of the skin and makes it pliable The antibacterial and fungicidal properties are debatable It is composed of amino acids, potassium, sodium, calcium, magnesium, phosphorus, sulphur, silicon, cholesterol, cerotic acid and neostearic acid and consistently has an acid pH Recently we ran cultures from the external ear of 50 individuals who had never had ear infections of any type The cultures were taken under aseptic technique, the results of which showed bacteria of two groups the micrococcus and diphtheroids These findings are quite consistent with those of other investigators

Cutaneous sensibility in the canal does not seem in any way peculiar Normal response to touch, pain, hot and cold are present, although there seems to be an increased sensitivity to itching at the entrance of the canal The relationship of the autonomic nerves to the cerumenous-gland tubules has been demonstrated Stimulation of the cerumenous glands can be caused by andrenergic drugs such as epinephrine, emotions such as anxiety and fear, mechanical irritation such as scratching, and in climatic temperature variations, bearing in mind that the pilo-sebaceous ear glands are really modified sweat glands

If one understands these anatomical and physiological concepts, he becomes better prepared to approach therapy of the canal, even then it may impose anatomical and physiological peculiarities that will tax his ingenuity to the utmost and restrict the type of therapy used It is an impossible and endless task to review all the therapy advocated in the literature, so general therapeutic rules must be followed It might be interesting to note the drugs advocated three decades ago and compare them to those currently in vogue Among those

EVALUATION OF TREATMENT IN EXTERNAL EAR INFECTIONS[*]

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Corpus Christi, Tex

External ear infections present a dual challenge to the otologist The immediate problem is the relief of pain and then the careful study of the cause and prevention of future episodes To accomplish this, an understanding of the anatomy and physiology of the canal is imperative Meticulous laboratory tests may be necessary, including cultures, sensitivity tests, biochemistry (including blood sugar, P B I) and in selected cases, biopsy This should be correlated with a painstaking history in an attempt to identify the etiologic trigger These basic factors are frequently overlooked in our eagerness to try out new drugs with which clinical experience may be minimal and pharmaceutical propaganda maximum

The external canal is a test tube of skin closely attached to the underlying structures of cartilage and bone with little subcutaneous tissue present, the outer one-third contains a complex system of glands that secrete cerumen and are the key to the door of the canal Their secretion is a mixture of secretory products of two types of glands found in the cartilagenous portion, sebum (from sebaceous glands) and apocrine sweat (from the cerumenous glands), the factors that control this production are not known, but some drugs, emotions, temperature variations, and mechanical irritation stimulate the smooth mucle fibers that produce contraction of the glands and make them secrete more than normal The amount and character of the cerumen seems to vary in different races and certainly in different individuals

In reviewing the literature, it is interesting to find that the Japanese Navy[4,5] had done a great deal of research on

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[*] Read at the meeting of the Southern Section American Laryngological Rhinological and Otological Society Inc New Orleans La Jan 15 1960

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The external ear has a high index of epidermal sensitivity and because of this, most of the antibiotics in ear drops have a very low rate of anaphylaxis Penicillin and sulfonamides frequently produce reactions while neomycin and polymyxin seldom do so The choice of antibiotics should be influenced by direct smears, cultures and sensitivity tests Once the bacterial infection is controlled, antibiotics should be discontinued and the situation re-evaluated The micro-environment of the infection in the host should be carefully studied This should include the predisposing factors such as heat and humidity trauma, allergy, diabetes, overweight, thyroid imbalance, daily habits and emotional problems The occasional use of staphlococcus toxoid may be helpful in preventing recurrence of circumscribed otitis externa (furunculosis) The use of Roentgen ray locally occasionally is of value in relieving itching and pain

The public is better informed on the wonder drugs because of the publicity given to them in poular magazines and television This has caused the individual to lose fear of infection, he generally thinks the wonder drugs will do the job in protecting him As a consequence of this attitude, respect for the medical profession has deteriorated somewhat as the reliance on the newer drugs increases Although there is an increasing danger that the chemotherapy of infection is approaching the ultimate, I believe we are on a firm footing in our treatment of ear infections if we do not lose perspective. Too often we are prone to give antibiotics credit for a cure when other factors have accomplished it

In the foregoing remarks I have given you some of the ideas of treatment and some of the drugs that have been used over the last three decades It started with simple clinical observations and has grown into a more detailed examination that includes more meticulous laboratory studies We must not lose sight of the fact that we are the doctor supervising the treatment of the patient and the otologist should still be the one to handle this type of infection

SUMMARY.

1 Predisposing factors must be considered in external ear

used in the early thirties[3] were thymol, iodine, cresatin, merthiolate, mercurochrome, salicylic acid, boric acid, sodium borate, either alone or combined with alcohol. The fungus ear was quite the thing. This era existed for many years. Then the pseudomonas appeared on the scene and distorted the picture quite as much as the aspergillus.

Research conducted and initiated by the Air Force in World War II at Randolph Field[2,6,8] paved the way for a better understanding of the condition. Extensive investigations including biopsies of the external ear were done. We were reluctant to do this except on very special occasions, but with the advent of antibiotics and increased liability insurance, we were bolder in our approach. From this work has come basic rules of therapy as they apply to the external ear today. In general, the more acute and inflammatory the dermatosis, the milder the medicament. Cresatin[1] which we advocated years ago, now is used only occasionally, because of the sensitivity it produced. The sheltered skin of the ear canal is very susceptible, and one must be extremely discreet in handling it. Careful and meticulous cleansing is paramount whether it is done by irrigating with hypertonic solution, applying suction with a fine cannula, or by a stream of gently blown compressed air. Therapy should be initiated with minimum effective concentration of the drug selected and over-treatment must be avoided, this is a very common mistake. Dryness of the canal is the keynote to success in preventing recurrences. Systemic therapy is a must in many cases, such as the administration of thyroid, regulation of diabetes, and most important, control of nasal allergy. Eosinophils in nasal and pharyngeal secretion are of great diagnostic value. Bacterial and fungous infections are not often the primary etiologic agent but accompany other causative factors. Antibiotics in combination with cortico-steroids are valuable when used topically. It was stated at the Seventh Annual Antibiotic Symposium that innumerable synthetic penicillins were possible, more than 200 have already been prepared. New antibiotics are constantly appearing, such as Declomycin (DMCT), Colistin, Refomycin, Asparomycin, Griseofulvin, and Streptozotocin.

LARYNGEAL TRAUMA*†

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Serious trauma to the larynx is fortunately infrequent, possibly because of the protection offered by the mandible and because of its mobility and elasticity The majority of cases are caused by automobile accidents and are associated with multiple injuries, especially in the maxillo-facial area and intracranially It would be interesting to know the incidence of crushed larynges in fatal automobile accidents, but I have been unable to find statistics on this In the typical case, the immediate problem of respiratory difficulty is solved by a tracheotomy, and the larynx is then forgotten in preference to treating the other injuries Several weeks later difficulty in decannulating the patient arises, by which time the problem of a chronic stenosis is established, and a lumen has to be created through a deformed larynx and mass of scar tissue with only compromising results Early definitive treatment will in most cases prevent these chronic problems and offer better results in regard to the voice and airway than treatment after a stenosis is established

In general, the injuries to the larynx and trachea from external trauma may be grouped as *1* lacerations and stab wounds, *2* crushing injuries, and *3* loss of substance by avulsion or gun blast etc The initial symptoms are dyspnea and hoarseness These may be obvious at first or may develop gradually over a period of a few hours There will usually be some dysphagia from edema in the hypopharynx There may also be hemoptysis and swelling of the neck from subcutaneous emphysema and ecchymosis Severe damage to the larynx occurs in some cases without visible damage to the skin of

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infections, correction of systemic faults may accomplish more than local therapy

2 The ear canal must be kept dry, clean and protected

3 Laboratory reports must be interpreted only in light of clinical data

4 The ear infection should be managed by the otologist

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Suite 14, Medical Center

SIXTH INTERNATIONAL CONGRESS ON DISEASES OF THE CHEST

The Sixth International Congress on Diseases of the Chest will be held at the University of Vienna from August 29 to September 1, 1960

inserted.[1] A rubber core mold would serve the purpose but would have to be changed frequently. An acrylic reproduction with a lumen drilled in would be preferable. This should be kept in for about six weeks.

When there is a closed crushing injury to the larynx, as determined by respiratory obstruction, findings of hematoma, etc., one can be certain that there will be a cicatricial stenosis later unless preventive steps are taken early. An adequate preventive measure may amount to no more than exposing the larynx in direct laryngoscopy and inserting an acrylic mold as described above. If the injury is severe, it may be wise to do a thyrotomy, mold the cartilage fragments in place and secure an acrylic stent in place by tantalum wire. For successful results, it is important to do this as early as possible, preferably within the first 48 hours.

A chronic stenosis may be the end result of the trauma and presents a more complicated problem. Attempts at overcoming chronic stenosis of the larynx probably began with Dr. L. Von Schwetter of Vienna in 1871 when he devised methods of dilatation using metal olives and rubber tubes.[2] O'Dwyer described his method of entubation in 1885. Open operations were also done in those early days and a method of treatment involving a laryngo-tracheostomy and dilatation of the stricture under direct vision was presented by John Winslow. The core-mold method of Jackson was a great development and with sufficient patience, almost any stricture can be cured by this method. Schmiegelow[3] began doing the open operation with excision of scar and insertion of rubber tube stents in 1910 and in 1929 reported 18 successful cases. Shortly thereafter, Arbuckle[4] first reported the use of skin grafting and since then there have been important modifications reported by Negus,[5] Figi,[6] Erich,[7] and LeJeune and Owens.[8] Woodward[9] has described a single-stage method which has been successful in several cases in which he excises the stricture and uses a free graft of bone from the body of the hyoid between the alae of the thyroid cartilage. He then inserts an acrylic reproduction of an O'Dwyer tube into the lumen and secures it there by wire.

the neck, so that absence of discoloration or abrasions may offer false reassurance

The lumen of the larynx and trachea depends upon the integrity of the cartilaginous framework, therefore, it is obvious that when there is fracture of the cartilage and overlapping of the fragments, the lumen will be decreased There is the additional factor of chondritis which further deforms the framework and of scar tissue resulting from torn mucous membranes and hematoma which stenose the lumen sometimes to complete closure Cartilaginous fractures in general become increasingly difficult to reduce after 48 hours because of the rapid development of fibrous tissue and the difficulty in freeing the cartilage fragments from it When the average case reaches the otolaryngologist for definitive treatment, about six weeks have elapsed and any possibility of manipulating the cartilage fragments is gone

TREATMENT

In case of the recently traumatized larynx, the situation will usually be obvious and the need for immediate heroic measures plain A tracheotomy is, of course, the first need as a rule, and this should be as low as possible If, for the sake of expediency, a cricothyroidotomy is done, an elective tracheotomy should be done within the next few hours Respiratory obstruction sometimes develops after a few hours from inflammatory edema, and thus all neck injuries can be potentially a danger to the airway, although it may not be obvious at first If there is any obstruction to the airway early in the course of events, a tracheotomy should be contemplated, because the obstruction can be predicted to increase some over the next 48 hours, also, a properly done tracheotomy will minimize subcutaneous emphysema and the possibility of thoracic complications

In case of an open wound of the larynx, careful cleansing and accurate approximation of the parts are important Cartilage fragments should not be removed if at all possible to support them adjacent to a blood supply If there is any possibility of stenosis later, a mold of some sort should be sutures that go through the rubber mold and are tied externally across the skin incision. A heavy silk suture is also placed through the sponge rubber and brought down through the tracheostomy to be tied to the tracheotomy tube. After ten days, the foam rubber mold is extracted through the tracheostomy. A model for an obturator is then constructed out of an appropriate size rubber tube and dental compound. This obturator must have a lumen, fit into the grafted area, and have a lip that protrudes out through the tracheostomy and still leaves room for a tracheotomy tube. After the model is constructed, the sponge rubber is inserted through the tracheostomy back into its original place until a dental laboratory can construct the obturator in acrylic. After this is obtained, the rubber mold is again withdrawn and the acrylic obturator inserted into its place. It should be left in place for at least six months, and usually this may be done without discomfort or need for special care. If the lumen is still adequate after the obturator is withdrawn, the tracheotomy may be closed. If the tube is left out for about a month, the stoma will close partially and then require only a simple procedure to finish closure. Occasionally, the stoma will be so large as to require multiple stages in order to close it.

Sometimes one finds the trachea completely separated from the larynx and it has to be mobilized and anastomosed. An acrylic stent should be used to prevent stricturing at the anastomosis.

Each case presents a different problem, and it is difficult to follow a set routine on each one. There are many other methods in which the surgeon should be versed in, in order to cope with any situation, and some degree of "*ad libing*" is required in each case.

CASE REPORTS

Case 1. A 23 year old male had worn a tracheotomy tube and had been unable to get any air through his larynx since an automobile accident three years previously. He was referred to the Ponce de Leon Infirmary where examination and X ray showed a complete stricture at the cricoid and upper trachea and a bilateral paralysis of the vocal cords. The tracheostomy was enlarged and the larynx opened. A complete stricture was found with destruction of the anterior half of the cricoid and loss of at least one tracheal ring. The trachea was dissected free downward

On the cases that we have encountered, we have been discouraged with dilatation and with the use of plain polyethylene tubing inserted as stents, but we have had success with Woodward's method of using a hyoid bone graft and acrylic mold and with the use of skin grafting, using a sponge rubber mold to hold the graft in place as described by Figi and modified by Erich

Most cases are best treated by an external approach If a tracheotomy is not already done, it should precede the corrective surgery and be placed as low as possible If it was originally too high, it may be necessary to lower it A general anesthetic may then be given and a thyrotomy done If the stricture is not too long, skin grafting will not be necessary As a rule, after excision of the scar tissue and webs, there will still be insufficient airway due to distortion of the cartilages, in which case we prefer to use the hyoid bone graft as described by Woodward An acrylic tube fashioned after an O'Dwyer entubation tube is inserted and secured in place by tantalum wire A central segment of the hyoid bone is then excised and after being fashioned to proper size is sutured between the alae of the thyroid cartilage or between the cut ends of the cricoid as the situation demands Small holes are drilled in the edges of the graft to hold the sutures

Acrylic stents and molds can be made by any good dental laboratory if they are provided with a model It is very important to specify that a lumen be made, as this adds greatly to the comfort of the patient They rarely cause any reaction and will usually be tolerated as long as is necessary

For the very extensive strictures, especially when the trachea is involved, the skin grafting technique as described by Figi and Erich is an appropriate solution If this type of operation is necessary, the tracheotomy should be large and at least 1 to 2 cm below the stricture The stricture is exposed by a mid-line incision through the larynx and trachea and is excised A split thickness skin graft is then obtained and sutured around a cylindrical piece of foam rubber sponge with the raw surface outward This is then inserted into the area of denuded epithelium and held in place by heavy silk

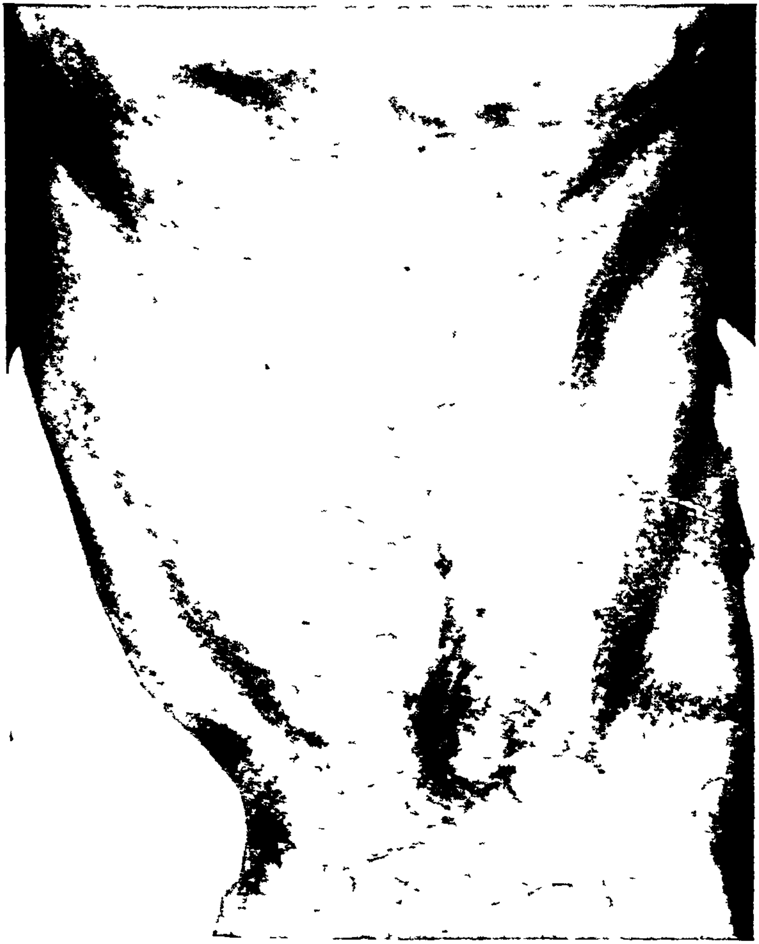

Fig 1-b

Fig 1 a and b This large tracheostomy was closed by inversion of skin edges and a rotating flap

had resumed his job of brick laying He was decannulated about three years ago and a recent communication revealed that he was still doing fine

Case 2 A 45-year-old woman had several procedures done for correction of a bilateral abductor paralysis of the vocal cords She developed adhesions between her vocal cords and remained dependent upon her tracheotomy A thyrotomy was done the adhesions divided and the Woodward technique of grafting a piece of hyoid bone between the alae of the thyroid cartilage and leaving an acrylic stent in the lumen was done The stent was removed after six weeks and she was able to be decannulated a month later Four years after surgery she is still able

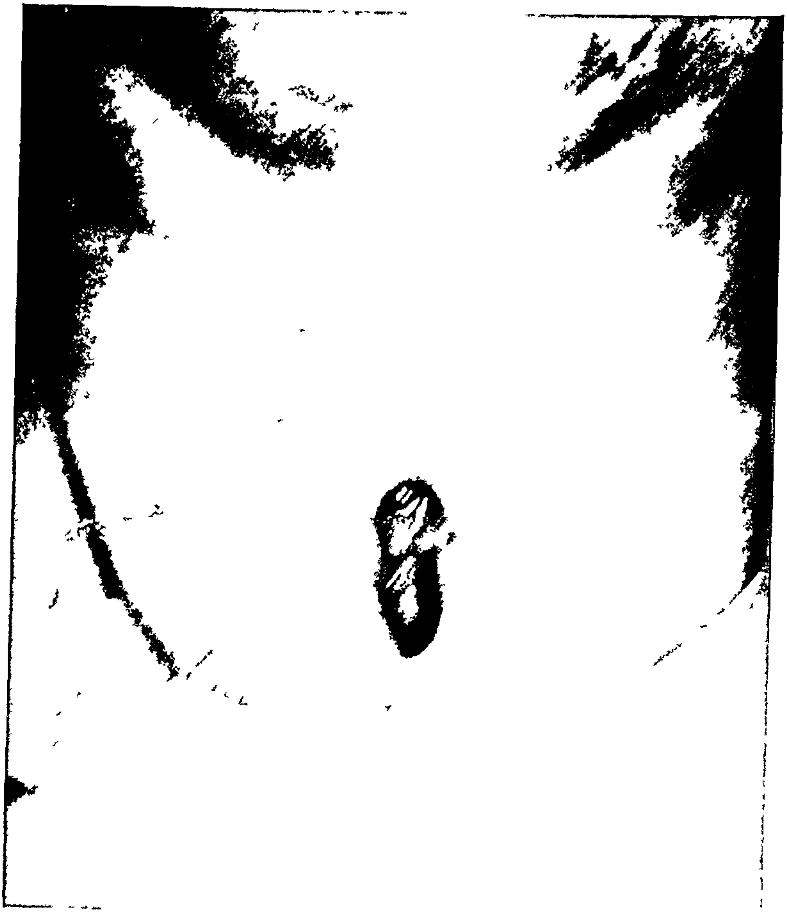

Fig 1-a

brought up and anastomosed to the remainder of the cricoid A section of the hyoid was grafted to bridge over the defect in the cricoid, and an acrylic stent was secured in place by a tantalum wire He was able to talk for the first time in three years After about six weeks he coughed up the acrylic mold The lumen shrank down as would be expected and on reopening of his larynx about two months later he was found to have an epithelized lumen about 4 mm in diameter This was a big improvement on his original state, however The Erich technique of skin grafting was then done after the scar tissue was excised To try to avoid the necessity of an arytenoidectomy later for the paralyzed vocal cords, a portion of the vocal cords was removed submucosally, and the acrylic obturator was constructed long enough to fit between the cords After six months the obturator was removed He had a good lumen, and the vocal cords remained fixed in abduction The large tracheostomy was closed in one stage with inversion of the skin about the edges and a sliding flap A check up one month later showed a good airway, and he

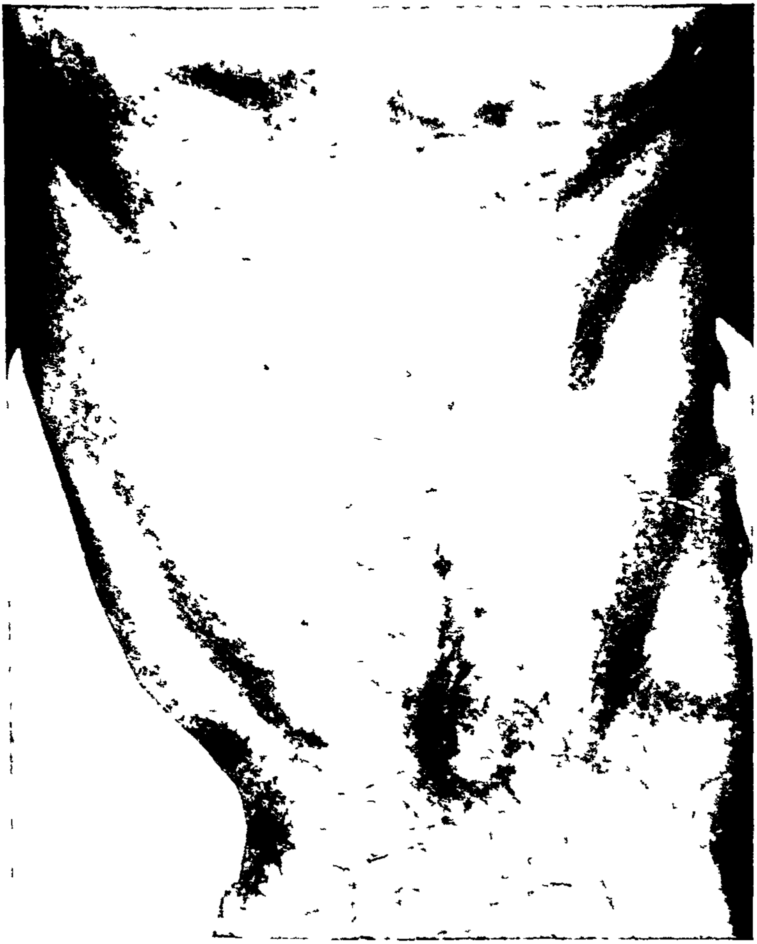

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Fig 2 X-ray showing acrylic obturator in place (Case No 1) after skin grafting

to do well without a tracheotomy I believe she would have more lumen, however, if the stent had been left in place longer

Case 3 A 35 year old female had remained dependent upon a tracheotomy three months following an automobile accident A thyrotomy was done, and there was found to be diminished AP diameter presumably due to fracture of the cartilages The lumen was increased by using a bone graft between the alae of the thyroid cartilages and an acrylic stent was left in place After two months, the stent was removed endoscopically She developed a cutaneous fistula and granulation tissue in the anterior commissure which promptly subsided after removal of a small sequestrum of bone by laryngoscopy. She was then decannulated and the tracheostomy allowed to close spontaneously. Three years after surgery she does well except for some dyspnea on exertion.

Case 4. A 21 year-old male had a severe injury to his neck in an automobile accident—a deep transverse laceration involving the larynx and pharynx was reportedly present. This was closed primarily, and a tracheotomy was done. A sub-dural hematoma and multiple fractures prevented further attention. We saw him about six months after the injury, at which time the tracheotomy was closed up but he was having a good deal of dyspnea on exertion and a very poor voice. Examination showed him to have a markedly decreased glottic lumen with distortion and projection of a sharp ledge into the lumen. A tracheotomy was done, and the larynx was opened up. The projection visible on mirror examination was found to be caused by a piece of cartilage. This was excised and a section of hyoid was removed and grafted in between the alae of the thyroid cartilages after an acrylic stent was secured in place within the lumen. The acrylic stent was removed via laryngoscopy six months later, and he was decannulated a month later. He is much improved, but still cannot do heavy labor, however, he is able to do the job that he was trained for and is doing well.

Case 5. An 18 year old female was involved in an automobile accident about two years ago and required a tracheotomy. She was referred to the Ponce de Leon Infirmary about six weeks later when decannulation was found impossible. Her pathology seemed to be limited to the area of the glottis in the form of posterior webbing and some decrease in AP diameter. A hyoid bone graft was done and an acrylic stent left in place for six months. She was decannulated, but a tracheotomy had to be done again three months later because of progressive dyspnea. She has had several dilatations with some increase in lumen but this could not be done often enough to be effective because of her living out of town. A medium size acrylic reproduction of an O'Dwyer tube was inserted per laryngoscopy and left in place four months. This was then replaced with an acrylic reproduction of a No 38 core-mold with a lumen drilled through it for two more months. This was removed only recently. Communication by telephone revealed that the airway is greatly improved so far, however she is pregnant so decannulation will not be considered for a few months. If the posterior webbing re-forms she will need skin grafting.

SUMMARY

A discussion of the immediate and late effects of external trauma to the larynx, and recommended treatment has been presented. Case reports representing a variety of situations are given. Practically any chronic stricture can be corrected if the surgeon is persistent, but the end results are much better if the stricture is prevented by early definitive treatment. This should be done within the first 48 hours if possible.

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CYSTIC LYMPHANGIOMA OF THE PAROTID REGION *†

RALEIGH E LINGEMAN, M D ,

Indianapolis, Ind

Cystic lymphangiomas are multilocular cystic tumors of a benign neoplastic nature which have a lymphatic origin and whose spaces are lined with a single layer of endothelium These tumors have the power of new growth and infiltration of surrounding structures by local extension This definition includes cystic hygroma which is considered to be an identical pathologic process Approximately 93 per cent of the reported cases in the literature are located in the cervical region, although these tumors have also occurred in the axilla, groin, popliteal fossa, retro-peritoneal space and mediastinum Primary involvement of the parotid area without associated involvement of the cervical region appears to be a very unusual occurrence A review of the literature on this subject, which has been published during the past 45 years, revealed only an occasional case of primary parotid involvement by these tumors, consequently, it is felt worthwhile to describe five cases with which the author has had experience during the past four years

The earliest report in the literature was made by Redenbacher in 1828, although the first important contribution to our knowledge of hygroma was made by Wernher in 1843 He was the first to name these tumors cystic hygromas He recognized that most of these tumors occurred in the cervical region, and he believed that they were the result of a new formation independent of the development of the fetus Birch opposed the idea of Wernher , he felt that there was no evidence of neoplastic tissue in cystic hygroma, but rather that these lesions respresented a hydrops of the subcutaneous

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[*] Read at the meeting of the Middle Section American Laryngological Rhinological and Otological Society Inc Chicago Ill Jan 18 1960

[†] From the Department of Otolaryngology Indiana University School of Medicine

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tissues Rokintansky believed that this tumor resulted from a tissue limiting process around an area of edema or serous exudate occurring in the fetus The limiting walls were formed from the thickened pre-existing connective tissue which surrounded the fluid The lining of the walls subsequently became smooth whereupon a secretory balance occurred between the walls and the fluid Gurlt agreed with Rokintansky and referred to the condition as hydrops universalis Virchow in 1863 agreed with Wernher that hygroma represented a new formation and was unrelated to developmental anomalies of the fetus Arnold in 1865 reported two cases of cystic hygroma and stated that the growths did not arise from intracarotid ganglia since the cystic cavities were lined with endothelium His conception of the growth was that the cellular elements of connective tissue disintegrate forming cystic spaces in which fluid accumulated secondarily Koester demonstrated the endothelial lining of cystic hygroma by the means of silver solutions, and he concluded that the cystic cavities of hygroma arose by widening of pre-existing lymph vessels He thought that this accounted for the penetration of those tumors into surrounding organs and tissues, and he designated the hygroma tumors as lymphangiectasis congenita Brost, in 1902, made an extremely important observation He studied lymphangioma located within lipoma and offered evidence of the formation of new lymph capillaries from the intra-cellular canalization of beds which sprouted from the endothelial lining of the dilated vessels[6] Dowd[7] reviewed the literature up to 1913 and reported 91 cases of cystic hygroma occurring in the neck and 35 cases of hygroma located in the axilla Dowd also quoted the work of McClure and Sylvester in 1909 concerning the embryology of the lymphatic system They were of the opinion that hygromas arise from sequestrations of lymphatic tissue derived from the primitive jugular sacs Dowd also thought that these lymphatic rests possessed an independent power of irregular growth Ribert thought that the growth of the hygromas and lymphangiomas was similar and the result of independent power of penetration on the basis of endothelial shoots which later became canalized and thus would give rise to cysts He also agreed with Dowd that hygromas are derived from the lymphatic sequestrations which are remnants of the primitive jugular sacs

EMBRYOLOGY OF THE LYMPHATIC SYSTEM

The generally accepted facts concerning the development of the lymphatic system have resulted from investigations and observations of Sabin, Huntington and McClure They have established that in the primary stage of development, a capillary plexus is formed from the jugular vein on each side In certain areas a part of the capillary plexus is cut off from the parent vein This group of isolated spaces lined by endothelium remains for a short time constituting the anlage of the lymphatic system These isolated capillaries then dilate and coalesce to form symmetrical, lined sacs In this manner the two primitive jugular sacs, one on each side, are formed (see Fig 1) The thoracic duct connects the pelvic and retroperitoneal sacs with the left jugular sac and then forms an opening into the veins at the jugular valve On the right,

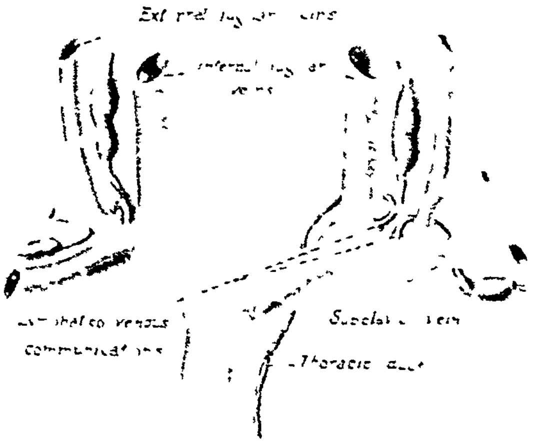

Fig 1 Reconstruction of the jugular sacs of an 11 millimeter cat embryo showing the relation of the lymphatic buds to the jugular and subclavian veins through which the adult communications are established (after McClure and Sylvester)

there is a somewhat similar development Another pair of lymph sacs appears in the pelvis which are related to the corresponding iliac veins into which they open The secondary stage of development then involves the peripheral growth of the lymphatic vessels Sabin has shown that this is accomplished by endothelial sprouts which are derived from the lining of the jugular sacs which in the third month, spread and invade the tissues of the neck, head, and arm From the

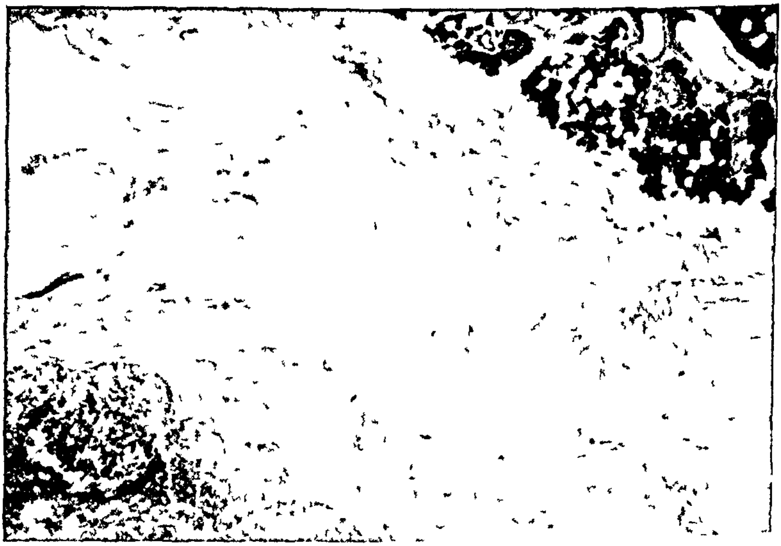

Fig 2 Cross section of a cystic lymphangioma of the parotid area showing the cystic spaces lined with a single layer of endothelium together with lymphoid and glandular tissue

pelvic and iliac sacs, outgrowths penetrate the hind limbs and the tissues of the pelvis and buttocks In addition, there is the element of secretion from the endothelium of the newly formed lymphatics This is the normal course of development of the lymphatic system Through some anomaly of growth, fibrosis or other accident, the primitive jugular sac or portions of it may fail to establish a communication with the venous system The lymphatic vessels thus sequestrated may then retain the power of inherent growth as demonstrated in the process of endothelial sprouting and penetration characteristic of the development of the normal lymphatic system

These then, are the factors necessary for an explanation of the origin and growth of cystic hygroma [11, 12]

Accordingly then, if one accepts the above theory, cystic lymphangiomas or cystic hygromas involving the parotid area result from sequestrated lymphatic tissue which had originally penetrated this region of the head from the primitive jugular sac Due to some accident, portions of it failed to establish a communication with the venous system Later on, unknown factors may cause activity of the inherent power of growth and penetration of this tissue, thus resulting in the cystic endothelial lined tumor involving this area These exciting causes are not definitely known, but it has been noted in this small group of cases that in two of these children, the growth of the tumor occurred quite suddenly and progressed very rapidly following an upper respiratory infection (see Fig 5) In the other three children, the growth was rather slow and progressive and did not appear to be related to any definite factor (see Figs 3 and 4)

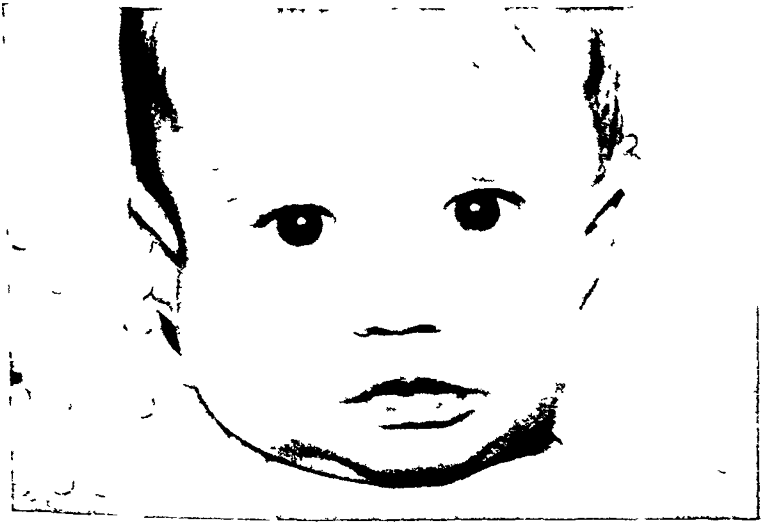

Fig 3 Eight-month-old boy with a cystic lymphangioma of the right parotid gland area.

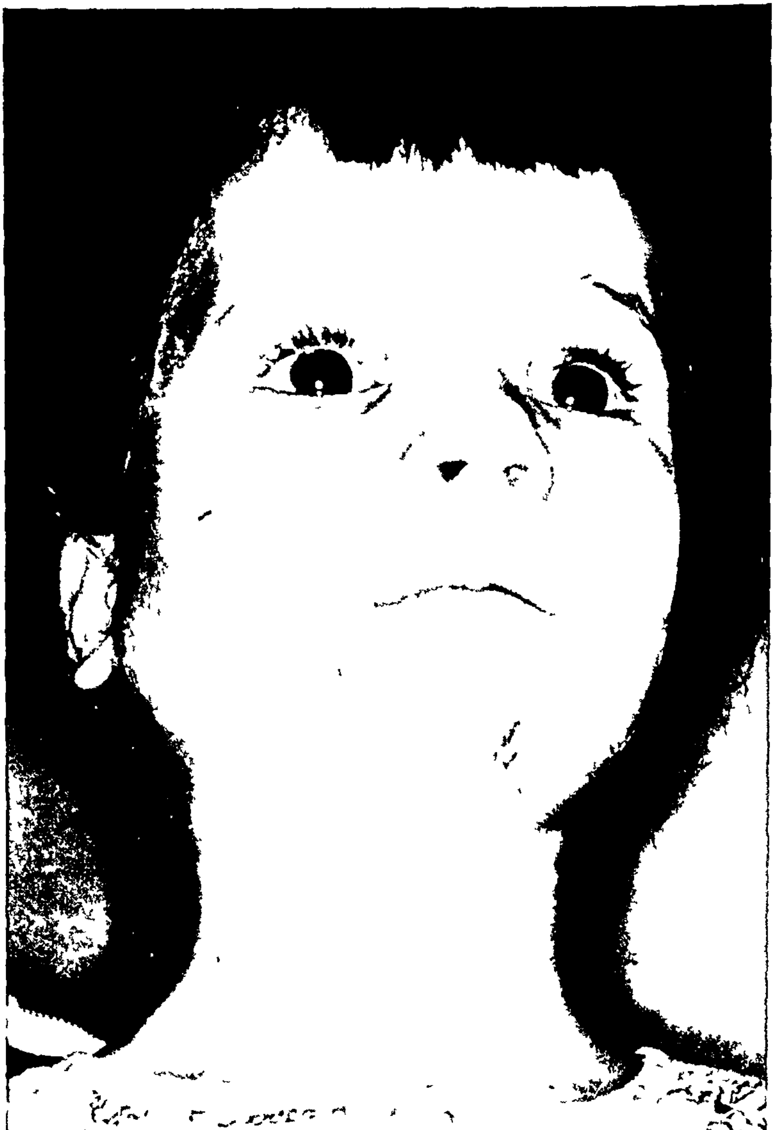

Fig 4 Seven-year-old girl with a cystic lymphangioma of the right parotid gland area

HISTOPATHOLOGY

Generally speaking, there are three varieties of lymphangioma First, simple or capillary lymphangioma which occurs quite superficially and usually involves the skin or subcutaneous tissues The second type is cavernous lymphangioma which consists of a framework of connective tissue in which there are numerous single and communicating lymphatic cysts There are many anastomosing channels and often irregular masses of lymphocytes, lymph nodes, and lymph follicles, and occasionally one sees neoplastic blood vessels in great numbers When vascularity is quite great the term hemangio-lymphangioma is often applied to them The third type, and the one with which we are concerned, is cystic lymphangioma These are tumors with larger thin walled convoluted cysts filled with lymphatic fluid The majority of these tumors occur in the cervical region and are often called cystic hygromas They may be present for a considerable period of time and then suddenly begin to enlarge Some pathologists feel that these tumors are at the onset cavernous lymphangiomas which become dilated with lymph, either as a result of a change in the drainage or an alteration in the function of the lining membrane This sudden onset of rapid growth may be explained by a very active cell production with an inherent power of growth on the part of the endothelium The extension of these tumors into the axilla and the mediastinum has been thought to corroborate this view

The characteristic gross pathologic appearance of these tumors is a multilocular cystic mass in which the individual cysts vary from the size of a pinhead to that of a lemon The walls of the younger cysts are extremely thin and often transparent, the walls of the older cysts are often thick and fibrotic There is frequently generalized fibrosis in the older tumors and often fragments of adjacent muscles, fascia, nerves, and blood vessels are adherent to the main mass The cysts themselves are usually soft and fluctuant but are rarely tense The fluid within the cysts is serous, watery, clear or straw colored, and occasionally blood stained Often the mass is intimately associated with groups of enlarged lymph nodes The nodes are encountered in the walls of the tumor and are doubtless directly involved in the pathologic process The cystic spaces within the tumor are found to be lined with a thin, white, glistening, almost transparent, membrane which resembles peritoneum or pleura The microscopic pathologic characteristics reveal these cysts to be lined with a single layer of endothelium The older cysts are characterized by thick fibrotic walls which at times are almost avascular Below the endothelial layer, there is often a layer of loose, extremely fine reticulum composed of a row of connective tissue resembling very fine adipose tissue Usually in the thin and younger cysts there are dense aggregations of lymphoid cells These vary in size from groups of a few cells to large accumulations containing true follicles with germinating centers which resemble those in lymph nodes (see Fig 2) Goetsch has reviewed the histologic characteristics of the manner in which these tumors penetrate adjoining normal tissues His excellent observations have demonstrated that from the growing margin of the cyst, fine delicate membranes appear as bands or cords composed of endothelium These radiate from the wall of the cyst and in a serpentine fashion penetrate the clefts of the adjacent tissue They terminate in several small fibrillae which are lost in the adjacent tissues These radical sprouts in turn send off lateral branches into the tissue clefts Between these fibrillae frequently are seen lymphoid cells and, at varying intervals along the sprouts, droplets of a foam-like faintly blue staining secretion is noted These droplets are identical in appearance with the coagulated contents often seen in the larger and older definitely formed cystic spaces As a result of the continued secretion, the droplets enlarge and are then enabled to spread apart and finally canalize the fibrillae These, thereupon, form the limiting endothelial walls of the newly formed lymph capillaries and minute cysts New cysts are brought into being by this characteristic process of secretion within and subsequent canalization of the fibrillae The penetrating endothelial fibrillae may surround and isolate individual muscle fibers and glandular tissue which later show atrophy and degeneration [6]

Unless infection or hemorrhage has occurred, the fluid characteristic of cystic lymphangiomas is thin, watery, and clear or straw colored It is particularly free of albumin and globulin and does not coagulate on cooling A study of the sediment following centrifugation shows large numbers of lymphocytes, mononuclear cells, and cholesterol crystals

SYMPTOMS

Cystic lymphangioma is a disease of infancy and early childhood These tumors are usually noted at birth and, in most instances, grow slowly, however, sometimes there is rapid growth with encroachment on vital structures of the surrounding area In the parotid area, the tumor may appear as a soft poorly defined swelling It is painless and is usually observed for a variable length of time before medical consultation is requested Some investigators interested in hygroma, have pointed out the periodic fluctuation in size which has frequently been observed This is more characteristic of the cervical type and was not noted in the five cases of involvement of the parotid area presented here In one of these cases, the parents and family physician noted the presence of the above described soft poorly defined mass at birth (see Fig 3) In two cases the onset of the tumor was noted within the first few years of life (see Fig 4) This was observed by the parents and because of the cosmetic deformity they sought medical attention In the other two cases, the growth of the tumor was quite rapid and appeared after an upper respiratory infection (see Fig 5) Apparently the infection had led to a plugging or partial obstruction of the normal lymphatic channels so that there was a backing up of the lymph in the cystic tumor which caused it to enlarge This would, of course, indicate communications existing between the lymphangioma and the normal lymphatic spaces

The picture may be complicated as a result of an infection produced by an attempt at incision and drainage Two of these cases were previously diagnosed as parotid abscesses One had been incised and drained nine times prior to admission to the Indiana University Medical Center (see Fig 5) The other case was aspirated and the centrifuged specimen examined microscopically Because of the large number of

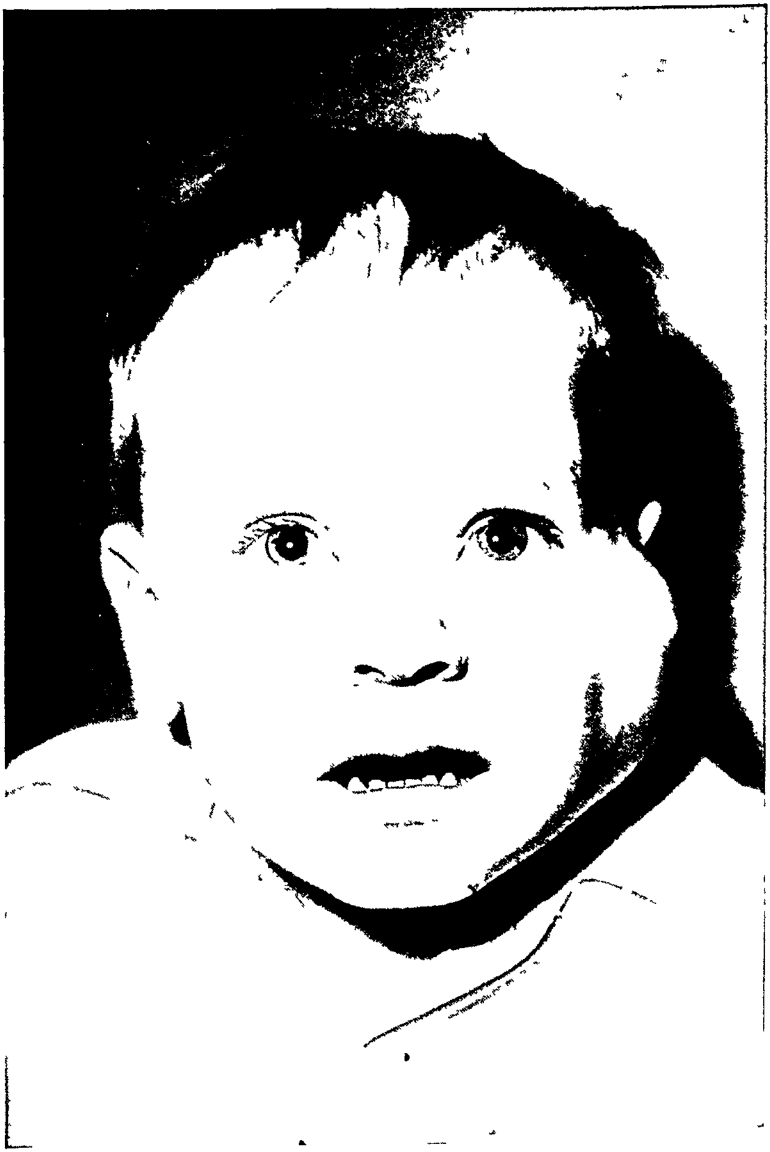

Fig 5 Three-year-old boy with a cystic lymphangioma of the left parotid gland area Incision and drainage had been performed nine times prior to removal of the tumor Two other cases were similar in appearance to this child

lymphocytes present, the possibility of cystic hygroma was strongly considered

DIAGNOSIS

Since the occurrence of cystic hygroma of the parotid area is unusual, diagnosis of this tumor is often not made prior to surgery It is our feeling that the most helpful aid in diagnosis, aside from keeping in mind the possibility of such a tumor, is aspiration under sterile conditions and cytologic examination of the sediment after centrifuging the collected fluid Cystic hygromas will usually show large numbers of lymphocytes and the absence of epithelial tissue The two most common conditions which would simulate cystic hygroma in the parotid area, are branchial cleft cysts of the first arch, and lipomas Branchial cleft cysts are usually associated with a dimple or a draining fistula into the external auditory canal They are usually tense, firm, well circumscribed, and often deep seated Aspiration of such a tumor will reveal a large amount of cholesterol present in the fluid together with presence of epithelial debris typical of the lining from such a cyst Lipomas are more difficult to differentiate They are usually extremely soft and poorly defined, non-tender, and are sometimes in a deep seated location They are not as fluctuant as cystic hygromas and grow quite slowly Other conditions which one must consider in a differential diagnosis are tubercular adenitis, abscesses of the parotid area, hemangioma, and occasionally a tumor of the malignant lymphoma group

TREATMENT

In the treatment of cystic lymphangioma, several forms of therapy are available It is best to consider these individually as they may apply to location in the parotid gland region

1 *Expectant Treatment* Some investigators have advised that surgical treatment should be withheld for an indefinite period of time because of the possibility that the hygroma may undergo spontaneous regression Most investigators are not in agreement with this idea Although it is true that

some cysts will decrease in size, they usually refill and there is always a progression in size if a patient is followed over a long period of time

2 *Aspiration Treatment* Some authors have advised that in very young children, aspiration treatment should be used until the child is old enough to withstand surgical removal of the tumor Repeated aspirations always are accompanied by the danger of secondary infection If this should occur in a lesion located in the parotid area, subsequent removal would be made more difficult

3 *Injection of Sclerosing Agents Following Aspiration of the Cysts* This type of treatment has been suggested and, in tumors located in other parts of the body, is probably acceptable Repeated aspiration of the involved area is done followed with injection of a 5 per cent solution of sodium morrhuate This is repeated several times and after a period of a month, the mass is removed Investigators who have studied the pathologic material available from these tumors, are in agreement that the thinness of the lining of cystic lymphangioma makes it almost ideal for injection therapy The sclerosing agent can easily diffuse to the fluid medium and would not have to penetrate deeply into the tissues in order to destroy the endothelial layer In the case of treatment of these tumors in the parotid area, one would have to consider possible damage to the facial nerve which may occur if the sclerosing agent were injected into branches of the nerve itself

4 *Radium or Roentgen Ray Therapy* Gorden New and Frederick Figi from the Mayo Clinic have been the principal proponents of radium therapy[5] The cases that these investigators reported were done approximately 30 years ago At that time, surgical excision of these tumors was considered a rather hazardous procedure in young children In regard to treatment of cystic lymphangioma in the parotid area, it may be stated that presently radium or X-ray therapy is not an accepted therapeutic measure The retardation effects on the future growth in this area and also the possibility of carcinogenic tendencies developing years later in individuals who

have had this type of treatment for benign lesions are contra-indications for this therapy

5 *Surgical Excision* The meticulous surgical removal of cystic lymphangioma from the parotid area is the most satisfactory method of treatment In general, the surgical procedure is similar to that used for removal of parotid tumors The skin incisions should be in a direction which will later correspond to the normal folds in this area The incision as used by Dr James Maxwell of Ann Arbor, Mich , is considered the best for our purposes here A skin flap over the parotid area is developed and carried anteriorly as far as is necessary Dissection is carried between the external auditory canal and the parotid gland itself to the tympanic plate of the temporal bone The tail of the parotid is separated from the anterior border of the sterno-cleido mastoid muscle, and dissection is carried deeper into the groove between the tympanic plate and the mastoid process The main trunk of the facial nerve is then identified as it emerges from the styloid mastoid foramen In our experience with the five cases reported here, the cystic lymphangioma seemed to originate from beneath the facial nerve in close proximity to the posterior facial vein In one case, the cyst was so closely attached to the vein that resection of the posterior facial vein, together with the cystic hygroma, was necessary From beneath the nerve, the tumor spreads throughout the parotid area penetrating between portions of salivary tissue, connective tissue, and fat The tumor seems to penetrate adjacent structures following the lines of least resistance It is interesting to note that the tumor seems to grow in an outward direction rather than to penetrate deeper structures in this area These cysts in the parotid area are multilocular They consist of a variation of cysts which are only a few millimeters in diameter to those which are several centimeters in size No attempt is made to dissect each individual cyst, but the dissection is carried superficially to the facial nerve until the branches of the facial nerve have been identified in their entirety The superficial lobe of the parotid gland together with portions of the cystic lymphangioma which involve it are first removed Following superficial parotidectomy and removal of the hygroma

in this area, piecemeal removal is carried out beneath the nerve until the dissection has been complete It is our feeling that it is mandatory to maintain the integrity of the facial nerve, and in doing this, it is necessary frequently to leave minute bits of lymphangiomatous tissue along their surfaces This does not militate against a successful outcome, since apparently the small remaining islands of lymphangiomatous tissue become sclerosed and do not give rise to subsequent recurrences A Penrose drain is brought out through a stab wound in the upper cervical region The wound is thoroughly irrigated and the skin edges are approximated using fine silk A pressure dressing is then applied in the usual manner There were no cases of temporary or permanent facial paralysis following surgical management of this type, and at the present time, there have been no cases which have shown evidence of recurrence

FIVE CASE HISTORIES

Case 1 R F The patient is an eight month old boy who was admitted to Riley Children's Hospital in Indianapolis, Ind, because of a cystic tumor involving the right parotid gland area The parents had noted the tumor shortly after birth The tumor slowly increased in size although it remained painless Physical examination and laboratory work were entirely normal Surgery was done on December 11, 1956, and consisted of parotidectomy on the right side with removal of a cystic lymphangioma with preservation of the facial nerve The child was discharged from the hospital on December 16, 1956 There has been no evidence of recurrence at this time

Case 2 F V N The patient is a five year old girl who was admitted to St Vincent's Hospital in Indianapolis, Ind, because of a cystic tumor involving the right parotid gland region Several weeks previously, the child had had what was thought to be unilateral mumps Aspiration of the involved area revealed a bloody fluid which on cytological examination revealed a large number of lymphocytes Cystic lymphangioma was entertained as the most likely diagnosis Parotidectomy was performed on July 9, 1957, with apparent complete removal of the cystic lymphangioma

Case 3 J J The patient is a three year old boy who was admitted to Riley Children's Hospital in Indianapolis, Ind, because of a tumor involving the left parotid gland region Onset of this tumor was noted in May, 1958, at which time diagnosis of unilateral mumps was made The swelling persisted and during the next five months, incision and drainage of the involved area was done nine times Surgery was done on October 3, 1958, and consisted of parotidectomy with removal of a cystic lymphangioma involving the left parotid gland The facial nerve was preserved intact Follow up to this time has revealed no evidence of recurrence

Case 4 D R The patient is a two and a half year old girl who was admitted to the Indianapolis General Hospital on October 30, 1958, be cause of a large preauricular cystic tumor involving the right parotid gland area The tumor had been present in this region for approximately 18 months Diagnosis of cystic lymphangioma was entertained, and surgery was done on October 31 1958 Surgery consisted of parotidectomy with removal of cystic lymphangioma from the right parotid gland region The facial nerve was preserved intact The patient's postoperative course was satisfactory, and she was discharged from the hospital approximately ten days later There has been no recurrence at this time

Case 5 D H The patient is a seven year old girl who was admitted to the Methodist Hospital in Indianapolis, Ind, because of a cystic tumor involving the right parotid gland region The parents had noted the onset of this tumor approximately 18 months previously, and it had slowly increased in size Diagnosis of cystic lymphangioma of the right parotid gland area was made, and surgery was carried out on February 9, 1959 Surgery consisted of parotidectomy on the right side with removal of a cystic lymphangioma The facial nerve was preserved intact The postoperative course was satisfactory and the child was discharged from the hospital on February 13, 1959 Subsequent follow-ups have revealed no evidence of recurrence to this time

SUMMARY

A review of the developmental anatomy of the lymphatic system and the etiology and behavior of cystic lymphangioma is presented

Experiences with five cases of cystic lymphangioma involving the parotid area are reported

It is the opinion of the author that the treatment of this tumor in this location is best accomplished by meticulous surgical excision

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TEMPLE UNIVERSITY POSTGRADUATE COURSES

Postgraduate course in laryngology and laryngeal surgery, September 26th to October 7th, 1960

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These courses are to be given in the Department of Laryngology and Bronchoesophagology, Temple University Medical Center, under the direction of Drs Chevalier L Jackson and Charles M Norris

The tuition fee for each course is $250 Application and information may be obtained by writing to Jackson-Research, Lab 604, Temple University Medical School, 3400 N Broad street, Philadelphia 40, Pa

DEEP MYCOTIC INVOLVEMENT OF THE RIGHT MAXILLARY AND ETHMOID SINUSES, THE ORBIT AND ADJACENT STRUCTURES

Case Report Evaluating the Use of Mycostatin Locally and Amphotericin B (Fungizone) Intravenously Against Aspergillus Flavus[*][†]

WENDELL A WELLER, Colonel, MC,
DONALD J JOSEPH, Lt Colonel, MC,[‡]
(By Invitation),
and
JAMES F HORA, Captain, MC,§
(By Invitation),
Denver, Colo

The etiological agent in this case was identified to be Aspergillus flavus The structures affected were the right maxillary sinus, the ethmoidal labyrinth, the right orbit and adjacent structures Several references recommended the treatment of this infection[1,2,3,4,5] to be the administration of a saturated solution of potassium iodide, the surgical excision of the granuloma as far as possible, and the avoidance of irradiation therapy[6] The use of Mycostatin (Nystatin) was suggested by the manufacturers[7,8] in February, 1956 Nystatin was the first antifungous antibiotic agent of high efficiency and low toxicity It was derived from cultures of Streptomyces noursei This antibiotic was used in the powdered form and insufflated into the nose and right maxillary sinus for weeks Obviously this was only a superficial topical application

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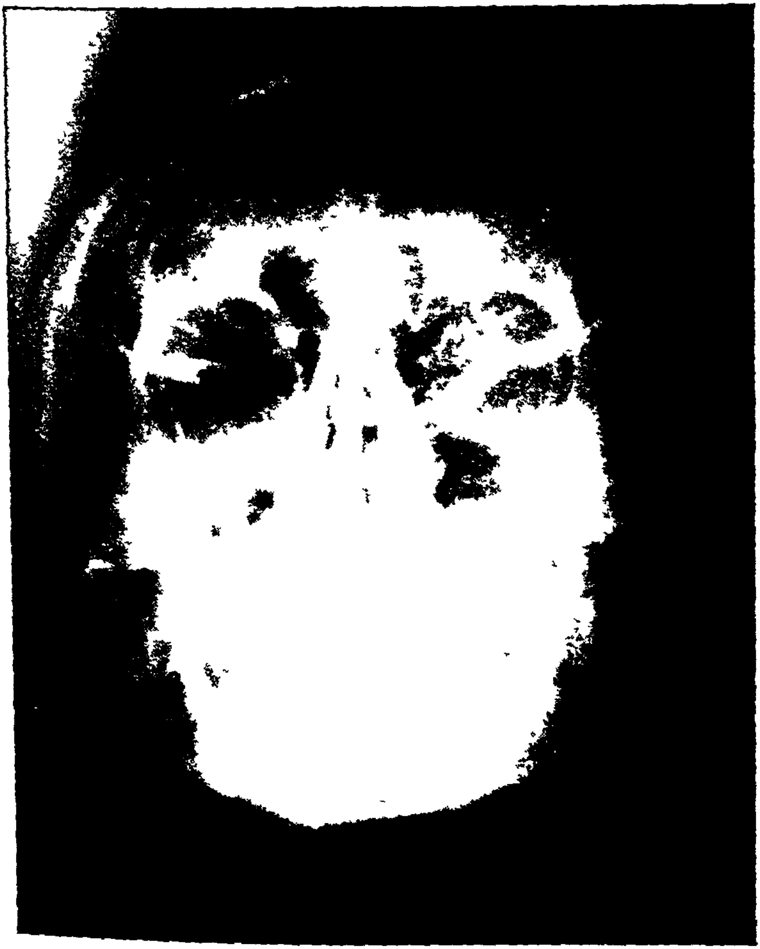

Fig 1 X-rays (Waters view) of sinuses showing increased density upper half of the right maxillary sinus The lower right ethmoid area is denser than the corresponding left side There is some mottling of the body of the right malar bone

December 13 1955 an exploration of the right maxillary sinus and ethmoid sinuses was done using the Caldwell Luc approach Cultures of the tissues from the sinuses grew Aspergillus flavus The fungus was also found in the tissues by PAS stain As much of the granuloma as possible was removed surgically from the maxillary and ethmoidal sinuses The bony floor of the right orbit had been eroded There was no demarkation between the granulomatous tissue and the normal structures of the floor of the right orbit (see Figs 3 and 4)

Postoperatively the patient was given a saturated solution of potassium iodide taking increasing doses up to 25 drops three times daily Locally Mycostatin powder was insufflated into the nose and right maxillary

Later, one course of this medication was given orally This drug was reported to be very poorly absorbed by mucous membranes or the intestinal tract It had been used in the treatment of yeast and mycotic infections following the administration of "broad spectrum" antibiotics There was no evidence of how repeated applications might influence this infection if it were used locally This strain of Aspergillus flavus was sensitive to 10 micrograms per milliliter of Nystatin *in vitro* No other more specific preparation was available at that time for the treatment of fungus infections

In April, 1957, the first course of amphotericin B (Fungizone®)* was begun This was given intravenously in 5 per cent glucose solution Amphotericin B is an antibiotic formed by a species of Streptomyces isolated from a soil sample obtained in South America A second course of treatment was started in November, 1957, a third course in February, 1959 The surgical treatment and clinical course is detailed below

CASE STUDY

Patient is a 31-year old colored male, with ten years' military service, who first noted prominence of the right eye in February, 1955 This enlisted man had been a heavy equipment supervisor for the Army Corps of Engineers He had served in Korea from 1950 to 1952, and another year in Northern Japan In 1953 he spent six months at Camp Polk, La He was stationed at Fort Hood, Tex , for about two years, and then sent to Fort Ord, Calif , in March, 1955 In May, 1955, while at Camp McCoy, Wisc , he came under medical observation for the exophthalmos and arrived at Fitzsimons Army Hospital in July In March, 1955, he began to have blurred vision in the right eye On admission to this hospital his vision was 20/50 in the right eye and 20/20 in the left eye *Vision had been 20/20 in the right eye previously* His exophthalmos measured 23 mm in the right eye and 19 mm in the left eye with the exophthalmometer Fundus examination of the right eye showed horizontal striae which involved the macular area Visual fields showed a loss of the upper portion of the field of vision above the horizontal X rays of the sinuses showed an increased density of the upper half of the maxillary sinus and some haziness of the inferior ethmoid cells on the right side (see Fig 1) The right maxillary sinus was opened through the canine fossa on July 20, 1955, and a biopsy of the tissue in the upper portion of the sinus taken The diagnosis of a non specific granuloma was made, and the patient was given a course of antibiotic therapy, including terramycin, INH, streptomycin, and penicillin The INH was given 300 mgs per day until September, 29, 1955 without evident improvement of the exophthalmos

In December, 1955, patient began to experience discomfort in the right eye The exophthalmos remained about the same (see Fig 2) On

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*Fungizone® is a Squibb trademark The Squibb Institute for Medical Research kindly furnished the drug for this clinical investigation

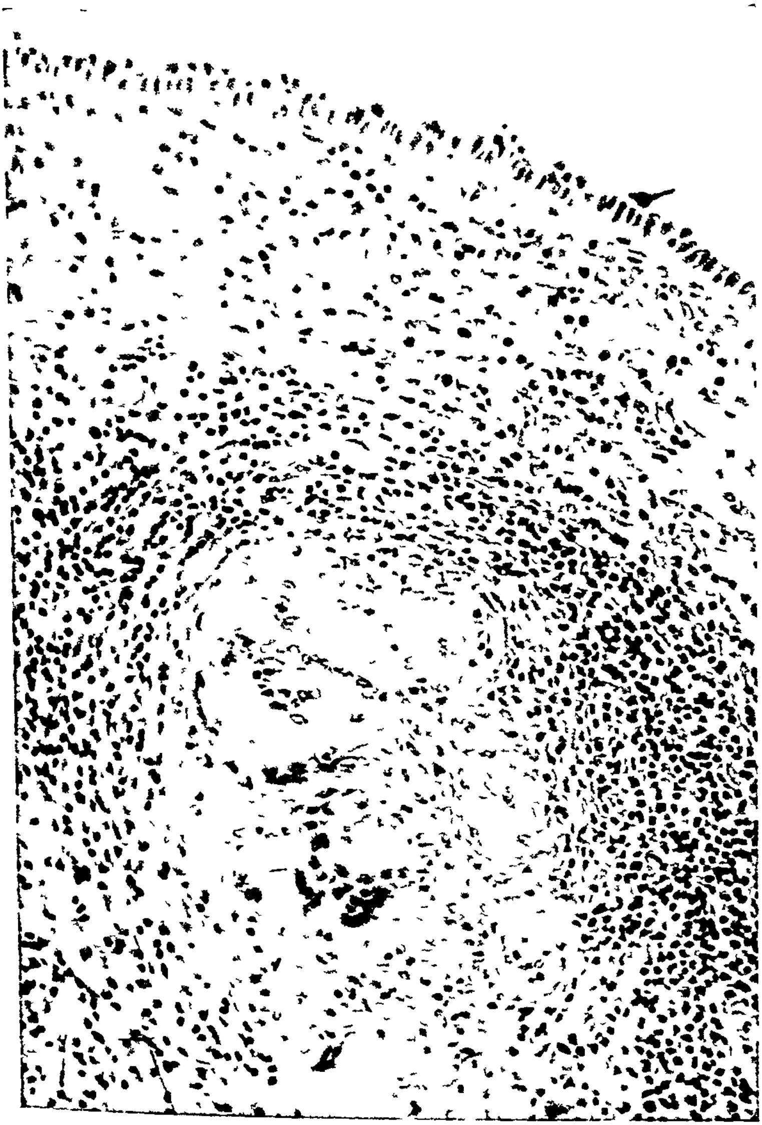

Fig [illegible] Photomicrograph of tissue removed from the right maxillary sinus December 13 1955 The respiratory epithelium is at the upper edge The granulomatous tissue is composed of relatively young fibroblasts with trabeculae of more mature fibrous tissue The infiltrate is of all types of chronic inflammatory cells with numerous scattered multinucleated giant cells (X200)

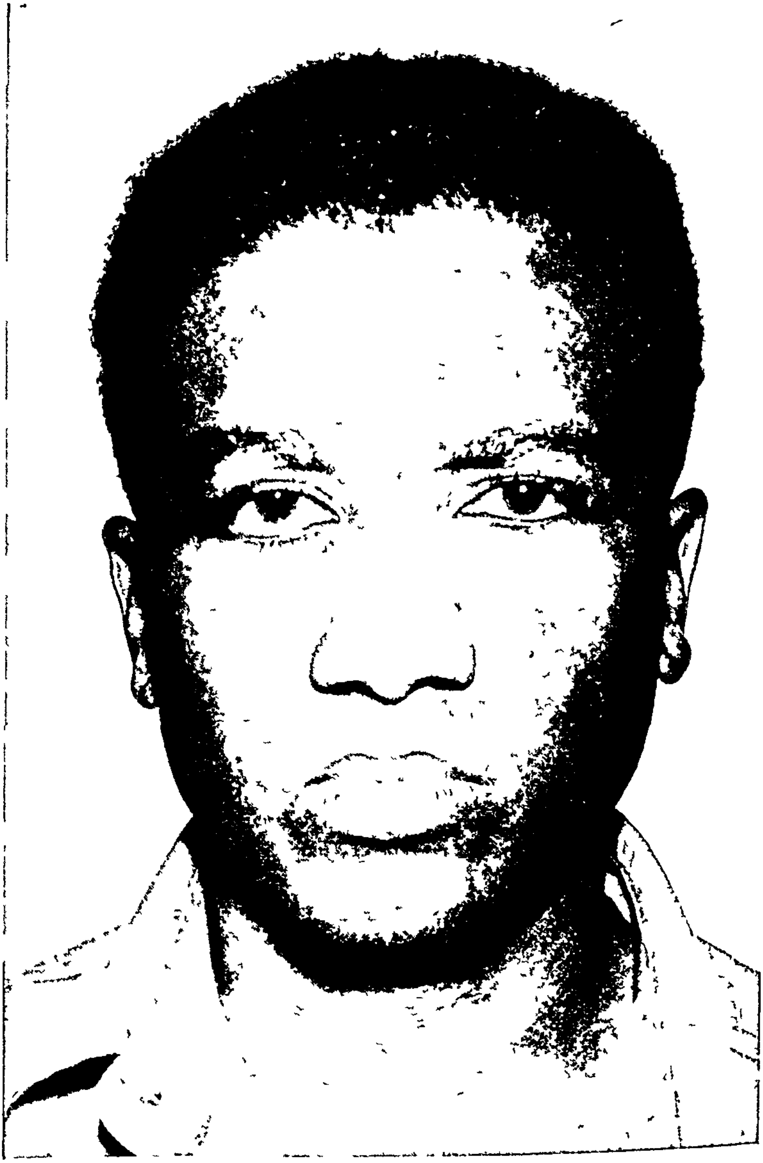

Fig 2 Photo of patient, December, 1955 Exophthalmos right eye measures approximately 5 mm

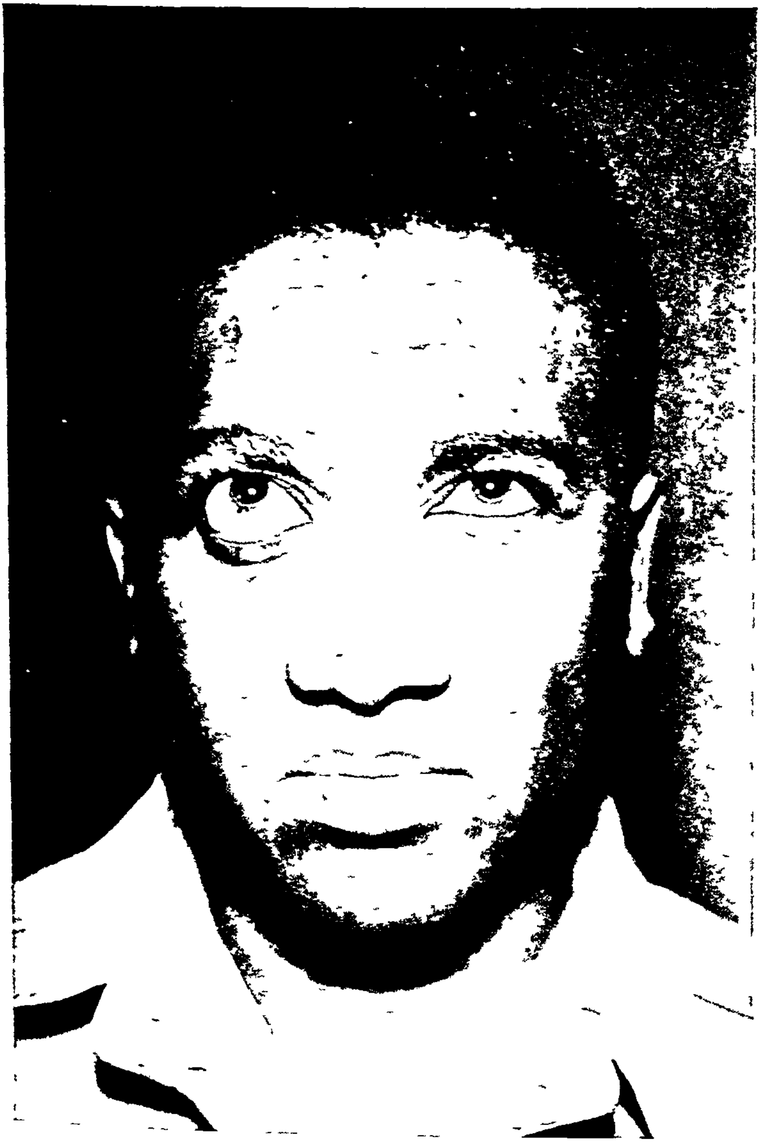

Fig [illegible] Photo of patient January 1957 at which time his exophthalmos measured 8 mm

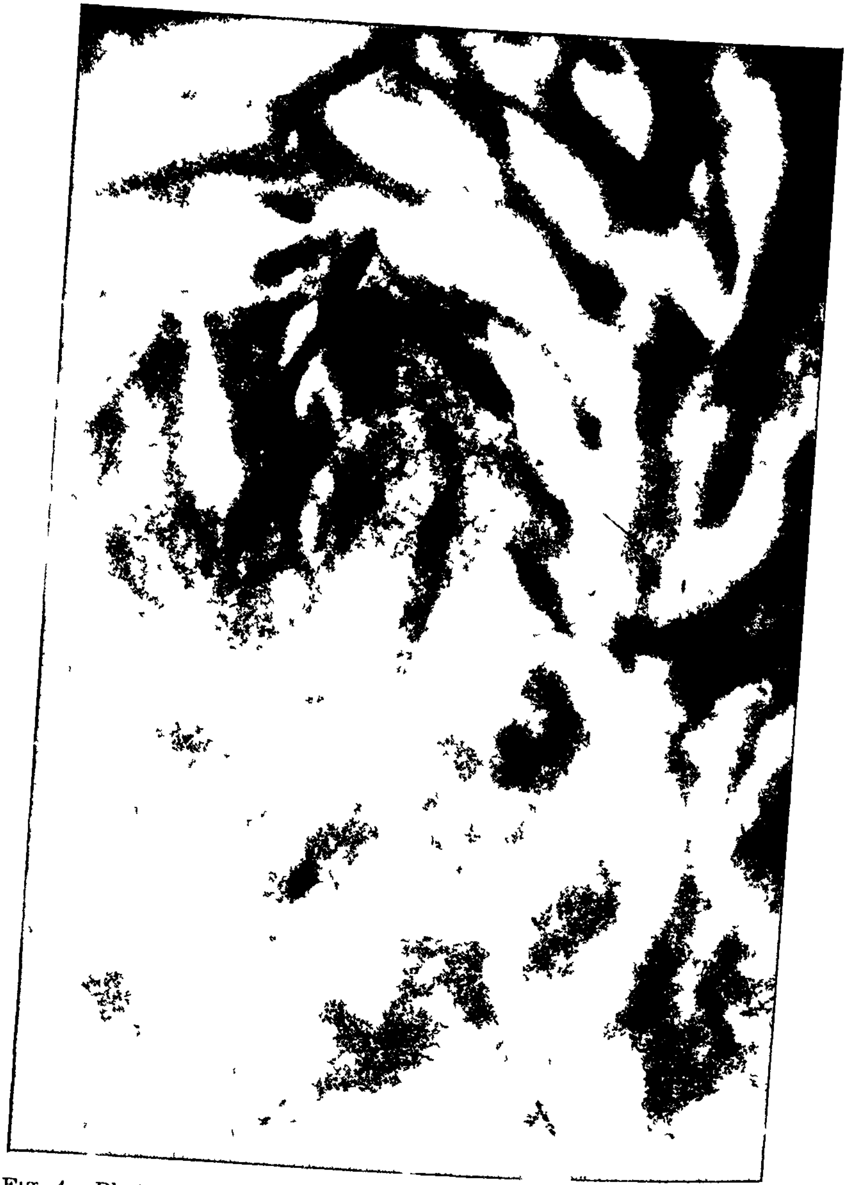

Fig 4 Photomicrograph of PAS stained tissue removed from right maxillary sinus December 13 1955 showing numerous septating mycelial elements Tissue cultures identified the Aspergillus flavus (×200)

to give him the medication which we had on hand Accordingly, on May 29, 1957, treatment was begun again This time the initial medication was 1 mg per kilogram, which was given in 300 cc of 5 per cent glucose, and the daily dosage was increased by 0 1 mg He received treatment six more days receiving from 66 mg to 92 4 mg daily during this period The reactions from this medication were exactly the same as he had experienced with the previous period of medication, having temperature from 98° to 102° during these six days, and having a chill every day with some nausea and anorexia The patient received a total of 27 days' medication, with a total amount of 1,540 mg of the drug Post medication laboratory examinations showed normal blood counts, blood chemistry within accepted average values, and negative urinalyses X-rays show further destruction of the walls of the right maxillary sinus and perhaps further right orbital involvement The patient received no other medication during the treatment with amphotericin B except aspirin or aspirin-phenacetin codeine compound There appeared to be no improvement symptomatically or objectively

On June 27, 1957, the right orbit was explored under local anesthesia A fibrous mass was found in the inferior portion of the orbit, and the mass of tissue was removed piecemeal It extended posteriorly to the region of the optic foramen and was adherent to the periosteum of the floor of the orbit It also extended medially into the left ethmoid region It appeared that the granuloma had developed from the roof of the right maxillary sinus and eroded through the bony floor of the orbit Powdered Nystatin was placed in the operative site Specimens were forwarded to the laboratory for pathological examination The diagnosis was again confirmed on July 1, 1957, by the pathology service and again demonstrated Aspergillosis The postoperative period was complicated by the formation of an abscess which drained through the incision on July 6, 1957 This infection gradually cleared with no more difficulty On July 27, 1957, there was 4½ mm of exophthalmos The first of August the patient was feeling considerably better The operative area was well healed and he was given 15 days convalescent leave, during which time he took potassium iodide drops He was re-evaluated on August 9, 1957, and was given another convalescent leave On September 9 1957, the right exophthalmos measured 6½ mm Funduscopic examination at that time continued to show some dilatation of the veins of the fundus In October, 1957, patient was given several injections of amphotericin B diluted with procaine or xylocaine into the floor of the right orbit[10] This procedure was quite painful to the patient, and no definite beneficial result could be noted On November 23, 1957 he showed an exophthalmos of 8 mm, the visual acuity was 20/200 on the right correctible to 20/20, and visual acuity was 20/20 on the left On November 26 1957, another course of intravenous amphotericin B was started This continued through January 2 Approximately 1,000 mg was given during this course of therapy The patient had his usual symptoms of some fever and chills during this period Inasmuch as exophthalmos was progressing and the patient was again experiencing pain, it was decided that further surgical intervention was necessary On December 2 1957, under endotracheal anesthesia the patient had a removal of granulations and adjacent bone of the right maxillary sinus, the floor of the right orbit, and the structures of the lateral wall of the right nasal passage The granuloma involving the floor of the orbit was removed as completely as possible, however fibrosis seemed to extend posteriorly beneath the optic nerve Two units of blood were given following this procedure and the course of intravenous amphotericin, which had been started on November 26, 1957, was continued The procedure decompressed the orbit from below and medially lessened the exophthalmos and provided the sinus space daily By April, 1956, the clinical and X-ray findings of the skull seemed stabilized Cultures of tissue removed from the nose and right maxillary sinus failed to grow fungus Vision in the right eye was correctible to 20/20, the visual fields, fundus findings and exophthalmos remained unchanged He was followed as an outpatient until January 14, 1957, at which time he was rehospitalized due to an increase in the exophthalmos in the right eye to 26 mm (see Fig 5) In March, 1957, the exophthalmos measured 28 mm On March 19, 1957, patient was placed on four tablets of Nystatin every six hours for 12 days and then five days' treatment with eight tablets every six hours The patient tolerated this medication without any untoward effects, in fact, he states he gained six pounds during this period There was no noticeable change in the exophthalmos or his symptoms following this medication His general condition was excellent

Inasmuch as the infection appeared to be progressing in spite of the medications which had been used, it was decided to give him amphotericin B to see whether it would perhaps have some effect on this infection In April, 1957, the use of amphotericin B (Fungizone) intravenously was begun in dosages recommended by the manufacturer[9] Prior to use of this drug, stool cultures were negative for enteric pathogens Complete blood counts were normal Urinalyses were normal Spinal fluid was examined on April 18, 1957, and the spinal fluid findings, including cell count, sugar chlorides, globulin, total protein, colloidal gold and serological tests, were normal Culture of the spinal fluid showed no growth in 21 days Liver and biliary function tests were normal X-rays of the chest were reported to be normal Stereoscopic views of the skull showed an increase in density throughout the right maxillary sinus and some mottling in the right zygoma There also appeared to be some increased density in the petrous pyramid and some demineralization of the middle cranial fossa on the right During this period the patient had few complaints except the decreased vision in the right eye, the proptosis, and vague aching pains which he frequently experienced in the eye

Treatment with amphotericin B was initiated on April 23, 1957 The initial dose was 0 25 mg per kilogram of body weight This was increased 0 5 mg per kilogram daily Since the patient weighed 146 pounds, or 66 kilograms, the initial dose was 16 5 mg The medication was given intravenously in 200 cc of 5 per cent glucose solution slowly over a period of six hours On May 8, 1957, the dosage had been increased to 1 00 mg per kilogram daily This medication was continued at this level until May 13, 1957, at which time he had received a total of 991 mg of the medication During the middle of this series of medications the patient remarked that he thought he was having a little less discomfort in the right eye, and he also felt that there was less swelling in the area Examination, however, did not verify this

The patient tolerated this medication well, however, he states that he had a chill every day that he received the drug His temperature during the periods of medication was usually 101° to 102°, although some days it was less, and on four or five days rose to 103 2° Near the termination of the medication the patient realized that if he took aspirin or aspirin phenacetin codeine compound early in the period of administration that he had less chills and felt considerably better Most of the chills came near the last half hour of the administration and frequently one half hour or an hour after the medication had been finished He complained of nausea on several occasions, anorexia on many occasions, and twice he vomited

Inasmuch as he had shown little clinical improvement, it was decided resumed a fairly normal appearance The position of the right globe was very nearly normal Measurements for exophthalmos were less reliable since the right lateral bony rim had been removed

The microscopic tissue studies show granulomatous inflammation with areas of necrosis There are predominantly lymphocytes with numerous giant cells, some of these showing hyphae Tissue culture yielded an abundant growth of the Aspergillus species Patient was discharged April 14, 1959, after 54 days of hospitalization

The minimal inhibitory concentration by the multiple tube dilution test for this patient's fungus is given below and is contrasted with the blood levels obtained after the administration of amphotericin B

Sensitivity M I C (Minimum Inhibitory Concentration)

Tissue Culture of	Amphotericin B	Nystatin
December, 1955	6 mcg /ml	10 mcg /ml 40 units
June, 1957	3 mcg /ml	40 units
February, 1959	2 5 mcg /ml	5 50 units/ml

Blood concentration (serum activity) of amphotericin B, after dosage of 1 mg per kilo administered intravenously was 0 2 0 4 mcg /ml maintained over a 12 hour period Obviously this is much below the minimum inhibitory concentration level for this patient's aspergillus infection

This patient has operated his own truck during the summer and fall of 1959 He reported for examination September 19 1959 His vision was O D 20/100 O S 20/20 correctible to 20/25 on the right The proptosis was measured as 6 mm variation between the two corneas There was limitation of adduction on the right The right fundus shows wrinkling of the retina Patient could not wear correcting lenses due to the exophthalmos Blood pressure was 140 mg systolic, 80 diastolic

Patient was readmitted to hospital on December 8 1959 There was definite increase of the proptosis of the right eye The vision was 20/80, correctible to 20/40 There is a noticeable exotropia The fundus picture is the same as described above with the wrinkling or stria about the macular area being visible The patient complains of constant pain over the right cheek There is no particular palpable change in the tissues locally (see Fig 6) It is possible that this complaint is based upon the pressure or infiltration of the second division of the Vth cranial nerve This could mean further extension of the granuloma in the middle cranial fossa Examination of the nasopharynx is negative The general condition is excellent except that this patient presents with a blood pressure of 240 systolic 120 diastolic He is at present under medical treatment which has precluded surgical treatment requiring the administration of a general anesthetic Liver function tests and blood studies are within normal limits There is evidence of some nitrogen retention as indicated by a slightly elevated blood urea nitrogen

TOXICITY

Within the dosage schedule used in the treatment of this patient with amphotericin B intravenously, the toxic effects

patient with a considerable degree of comfort X rays following this procedure showed essentially the same findings with the exception of the operative defect The operative site healed quickly with no complications For the next six to eight weeks the patient was seen daily, the nose was cleansed, and Nystatin powder again dusted into the operative cavity through the right side of the nose Tissue cultures again grew fungus On December 20, 1957, the blood levels of amphotericin B after the daily dosage of 1 mg per kilo body weight were determined to be 0 204 mcg /ml

Early in February, 1958, the patient began to notice a swollen area just above and anterior to the right ear This area has continued to be present, however, it varies in size from time to time The area is not tender and appears to be more of a transitory edema than an extension of the infective process Vision is 20/100 in the right eye, and 20/20 in the left eye, correctible in the right eye to 20/20, however, the patient is unable to wear lenses because of the exophthalmos The retina, right eye, shows stria around the posterior pole from retrobulbar pressure Exophthalmos is 6 mm in the right eye

The patient has been temporarily retired from active military service He has been followed on an outpatient status Tissue biopsies from the lateral wall of the right nasal passage on June 25, August 16, and November 4, 1958, were cultured and have failed to grow Aspergillus On November 13, 1958, some tissue was removed from the midportion of the floor of the right orbit, going through the right inferior fornix Three separate specimens were taken These three fungus cultures were negative after 26 days The patient's general condition was excellent on examination November 20, 1958 He had a right exophthalmos of 6 7 mm Vision in the right eye was 20/70 correctible to 20/25, and 20/20 in the left eye The fundus of the right eye showed horizontal striae or traction lines extending through the macular area X rays of the sinuses and skull showed little, if any, change during the last nine months

Due to the slowly increasing proptosis of the right eye, more evident swelling of the right temple area and more pain in the right cheek, the patient was rehospitalized on February 20, 1959 Despite the recent negative tissue cultures, it was evident there was recurrent activity of the granuloma Another course of amphotericin B intravenously was begun the next day, using the same dosage schedule as given above On February 24, 1959, under endotracheal anesthesia the right temporal fossa was explored through a horizontal incision extending posteriorly from the external canthus of the right eye Granulomatous tissue had eroded the lateral bony orbital wall near the floor about 3 cm posterior to the lateral orbital rim The involved temporal muscle was removed The bone of the lateral wall of the orbit was removed A large granulomatous mass was encountered beneath and lateral to the globe This was excised by blunt and sharp dissection There were areas of necrosis in the temporal fossa and in the floor of the orbit There were also areas of firm fibrotic material The granuloma extended to the apex of the orbit and beneath this into the pterygomaxillary space There was direct extension of the fibrous tissue into the right maxillary sinus space Dependent drainage was established through the floor of the orbit into the sinus and into the nose through the previously placed window in the medial sinus wall A mushroom tipped catheter was placed in the right maxillary sinus The superior aspects of the orbit seemed to be free of involvement A large Penrose drain was sutured in the initial incision The intravenous administration of amphotericin B was continued postoperatively for a total of 900 mg Locally, the postoperative course was accompanied by protracted conjunctival edema Gradually the tissues resumed a fairly normal appearance The position of the right globe was very nearly normal Measurements for exophthalmos were less reliable since the right lateral bony rim had been removed

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This patient has operated his own truck during the summer and fall of 1959 He reported for examination September 19, 1959 His vision was O D 20/100 O S 20/20 correctible to 20/25 on the right The proptosis was measured as 6 mm variation between the two corneas There was limitation of adduction on the right The right fundus shows wrinkling of the retina Patient could not wear correcting lenses due to the exophthalmos Blood pressure was 140 mg systolic 80 diastolic

Patient was readmitted to hospital on December 8 1959 There was definite increase of the proptosis of the right eye The vision was 20/80, correctible to 20/40 There is a noticeable exotropia The fundus picture is the same as described above with the wrinkling or stria about the macular area being visible The patient complains of constant pain over the right cheek There is no particular palpable change in the tissues locally (see Fig 6) It is possible that this complaint is based upon the pressure or infiltration of the second division of the Vth cranial nerve This could mean further extension of the granuloma in the middle cranial fossa Examination of the nasopharynx is negative The general condition is excellent except that this patient presents with a blood pressure of 240 systolic, 120 diastolic He is at present under medical treatment which has precluded surgical treatment requiring the administration of a general anesthetic Liver function tests and blood studies are within normal limits There is evidence of some nitrogen retention as indicated by a slightly elevated blood urea nitrogen

TOXICITY

Within the dosage schedule used in the treatment of this patient with amphotericin B intravenously, the toxic effects

patient with a considerable degree of comfort. X-rays following this procedure showed essentially the same findings with the exception of the operative defect. The operative site healed quickly with no complications. For the next six to eight weeks the patient was seen daily, the nose was cleansed, and Nystatin powder again dusted into the operative cavity through the right side of the nose. Tissue cultures again grew fungus. On December 20, 1957, the blood levels of amphotericin B after the daily dosage of 1 mg per kilo body weight were determined to be 0.204 mcg/ml.

Early in February, 1958, the patient began to notice a swollen area just above and anterior to the right ear. This area has continued to be present, however, it varies in size from time to time. The area is not tender and appears to be more of a transitory edema than an extension of the infective process. Vision is 20/100 in the right eye, and 20/20 in the left eye, correctible in the right eye to 20/20, however, the patient is unable to wear lenses because of the exophthalmos. The retina, right eye, shows stria around the posterior pole from retrobulbar pressure. Exophthalmos is 6 mm in the right eye.

The patient has been temporarily retired from active military service. He has been followed on an outpatient status. Tissue biopsies from the lateral wall of the right nasal passage on June 25, August 16, and November 4, 1958, were cultured and have failed to grow Aspergillus. On November 13, 1958, some tissue was removed from the midportion of the floor of the right orbit, going through the right inferior fornix. Three separate specimens were taken. These three fungus cultures were negative after 26 days. The patient's general condition was excellent on examination November 20, 1958. He had a right exophthalmos of 6.7 mm. Vision in the right eye was 20/70 correctible to 20/25, and 20/20 in the left eye. The fundus of the right eye showed horizontal striae or traction lines extending through the macular area. X-rays of the sinuses and skull showed little, if any, change during the last nine months.

Due to the slowly increasing proptosis of the right eye, more evident swelling of the right temple area and more pain in the right cheek, the patient was rehospitalized on February 20, 1959. Despite the recent negative tissue cultures, it was evident there was recurrent activity of the granuloma. Another course of amphotericin B intravenously was begun the next day, using the same dosage schedule as given above. On February 24, 1959, under endotracheal anesthesia the right temporal fossa was explored through a horizontal incision extending posteriorly from the external canthus of the right eye. Granulomatous tissue had eroded the lateral bony orbital wall near the floor about 3 cm posterior to the lateral orbital rim. The involved temporal muscle was removed. The bone of the lateral wall of the orbit was removed. A large granulomatous mass was encountered beneath and lateral to the globe. This was excised by blunt and sharp dissection. There were areas of necrosis in the temporal fossa and in the floor of the orbit. There were also areas of firm fibrotic material. The granuloma extended to the apex of the orbit and beneath this into the pterygomaxillary space. There was direct extension of the fibrous tissue into the right maxillary sinus space. Dependent drainage was established through the floor of the orbit into the sinus and into the nose through the previously placed window in the medial sinus wall. A mushroom tipped catheter was placed in the right maxillary sinus. The superior aspects of the orbit seemed to be free of involvement. A large Penrose drain was sutured in the initial incision. The intravenous administration of amphotericin B was continued postoperatively for a total of 900 mg. Locally, the postoperative course was accompanied by protracted conjunctival edema. Gradually the tissues resumed a fairly normal appearance. The position of the right globe was very nearly normal. Measurements for exophthalmos were less reliable since the right lateral bony rim had been removed.

The microscopic tissue studies show granulomatous inflammation with areas of necrosis. There are predominantly lymphocytes with numerous giant cells, some of these showing hyphae. Tissue culture yielded an abundant growth of the Aspergillus species. Patient was discharged April 14, 1959, after 54 days of hospitalization.

The minimal inhibitory concentration by the multiple tube dilution test for this patient's fungus is given below and is contrasted with the blood levels obtained after the administration of amphotericin B.

Sensitivity M.I.C. (Minimum Inhibitory Concentration)

Tissue Culture of	Amphotericin B	Nystatin
December, 1955	6 mcg./ml.	10 mcg./ml. 40 units
June 1957	3 mcg./ml.	40 units
February, 1959	2.5 mcg./ml.	5.50 units/ml.

Blood concentration (serum activity) of amphotericin B after dosage of 1 mg. per kilo administered intravenously was 0.204 mcg./ml. maintained over a 12 hour period. Obviously this is much below the minimum inhibitory concentration level for this patient's aspergillus infection.

This patient has operated his own truck during the summer and fall of 1959. He reported for examination September 19, 1959. His vision was O.D. 20/100, O.S. 20/20, correctible to 20/25 on the right. The proptosis was measured as 6 mm. variation between the two corneas. There was limitation of adduction on the right. The right fundus shows wrinkling of the retina. Patient could not wear correcting lenses due to the exophthalmos. Blood pressure was 140 mg. systolic, 80 diastolic.

Patient was readmitted to hospital on December 8, 1959. There was definite increase of the proptosis of the right eye. The vision was 20/80, correctible to 20/40. There is a noticeable exotropia. The fundus picture is the same as described above with the wrinkling or stria about the macular area being visible. The patient complains of constant pain over the right cheek. There is no particular palpable change in the tissues locally (see Fig. 6). It is possible that this complaint is based upon the pressure or infiltration of the second division of the Vth cranial nerve. This could mean further extension of the granuloma in the middle cranial fossa. Examination of the nasopharynx is negative. The general condition is excellent except that this patient presents with a blood pressure of 240 systolic, 120 diastolic. He is at present under medical treatment which has precluded surgical treatment requiring the administration of a general anesthetic. Liver function tests and blood studies are within normal limits. There is evidence of some nitrogen retention as indicated by a slightly elevated blood urea nitrogen.

TOXICITY

Within the dosage schedule used in the treatment of this patient with amphotericin B intravenously, the toxic effects

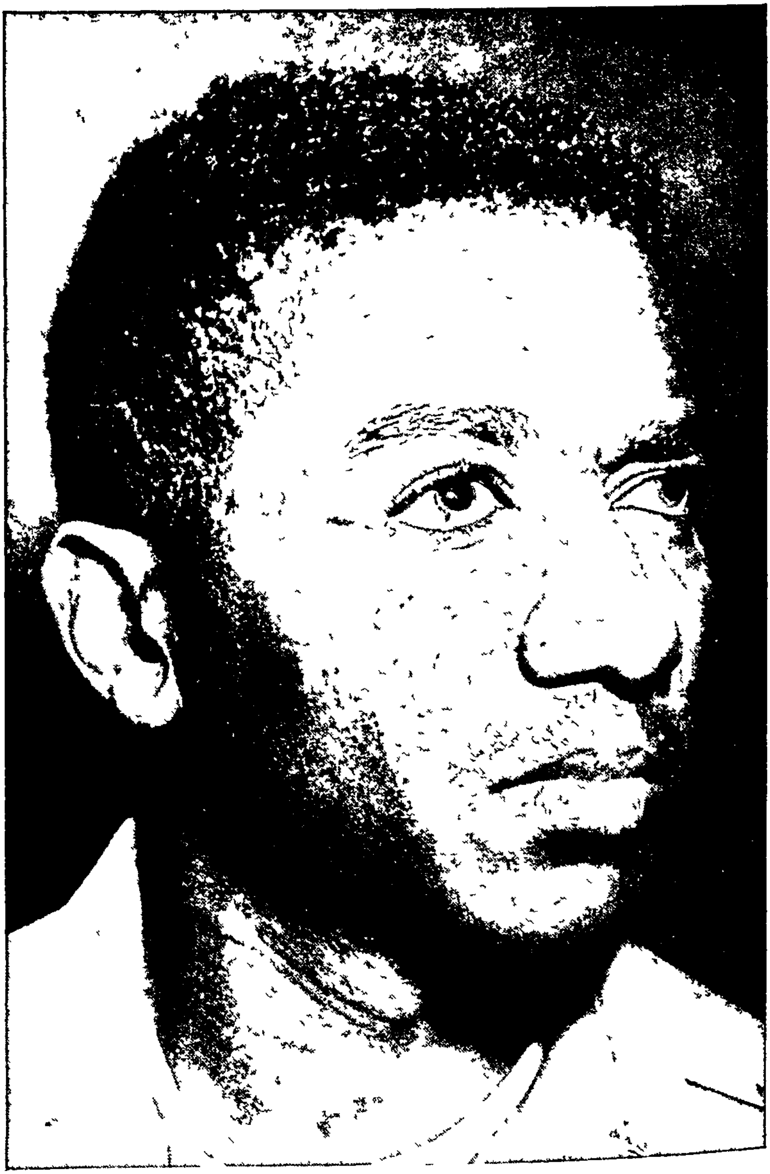

Fig 6 Appearance of patient in December 1959 after surgical decompression of the right orbit below medially and laterally Nystatin locally and three courses of Fungizone There is recurrent fullness of the right temporal area and an increase in the exophthalmos of approximately 6 mm in the past several months This is believed to indicate further growth of the granuloma within the orbit

TABLE I

Outline of Therapy

Date	Clinical Symptoms	Antibiotic Therapy	Surgical Procedures	Nystatin Therapy	Amphotericin B (Fungizone)
Feb, 1955 —	Onset diminishing vision and exophthalmos, right eye				
July 8, 1955—	O D 23 mm O S 19 mm O D 20/50 O S 20/20				
July 20, 1955—		Terramycin, INH Streptomycin Penicillin	Biopsy, right maxillary sinus		
		INH 300 mg Strept gm I q3d to Sept 29			
Aug Dec, 1955—	Increasing scotomata above macula				
Dec 13, 1955—	O D 21 mm O S 16 mm Vision same Increasing pain, right eye	Penicillin and Streptomycin 4 days	Caldwell Luc operation Tissue removed right maxillary and ethmoid sinuses		

TABLE I (Cont)

Outline of Therapy

Date	Clinical Symptoms	Antibiotic Therapy	Surgical Procedures	Nystatin Therapy	Amphotericin B (Fungizone)
Dec 22, 1955—				Powder insufflated into right maxillary sinus daily	
Jan 11, 1956—	Tissue culture, lining right maxillary sinus—no growth				
Feb 15, 1956—	O D 22 mm O S 17 mm Base 102 Altitudinal hemianopsia, same past 2 months Macula spared Macular striations			Powder continued	
April 5, 1956—	Returned to duty Followed as Outpatient Sat Sol KI continued				
Apr 12, 1956—	Tissue culture, lining right maxillary sinus—no growth				
May to Dec, 1956—	Increase in symptoms				
Jan 14, 1957—	Admitted to hospital More pain O D 26 mm O S 18 mm Base 103				
Mar 19, 1957—	Gained 6 lbs			Oral 4 tablets q6h for 12 days, 8 tablets q6h for 5 days	
Apr 23 1957—					1 540 mg i v in 27 days

June 27, 1957—		Achromycin 6 days	Floor right orbit explored Firm tissue removed Local		
July 3, 1957—			Abscess drained through right cheek		
Oct 14, 1957—					4 local injections of ½ mg drug Painful even with 2 per cent procaine
Nov 26, 1957—	O D 26+ mm O S 18 mm O D 20/200 O S 20/20				1,000 mg i v in 23 days
Dec 2, 1957—			Removal floor and medial wall, right orbit	Nose powdered postoperatively	
Apr 15, 1958—	Retired from military service				
Feb 21, 1959—					900 mg i v in 20 days
Feb 24, 1959—			Removal of granuloma, right temporal fossa and orbit Removal lateral bony wall		
Sept 23, 1959—	Retirement re examination				
Dec 8, 1959—	Admitted to hospital 1 Exophthalmos 6 mm 2 Swelling right temple 3 Pain right cheek 4 Hypertension				

observed were principally, *1* a chill and febrile response, *2* sensation of nausea and anorexia with occasional vomiting, *3* headache and general malaise which were lessened by aspirin or aspirin-phenacetin-codeine compound

It should be recalled that this patient's general bodily condition has been excellent His laboratory examinations, including blood, liver function, intestinal cultures, and cerebral spinal fluid tests, have shown little if any variation from average normal findings until the present admission The cause of the elevation of the blood urea nitrogen could possibly be attributed to a late complication of amphotericin B therapy Other cases treated at this hospital with the same drug have not shown this residuum[11] His reaction to this therapy would be expected to be much better than in a chronically ill or emaciated patient

TABLE II

Outline of Therapy

Date of Surgery	Tissue	
	Pathology	Culture
July 20, 1955	Granuloma	
Dec 13, 1955	Granuloma	Positive for Aspergillus flavus
Jan 11, 1956 (Maxillary Sinus Biopsy)		Negative
April 12, 1956 (Maxillary Sinus Biopsy		Negative
June 27, 1957	Granuloma	Positive for Aspergillus flavus
Dec 2, 1957	Granuloma	Positive for Aspergillus flavus
June 25, 1958 (Nasal Biopsy)		Negative
Aug 16, 1958 (Nasal Biopsy)		Negative
Nov 4, 1958 (Nasal Biopsy)		Negative
Nov 13, 1958 (Orbital Biopsy)		Negative after 26 days
Feb 24, 1959	Granuloma	Positive for Aspergillus flavus

SUMMARY

This is a case report of a 31-year-old colored male who noted unilateral exophthalmos in February, 1955 A granulomatous type of tissue was present in the right maxillary sinus which showed no response to antibiotic therapy Cultures of tissue subsequently removed from the right maxillary antrum were positive for Aspergillus—later identified as Aspergillus flavus Nystatin was used locally in the sinus and a solution of potassium iodide taken orally A short course of Nystatin was given by mouth In April, 1957, amphotericin B (Fungizone®) was given intravenously Cultures of tissue removed from the floor of the right orbit were again positive for the same fungus in June, 1957 The tissue removed was much firmer than that previously removed from the right maxillary and ethmoid sinuses The pathological picture was similar Local injections of amphotericin B into the right inferior orbit were quite painful Decompression of the right orbit below and medially again yielded positive tissue fungus cultures in December, 1957 At this surgical exploration there was definite involvement of the pterygomaxillary fossae and about the right optic foramen A second course of amphotericin B was given intravenously Nasal tissue cultures and cultures of tissue removed from the midportion of the floor of the right orbit in November, 1958, have shown no fungus growth after 26 days The patient's general condition has remained excellent The exophthalmos was stabilized at 6-7 mm Due to an increase of the proptosis of the right eye, a lateral decompression of the right orbit was performed February 24, 1959 A large mass of granulomatous material was removed from the inferior portion of the orbit The granuloma extended posteriorly below the optic foramen A third course of amphotericin B was administered Tissue cultures were positive for Aspergillus flavus Patient was re-admitted to the hospital December 8, 1959, for further evaluation

The prognosis in this patient should be guarded Local applications of Nystatin powder and three courses of amphotericin B may have retarded advance of the granuloma but have not halted the process This is believed to be due to the inability to get a sufficiently high blood concentration of the drug to produce a favorable clinical response against this patient's Aspergillus flavus infection

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THE

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VOL LXX AUGUST, 1960 NO 8

CONNECTIVE TISSUE A SYNTHESIS OF MODERN THOUGHT AND ITS IMPACT ON THE UNDERSTANDING OF NASAL DISEASE *†‡

ALEX WEISSKOPF, M D ,

San Francisco, Calif

The past 15 years have witnessed a dramatic change in the concept of body response to disease, primarily due to altered views about connective tissue physiology The present paper is a review of the literature, correlated with clinical experience and substantiated by experimental work A broad understanding of connective tissue can prepare otolaryngologists for the changing concepts of disease and therapy

HISTORICAL BACKGROUND

Virchow stressed the importance of connective tissue and the unity of all types of connective tissue The following quotation from Theophile Bordeaux, a famous scientist of the Eighteenth Century, is an excellent description of the modern concept of connective tissue ground substance "It is the most extensive and useful part of the body It nourishes all the organs It connects one with the other and favors

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*Presented as Candidate's Thesis to the American Laryngological Rhinological and Otological Society Inc March 1959 and read at the Sixty-third Annual Meeting Miami Beach Fla March 17 1960

†From the Division of Otorhinolaryngology University of California School of Medicine San Francisco Calif This study was supported in part by the Breon Fund granted by the Committee on Research University of California School of Medicine

‡Joint recipient of the Harris I Mosher Memorial Award 1960

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and entertains their mutual relations It is the seat of numerous diseases and the site of many manifestations of animal life"[1] Claude Bernard alluded to this in his discussion of the "milieu interne" During the Nineteenth Century, however, the study of cellular activity became more important The concept of connective tissue slipped from a predominant position to that of a supporting structure lacking vitality and activity

Interest in connective tissue grew with the industrial chemists' study of leathers, using the techniques of crystallography and X-ray diffraction Progress was made when Duran-Reynals[2] discovered that testicular extract increased the activity of vaccinal virus In 1933, Klinge[3] compared rheumatic fever lesions with those in animals receiving protein injections and postulated that the pathogenesis of rheumatic fever was due to mesenchymal ground substance alteration induced by allergy to bacteria

The classical presentation of Klemperer, et al ,[4] defined diffuse collagen disease in 1949 He combined various diseases into a single group showing connective tissue alterations Subsequently, the outburst of effort, primarily in the United States, Great Britain and the Scandinavian countries, clarified and filled in a few of the voids in our knowledge of the intercellular spaces

This work is an attempt to organize the vast accumulation of fact and theory into a systematic study, to relate it to nasal problems and to provide a working hypothesis for further experimental study

STRUCTURE OF CONNECTIVE TISSUE

The connective tissue is a continuous matrix, the consistency of which varies from the loose jelly of the umbilical cord to the dense structure of bone Within this matrix lie the interlacing fibrous structures, from which the tissue derives its name, and the cellular elements which appear to control its plasticity and metabolic activity This matrix pervades all tissues Its characteristics are determined by its location Of primary significance is the fact that the connective tissue is continuous throughout the body and that no organ system is free from the environment of the connective tissue

I Cells of the Connective Tissue

A Fibroblasts

These originate from the primitive mesenchymal cells as do all the cells of the connective tissue The morphological characteristics of these cells are described in standard textbooks of histology The fibroblast controls the characteristics of the ground substance and responds to many stimuli in a rapid, active fashion

The fibroblast is the most common cell of the connective tissue It appears to have two important functions the secretion of the precursor of fibers and of the acid mucopolysaccharides in the ground substance Experimentally, the increase in response of the cell was found to parallel the increase in connective tissue ground substance[5,6] Subsequent work has given further evidence of the importance of fibroblasts[7-12]

The name fibroblast is really a misnomer More properly it should be termed a mesenchymal cell differentiated and undifferentiated[13] The present study demonstrates the constant relationship between the undifferentiated mesenchymal cell and the ground substance

B Macrophages

These are a second derivative of the primitive undifferentiated mesenchymal cell and are indistinguishable from the histiocytes, in fact, they are probably histiocytes which have developed phagocytic properties due to external stimuli

C Mast Cells

These cells were given their name by Ehrlich in his first scientific publication in 1877 It has been suggested that the mast cells originate from mesenchymal cells in the adventitia of blood vessels[14,15] The function of these cells is still open to question These cells are particularly striking because of the metachromatic staining of the cytoplasmic granules which and entertains their mutual relations It is the seat of numerous diseases and the site of many manifestations of animal life"[1] Claude Bernard alluded to this in his discussion of the "milieu interne" During the Nineteenth Century, however, the study of cellular activity became more important The concept of connective tissue slipped from a predominant position to that of a supporting structure lacking vitality and activity

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The formation of collagen is still problematical In 1947, Meyer[35] theorized that young fibroblasts secreted both hyaluronic acid and a soluble precursor of collagen into the ground substance Acidification resulted in an insoluble fiber on the surface of which was the polysaccharide As the collagen aged, the polysaccharide thinned and hyaluronic acid was gradually replaced by chondroitin sulfate [36,37]

The electron microscope showed that the fibers have a very distinct periodic striation averaging 640 Angstroms and that collagenous fibers arose from the fibroblasts [38,39] These fibers were embedded in an amorphous mucopolysaccharide ground substance [38,39] Of particular interest was the discovery of procollagen by Russian workers and evidence of formation of collagen *in vitro* [40]

B Reticulin

This substance is identified, as a rule, by silver impregnation Its heaviest concentration is usually in the basement membrane of connective tissue where it forms an argyrophil network of fibers intimately associated with amorphous ground substance Electron microscopy showed that the fibrils have the typical 640 Angstrom periodicity identifying collagen fibers There was also some indication in this study that the reticulin fibers may change into collagen as they mature, forming a fibrous continuum [41]

C Elastin

The exact nature of this fiber is unidentified X-ray diffraction suggested a long chain of protein which appeared to be structurally similar to rubber and other elastic materials [42,43] It was also intimately associated with the amorphous matrix of sulfated mucopolysaccharides and related to the collagens differentiate them from the other connective tissue cells. Jorpes, et al.,[16] correlated the metachromasia with the Schiff positive granules. Since that time, there have been many studies on mast cells, especially by the Scandinavian school as represented by Asboe-Hansen.[17-19] Fawcett[20] showed that the mast cell granules were released as a functional part of the activity of the mast cell. Especially noteworthy was the fact that histamine liberators also caused extensive degranulation of the mast cells.

A number of functions have been attributed to the mast cells. Among these, are:

1. Defense. This has been related to the secretion of heparin by the mast cells. Experimental evidence showed their increased activity in areas irritated by carcinogens.[17,21-24]

2. Fat metabolism. Heparin is known to diminish lipemia. The mast cell relationship to atherosclerosis is still under investigation.

3. Hyaluronic acid secretion. It has been claimed that the mast cells are the primary source of this important ground substance factor. This is questionable and will be discussed later.[25-30]

4. Histamine production.[31,32]

5. Hormonal end organ. There is an increase in mast cells in myxedema and a decrease with thyroxin or in thyrotoxicosis. The controlling factor appeared to be the hormones of the pituitary which caused stimulation of the mast cells.[25,26] The adreno-corticoids caused a decrease in the number of mast cells with degranulation and vacuolation.[26-29,33]

II. Fibrillar Components of Connective Tissue

A. Collagen

This is the chief fibrillar component of connective tissue and consists of bundles of oriented fibers which stain intensely with picrofuchsin and the acid aniline dyes. Although collagen is histologically and chemically identical, it is found in different physical and functional states depending on its location in the body It ranges from the tough, heavy tendons and fascial sheath to the fine supporting lacework of the alveoli in the lung Collagen is identified chemically by its chief amino acid, hydroxyproline, found only in this substance[34]

The formation of collagen is still problematical In 1947, Meyer[35] theorized that young fibroblasts secreted both hyaluronic acid and a soluble precursor of collagen into the ground substance Acidification resulted in an insoluble fiber on the surface of which was the polysaccharide As the collagen aged, the polysaccharide thinned and hyaluronic acid was gradually replaced by chondroitin sulfate[36,37]

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A *Biochemical Studies*

The most intensive work has been done by Karl Mayer and his group[46-54]. The chemical substances forming the gel of the ground substance are the acid mucopolysaccharides which are high polymers of hexosamine. These are long chain flexible polymers with a very high negative charge and a strong affinity for cations and water molecules. Their physical molecular structure gives them a very high viscosity in low concentration. The physio-chemical changes found in the ground substance relate directly to the changes in state of polymerization and pH which govern the gel-sol state.

1. *Hyaluronic acid.* This is the most important mucopolysaccharide. It is found in practically all connective tissue. It is preeminent in the maintenance of the plastic, labile, functionally active ground substance. Hyaluronic acid has been found in various degrees of polymerization, its molecular weight ranging from 20,000 to over 8,000,000. The polymers form coiled chains which are hydrophilic and maintain water in physical union. When depolymerized, either by bacterial or testicular hyaluronidase (the spreading factor), the polymer linkage is broken and yields acetyl glucosamine and glucuronic acid. Hyaluronic acid is the only mucopolysaccharide to be hydrolyzed by bacterial hyaluronidase.

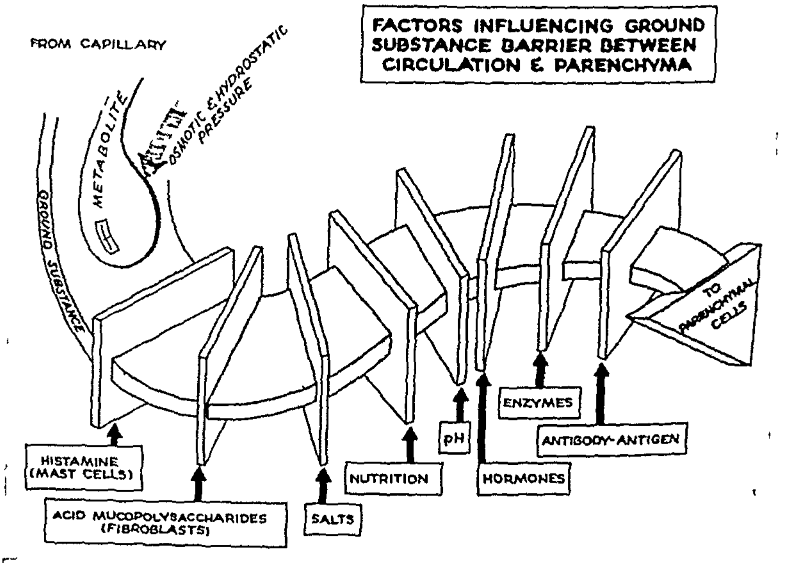
2. *Chondroitin sulfates.* Chondroitin sulfate A is the chief acid mucopolysaccharide of hyalin cartilage. Chondroitin sulfate B is identical with Chondroitin sulfate A except for minor physical properties. It is an important constituent of skin and other loose connective tissue and increases with age. It is associated with heavier formations of gross collagen and diminished tissue activity. It is characterized by resistance to any hyaluronidase activity. The other sulfated mucopolysaccharides are Chondroitin sulfate C, Chrondroitin and Kerato-sulfate.

B *Hyaluronidase Studies*

This is the most important and best studied of all of the spreading factors. This enzyme has the unique property of depolymerizing the acid mucopolysaccharides and significantly

D Oxytalan Fibers

These have only recently been identified by Fullmer and Lillie[14] Their exact significance is still obscure They are located in areas where stress is distributed from one medium to another such as the junction of tendon and bone and at the periodontal membrane


FACTORS INFLUENCING GROUND SUBSTANCE BARRIER BETWEEN CIRCULATION & PARENCHYMA
FROM CAPILLARY
METABOLITE
OSMOTIC & HYDROSTATIC PRESSURE
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TO PARENCHYMAL CELLS
HISTAMINE (MAST CELLS)
ACID MUCOPOLYSACCHARIDES (FIBROBLASTS)
SALTS
NUTRITION
pH
HORMONES
ENZYMES
ANTIBODY-ANTIGEN

Chart 1 A diagramatic presentation of the role of ground substance on the functional state of the parenchymal cells

III Ground Substance in Connective Tissue

This is the most important element of connective tissue activity and it is not demonstrated by ordinary histologic techniques Its very presence was questioned until proven by Bensley[15] in 1934 The ground substance is the amorphous colloid in which lie the cells, fibers and vessels of connective tissue It consists of serum-like components of tissue fluid, local cellular metabolites, hormones, minerals, etc, and the factors distinctive to the connective tissue—the acid mucopolysaccharides The latter are the paramount factors in the functional activity of connective tissue

1 *Inflammatory States* The response of the ground substance to inflammation is of great clinical interest since it leads to an understanding of the fundamental processes in disease It was shown that injury caused immediate increase in capillary permeability with edema, accumulation of lactic acid and drop in pH, followed by an infiltration of blood and tissue cells[59] The invasion of mesenchymal cells paralleled the increase in the acid mucopolysaccharides as shown by increased metachromasia[60] The maximum amount of mucopolysaccharide was found in tissues characterized by glycolytic metabolism[61] Injection of hyaluronidase into rat skin connective tissue resulted in the picture of early trauma The ground substance showed evidence of depolymerization, metachromasia was lost, collagen fibers separated, the mast cells were disrupted and the fibroblasts swollen and vacuolated[62] Hyaluronidase also caused increased permeability in semipermeable membranes[63] Marked increase of tissue hyaluronidase was found after injury[64] Other substances such as proteinases have been implicated in the depolymerization process, which controls the gel-sol state of the ground substance[65] The rapid rate of turnover of hyaluronic acid was shown by S^{35} studies[66-68]

The above experiments have demonstrated two factors involved in early inflammation · one was increased capillary permeability and edema due to histamine and hyaluronidase, the other was depolymerization and tissue disorganization caused by hyaluronidase and other factors A "vicious cycle" was thus started The edema acted to maintain and increase the spreading by hydrostatic pressure[69]

Infection and its spread is particularly pertinent since a large proportion of the inflammation seen by rhinologists is due to infection by bacteria and viruses in the presence of hyaluronidase[2,70] Streptococci from throat infections and otitis media produced hyaluronidase quite consistently[71] No correlation between the virulence of streptococci and the formation of hyaluronidase was found[72-75] Some have even suggested that hyaluronidase has a protective effect[72]

The importance of the hyaluronic acid-hyaluronidase system altering their responses This changes the gel to a sol, causing increased permeability and water solubility with resulting increase in the spread of particles in the tissue

The chief commercial source of hyaluronidase is testicular extract from cattle Other animal sources are snake and leach venom Clinically, its spreading effect has been used in hypodermoclysis and local anesthesia It depolymerizes all the acid mucopolysaccharides except Chondroitin sulfate B Bacterial hyaluronidase is prepared from cultures of streptococci, pneumococci, staphylococci and Clostridium welchii These enzymes will not depolymerize the sulfated mucopolysaccharides

Other substances implicated as spreading factors are mammary gland extracts, thyroid, azoproteins, ascorbic acid and mercurial diuretics among others[55] The nature of their action is still speculative

C Biological Studies

The ground substance and its cells interact in a constant, highly dynamic equilibrium of limited stability Interest in the ground substance was stimulated with the knowledge of the spreading factor Duran-Reynals[2] and Meyer[35] first discussed the interrelationship of hyaluronidase and hyaluronic acid in connective tissue In 1949, Gersh[56] and Catchpole[6] presented one of the early analyses of the connective tissue ground substance They emphasized the physiological activity of the ground substance and response to injury Angevine[57] stressed the importance of the hematoparenchymal barrier A substance passing to the tissues must first travel through the capillary wall, through the connective tissue ground substance, and then through the cell membrane Any change along this path altered the metabolism of the cell The mechanism of fluid transport was demonstrated to be due to molecular capillarity along connective tissue fibrils[58] Edema with increased hydrostatic pressure accentuated the spreading This may explain the activity of many of the anti-spreading factors used clinically such as epinephrine, morphine, the anti-histamines, salicylates, rutin and adrenal-cortical extracts

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The importance of the hyaluronic acid-hyaluronidase system in infection is substantiated by the presence of a non-specific enzyme "anti-invasin" This destroys hyaluronidase and is a part of the essential defense mechanism of the body to prevent invasion during severe infections[76,77] Subsequent work has shown that there was an increase in the inhibitor in many unrelated infections[78,79] The increased serum inhibitor appeared to be a response of the organism to hyaluronidase released by the tissues themselves[80] The anti-hyaluronidase role of heparin was suggested by the concomitant increase of heparin and "anti-invasin" in the blood[81,82]

It has been demonstrated that the increase in acid mucopolysaccharides is a step in the process of inflammation This reaction is non-specific and was found in a wide variety of diseases including rheumatic fever, arthritis, diabetes, myxedema, pregnancy, allergic reactions, prolonged administration of estrogen, androgen or desoxycorticosterone, inflammatory lesions due to bacteria or viruses, cold, trauma, anoxia and radiation[61] The degree of response depended upon tissue specificity Some investigators suggest that the eventual stigmata of these diseases was due to deterioration of the acid mucopolysaccharide itself[3,4,6 19,56 83-86] Others, particularly the German school, believed that the ground substance was not important in the formation of fibrinoid but that it was due to the influx of extrinsic material from fibrin or serum protein[87-89]

2 Hormonal Effect on the Ground Substance Response of connective tissue to hormones has been observed clinically for many years Rhinologists are aware of the tissue changes associated with the menstrual cycle and pregnancy[90,91]

a Adreno-corticoids The theory of the General Adaptation Syndrome by Selye[92] in 1946, aroused general interest in the adrenal corticoids Cortisone and ACTH depressed the activity of all connective tissue elements[28,93-97] The decrease in acid mucopolysaccharides as demonstrated by the absence of metachromasia has been considered a prime factor in delaying fibroplasia and healing[98] Lack of hyaluronic acid diminished the tensile strength of wounds[99] Its addition to wounds inhibited by Cortisone stimulated fibroplasia[100] Corticoids also diminished the activity of the mast cells[34 101] Atrophy of epidermal structures has been reported[102] showing the close functional relationship of connective tissue to neighboring structures Cortisone acts at the level of the cell membrane and may be an "antihyaluronidase" factor The permeability of semi-permeable membranes was decreased by the corticoids[63] This effect was seen on capillary permeability[103] and was associated with vasoconstrictive factors on an autonomic basis[104, 105] It caused decreased invasiveness of experimentally produced carcinomata[106]

Desoxycorticosterone caused a reversal in response The granulation tissue was exuberant with young giant fibroblasts (mesenchymal cells) showing great activity and with a heavy ground work of collagen Similar results were seen with testosterone[107] Anterior pituitary somatotropic hormone also prevented the inhibitory effect of Cortisone on granulation tissue[108] and the massive systemic infection of multiple abscesses seen after giving Cortisone and ACTH to experimental animals[109] Hypophysectomy decreased the production of hyaluronic acid and chondroitin sulfate[110] Anterior pituitary growth hormone increased this acid mucopolysaccharide production[110 111] The adrenal-pituitary axis appears to be an important and delicately balanced mechanism in the activity of the connective tissue

b Thyroid hormone. Articles in the past have alluded to the role hypothyroidism plays in vasomotor rhinitis[112-114] The use of thyroid hormone in otolaryngological conditions has been questioned Walsh,[115] in 1950, advocated the use of thyroid hormone as determined by history and clinical findings Further evidence relating the hypometabolic state to thyroid hormone has been presented on a theoretical basis[116] Increase in hyaluronic acid and chondroitin sulfate was found in pretibial myxedema[117] and in hypothyroid myxedema[118] This has been explained by the action of the thyrotrophic hormone which stimulated acid mucopolysaccharide production[119 120]

c Sex hormones The relationship between sex hormones and the nose has received clinical attention[90 91 121] The changes in the sex skin of the Macaco monkey are related to the menstrual cycle This was shown to be controlled by estrogens[30,122] The material causing the edema was hyaluronic acid and hyaluronidase caused immediate softening[123] Testosterone presented an identical picture in the cock's comb[124] and has been related to gonadotropic hormones[125] Gonadotropin decreased polymerization with increased tissue permeability and spread of tuberculosis in rabbits[126] The gonadotropic-sex hormone mechanism probably involves the adrenal cortex as well[127]

Relaxin, a sex hormone associated with pregnancy, has a profound effect on the connective tissue It has a specific action on the connective tissue of the symphysis pubis, loosening the joint in preparation for delivery[128] On theoretical grounds this evidence for specificity of connective tissue response could explain the changes in the nasal and laryngeal tissues due to hormonal factors

d Parathyroid hormones The parathyroid glands have been involved in connective tissue ground substance Parathyroid extract depolymerized the ground substance Cases of fibrous dysplasia showed a high serum mucoprotein and suggested the interrelationship of the parathyroid and the ground substance[129,130] Evidence permits only speculation as to the possible etiology of otosclerosis, a connective tissue disease The localization of the otosclerotic focus may be a specific connective tissue response in the embryonal cartilage of the fissula ante-fenestrum

3 Ascorbic Acid Ascorbic acid is the primary vitamin implicated with connective tissue functions as shown by the clinical course of scurvy with increased capillary fragility, petechiae and poor wound healing The ground substance was depolymerized as evidenced by a rise in serum glycoprotein Normal collagen was not formed and the existing collagen was weakened due to its abnormal matrix[131-136] Ascorbic acid is an essential factor in connective tissue formation as shown by its chemical relationship to hyaluronic acid[137] and the similarity between scorbutic tissue and that of collagen diseases[138]

HISTOCHEMICAL TECHNIQUES

The combined efforts of biochemists, biophysicists, anatomists, histologists and the clinicians have developed many ways to study connective tissue. All of these methods have very distinct limitations which have been constantly re-emphasized by workers in the field.[46,139] The present study has stressed histochemical methods[140-153] as the most useful for clinical study.

Histochemical technique differs from ordinary histologic technique in that the structural organization of the tissue is correlated with its functional state. The limitations of histochemical studies have frequently resulted in the completely contrary findings reported in the literature. A number of factors may explain this. One of the most important is that the presence of many constituents in the tissue, such as protein, may obscure the stain.[154-156] Some of the investigations on histochemical stains were done on chemically pure substances and in many instances, the results differed from those found in tissue[150,157] since chemically pure substances are never found in life. When the stains were used on the tissue complexes, they were effective.[158,159]

I. Stains

Three stains have been most widely used because of their specificity, sensitivity and reproducibility.[11]

A. Periodic Acid Schiff (PAS)

This stain[140,141] was considered to be the least specific in this study. It stains the acid mucopolysaccharides and also stains glycogen, mucoproteins, glycoproteins, collagen, gelatin, reticulin, plasma and serum.[101] The PAS material stains a vivid pink to red. Using the controlled simultaneous preparation method,[11] no difference was found between the staining characteristics of tissue, prepared serially, with and without hyaluronidase. The experiment showed that factors other than acid mucopolysaccharides were stained; therefore, the PAS stain was not used in the present study.

B Metachromatic Reactions

The most common dyes used are the basic dyes such as thionin, azure A or toluidine blue Ehrlich used the term "metachromasia" to indicate that certain tissues and structures stain in a different color than that of the original dye solution The alteration is based upon electro-chemical changes in the dye molecule[160] The metachromatic reaction is an important source of information about the acid mucopolysaccharides of the ground substance They will stain metachromatically, depending upon two factors the state of polymerization and the amount of sulfation The toluidine blue method was used in this study

C Colloidal Iron Adsorption

This staining technique was first reported by Hale[142] in 1946 and has been modified[145] Acid mucopolysaccharides will adsorb colloidal iron which then stains positively with the Prussian blue reaction of ferrocyanide The collagen is stained with picro-fuchsin A modified Rinehart and Abul-Haj stain[118] was used in this study

EXPERIMENTAL DATA

This work originally began as a study of nasal tissue in a variety of vasomotor states An attempt was made to correlate vasomotor rhinitis with various conditions involving the endocrines such as pregnancy, hypothyroidism and changes of the menstrual cycle It was discovered that the routine histologic stains presented little information about the functional state The development of histochemical stains provided the means for visualization of these changes

I Normal Turbinate Mucosa

A series of biopsies were taken from normal middle turbinates No vasoconstricting agents were used The normal tissues were used to develop a standard technique and to study the variations in the range of normal connective tissue, Weisskopf and Burn[11]

Metachromasia was demonstrated with toluidine blue The most striking violet color was in the intima of the arterioles and the connective tissue immediately around these vessels Associated with the vessels were the very prominent mast cells They were large, round or oval cells with the nucleus almost completely obscured by the concentration of deep violet granules Occasional mast cells were seen with granules scattered into the surrounding medium, suggesting an actual secretion by these cells rather than mere mecnanical disruption An occasional mast cell was also seen in the lamina propria among the mesenchymal cells, fibroblasts and other cellular components The average fibroblast is small and dense with minimal cytoplasm visible Its nucleus is pyknotic without the prominent nucleolus seen in the active fibroblast The ground substance of the lamina propria did not stain nor did the basement membrane It appeared to have a perfectly clear background except for the faint suggestion of fine blue fibrils The mucous glands varied in staining reaction from the orthochromatic blue to a very vivid metachromatic violet Of interest was the metachromasia seen in the ciliated epithelium, mainly concentrated near the ciliated border, indicating their functional activity in the production of mucus Incubation with testicular hyaluronidase (Wydase®)* substantiated the experimental evidence There was loss of metachromasia in and about the blood vessels but no change in the mast cells or the mucus producing cells With bacterial hyaluronidase, there was a persistence of faint metachromasia about the vessels This suggests that both hyaluronic acid and chondroitin sulfate B were present The heparin in the mast cells remained unchanged as did the neutral mucopolysaccharides of the mucus

The colloidal iron stain corroborated and enriched the picture derived from the toluidine blue stain Again the greatest concentration of acid mucopolysaccharides was found in and among the blood vessels It was seen to form an important part of the structure of the vessel wall The fibrillar material, now determined to be collagenous by its brilliant red stain was surrounded by the fine blue mucopolysaccharide layer which pervaded everywhere The clear ground substance seen with toluidine blue was found to contain fine collagen granules and the acid mucopolysaccharides The colloidal iron stain distinguished the unsulfated and more depolymerized acid mucopolysaccharides as compared to the toluidine blue The basement membrane was beautifully demonstrated and given some functional significance It was approximately seven to 15 micra thick and was seen to consist of fine amorphous blue staining ground substance intermingling with the red fibrils of reticulin There was a continuity between these fibrils and the heavier collagen of the lamina propria The greatest density of ground substance was found in the peripheral half of the basement membrane, gradually becoming less blue as it streamed into the lamina propria The mucus likewise stained blue Hyaluronidase incubation was again enlightening There was a marked "washing out" of the background, revealing fine, red interlacing fibrils and fibers of collagen The reticulin network of the basement membrane was more evident There was persistence of some fine blue staining in the basement membrane as well as in the immediate vicinity of the vessels

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*Wydase® was kindly supplied by Wyeth Laboratories Inc

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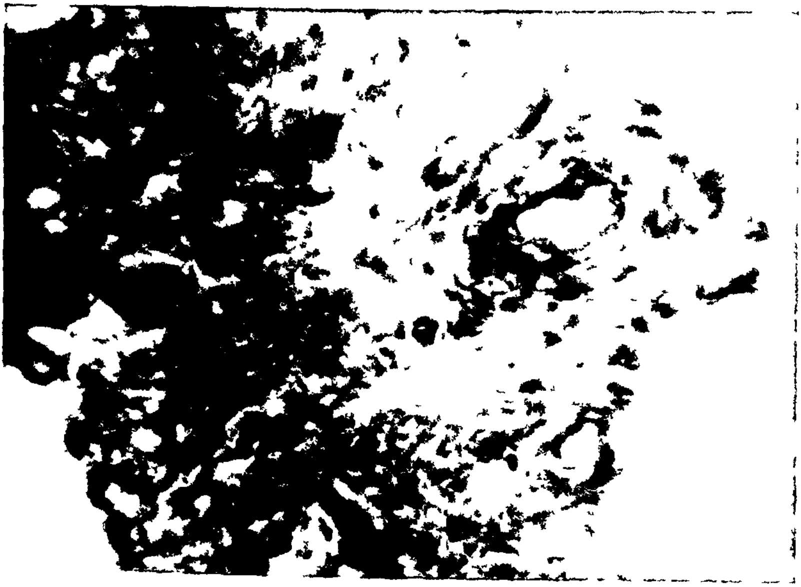
II Allergy and Nasal Polyps

Once the consistency of these histochemical techniques was determined, the study was extended to various disease states The relation of allergy to mesenchyme has been postulated on theoretical grounds[161,162] Evidence suggesting nasal connec-



1 Normal middle turbinate 430x Colloidal Iron The ground substance of the basement membrane and lamina propria are stained blue The collagen fibers stain red

2 Normal middle turbinate 430x Colloidal Iron after Hyaluronidase incubation The loss of blue stain confirms the presence of Hyaluronic acid The persistence of a fine blue residue in the ground substance and blood vessel wall suggests the presence of Chondroitin Sulfate B

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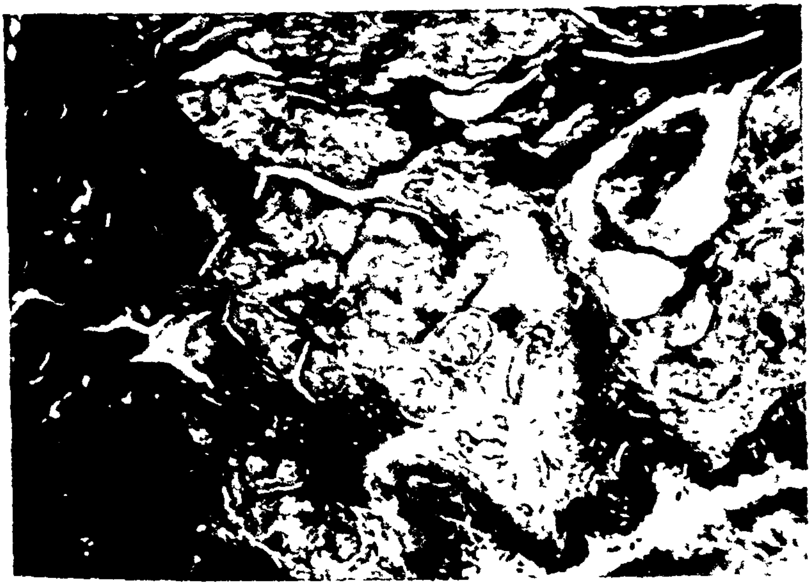
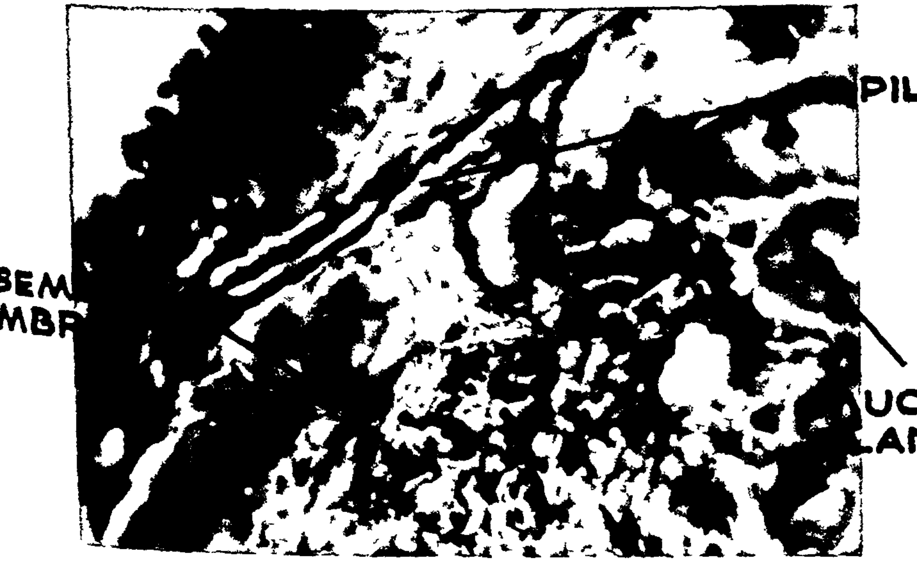
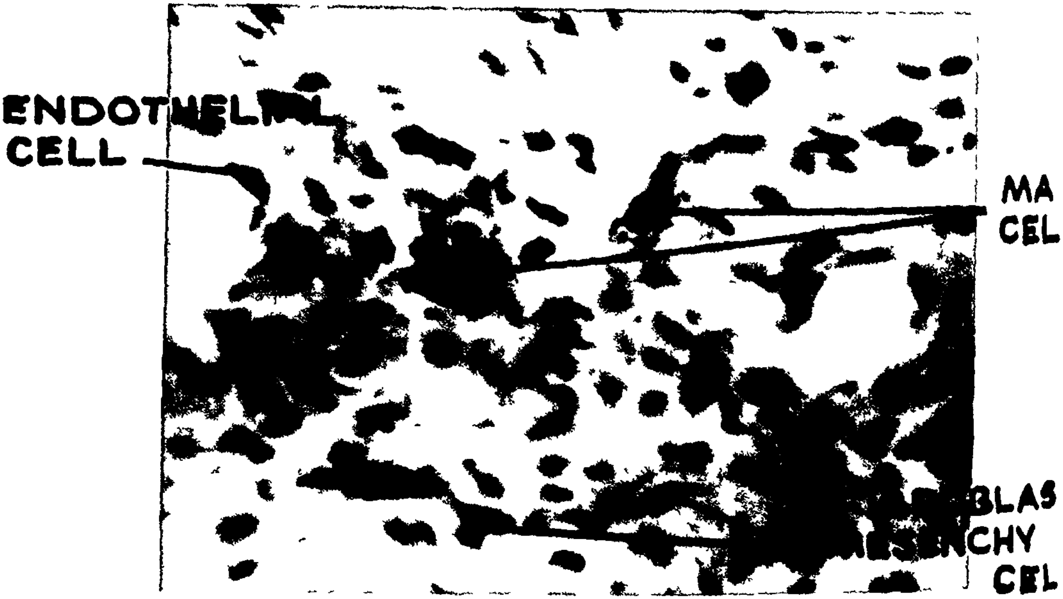


5 Middle turbinate, hypothyroid 100x Colloidal Iron Note increased mucous glands and the density of the blue staining ground substance



6 Middle turbinate, hypothyroid 430x Colloidal Iron The increased ground substance of the basement membrane and lamina propria is striking



3 Normal middle turbinate 970x Toluidine blue The metachromatic granules of the mast cells are striking There is a faint metachromasia in and about the capillary walls

4 Laryngeal granuloma 970x Toluidine blue The large fibroblasts (mesenchymal cells) and the surrounding metachromatic ground substance reveal the active nature of this granulation tissue as compared to the normal Fig 3

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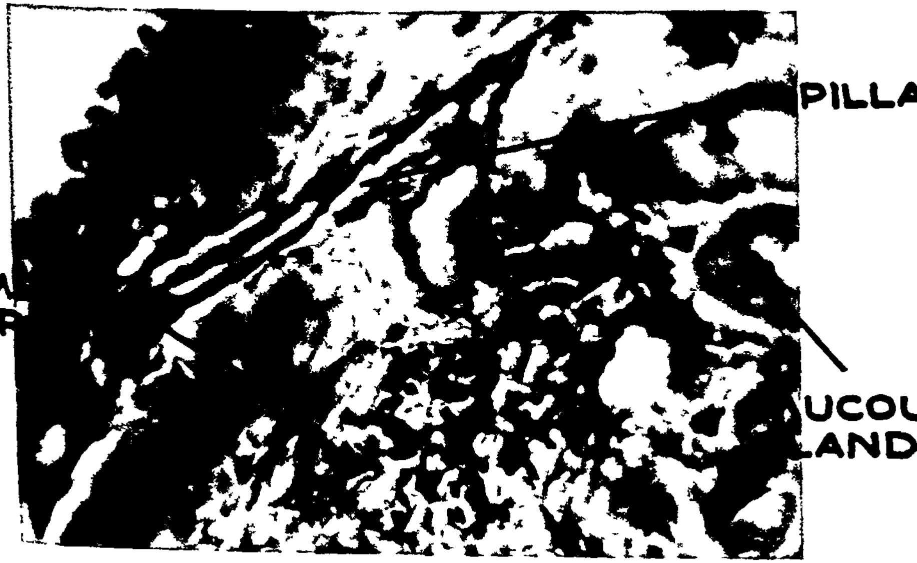

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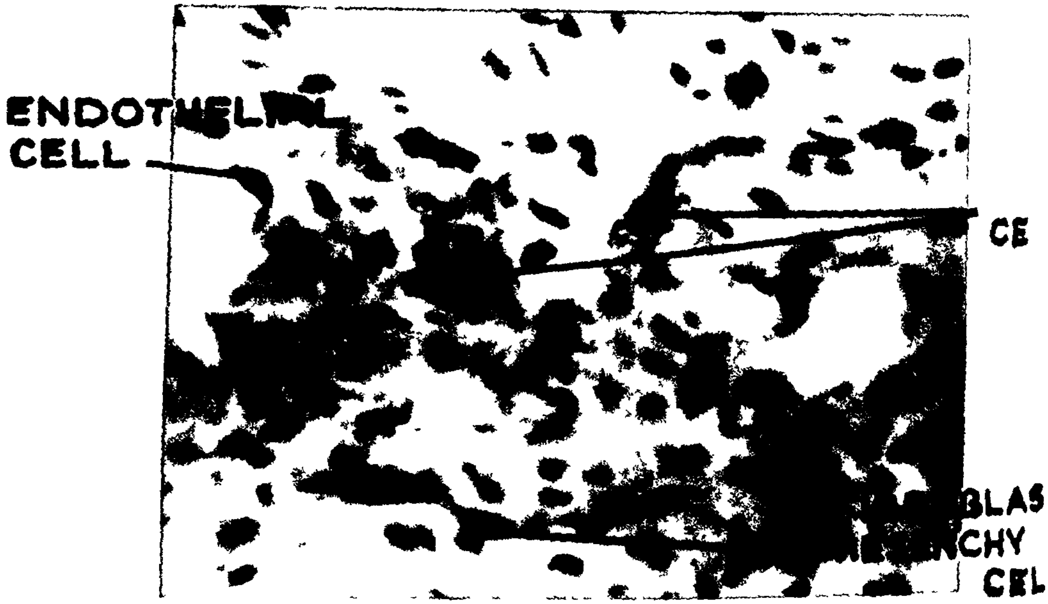

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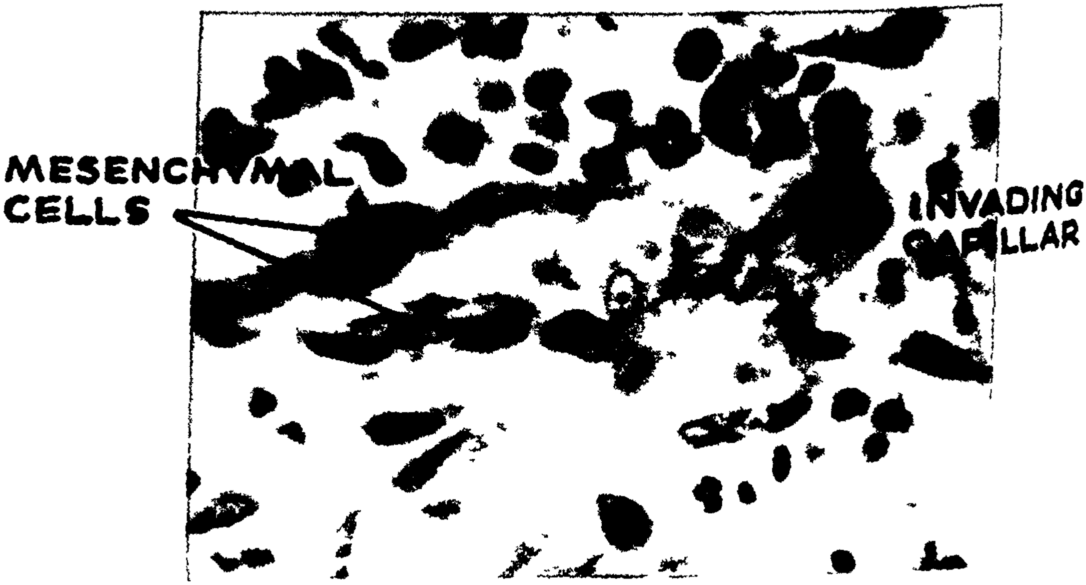
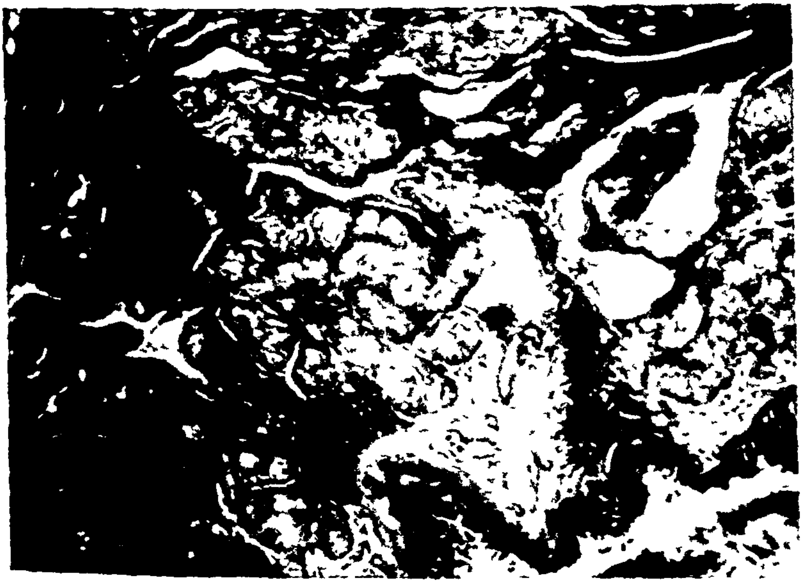
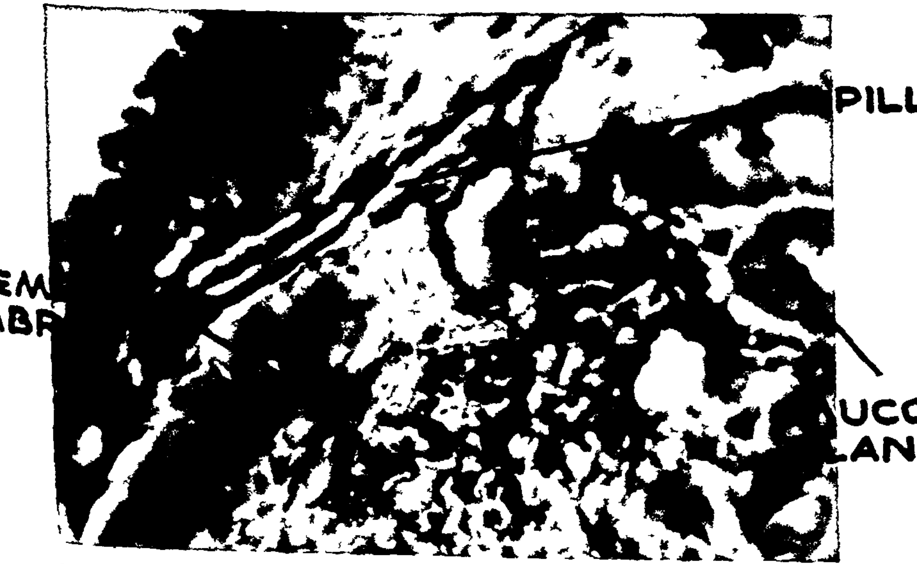


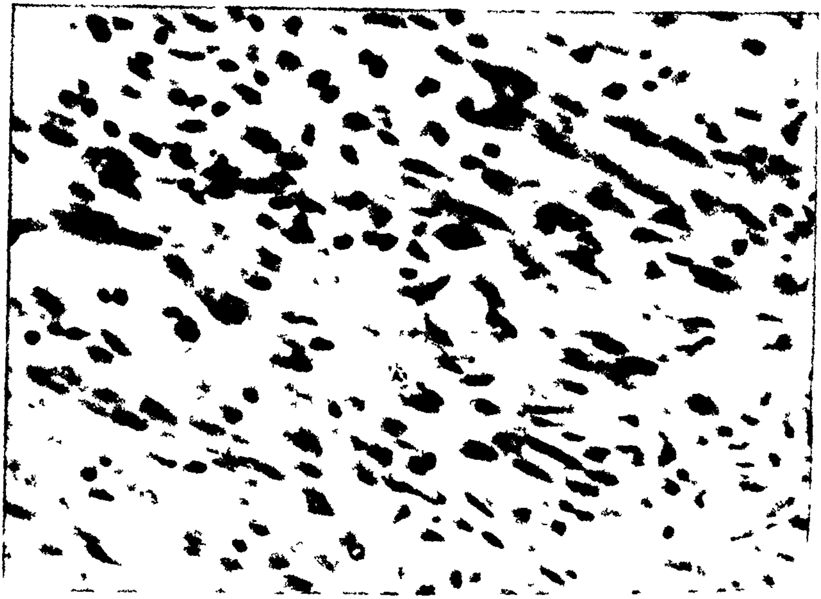
4 Laryngeal granuloma 970x Toluidine blue The large fibroblasts (mesenchymal cells) and the surrounding metachromatic ground substance reveal the active nature of this granulation tissue as compared to the normal Fig 3



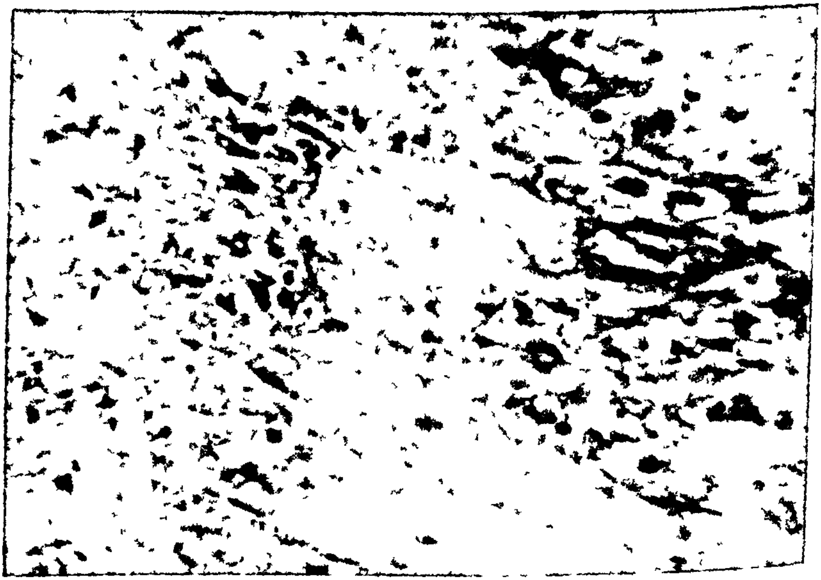
5 Middle turbinate, hypothyroid 100x Colloidal Iron Note increased mucous glands and the density of the blue staining ground substance



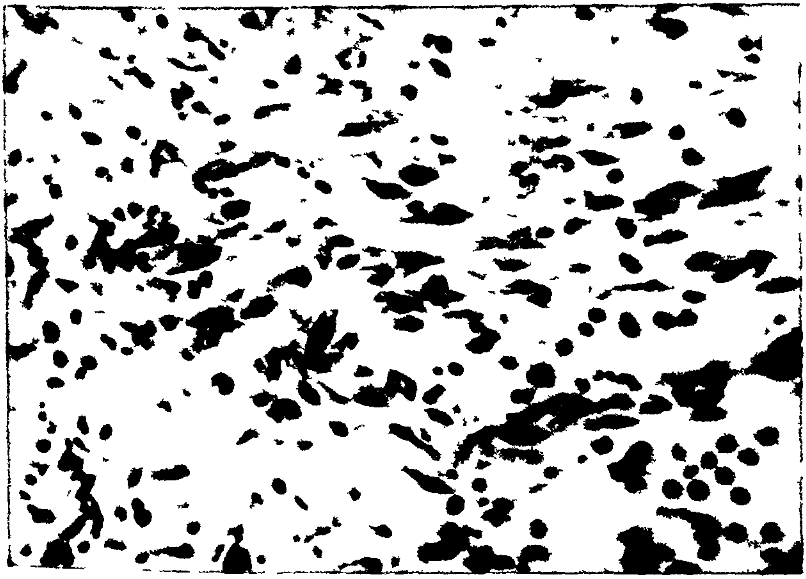
6 Middle turbinate, hypothyroid 430x Colloidal Iron The increased ground substance of the basement membrane and lamina propria is striking



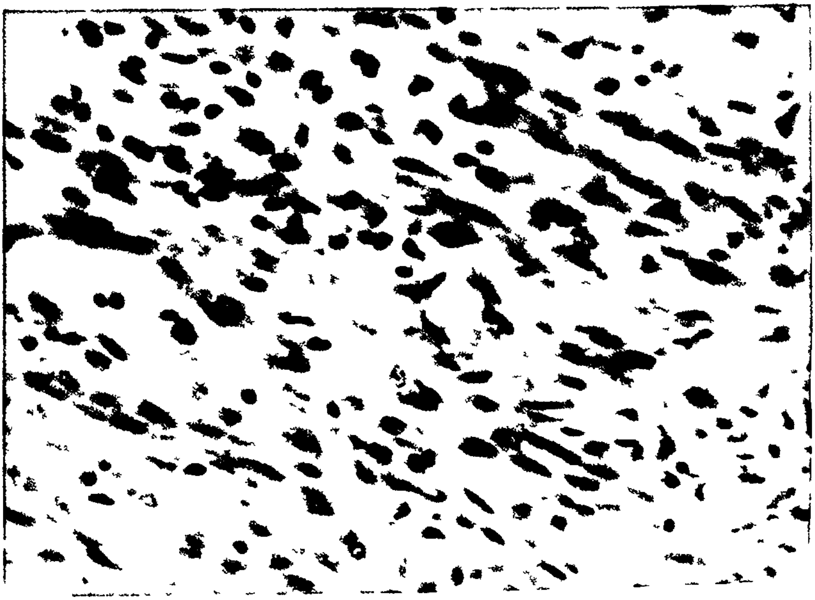
7 Laryngeal granuloma 430x Hematoxylin and eosin The invading capillaries and mesenchymal cells are seen but no evidence of ground substance activity



8 Laryngeal granuloma 430x Colloidal Iron Compare with Fig 7 The streamers of fibroblasts are embedded in the acid-mucopolysaccharide



9 Laryngeal granuloma 430x Toluidine blue The metachromasia about the mesenchymal cells correlates well with the blue stain of Fig 8



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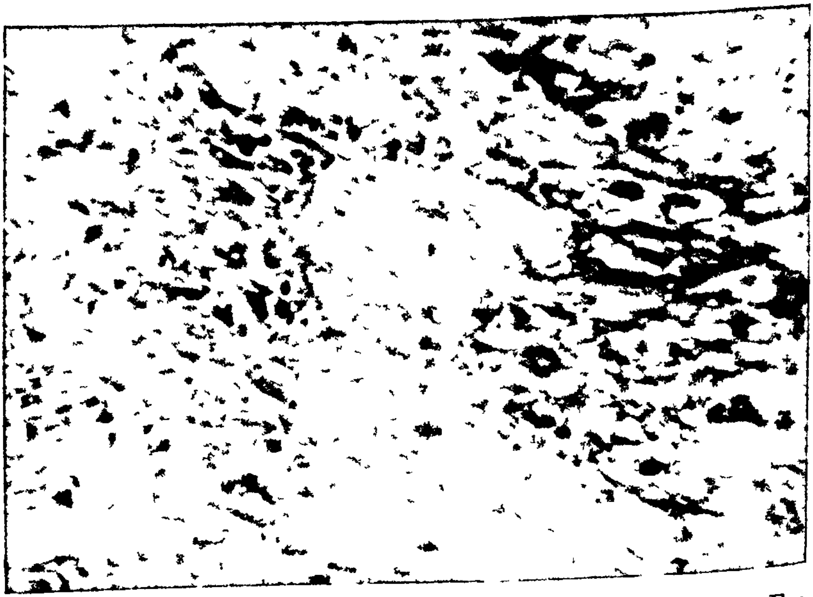

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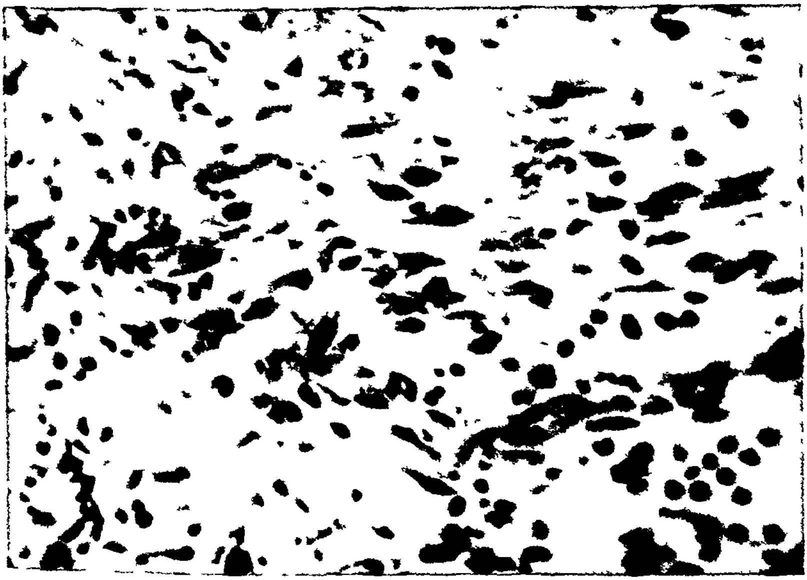

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tive tissue breakdown and depolymerization in allergy was presented in 1953[163] Further evidence was presented[164] that the ground substance was involved in allergy and in the formation of nasal polyps Study of nasal polyps gives considerable insight into the formation of the polyp and the functional mechanisms in the connective tissue The polyp was found to contain acid mucopolysaccharides in varying states and concentrations even in the so-called edema fluid Areas of increased activity in polyps were characterized by "puddles" of acid mucopolysaccharides showing metachomasia Almost invariably, this was associated with the large active mesenchymal fibroblasts which are assumed to be the source of the mucopolysaccharide Mast cells did not appear to play an important role and were conspicuous by their rarity On the other hand, this might indicate their extreme activity with the absence of granules making them unrecognizable There was relatively little collagenous material as compared to the normal nasal mucous membrane The basement membrane was atrophic, absent or frequently replaced by collagenous fibers with relatively little ground substance The picture suggests activity of the connective tissue with formation of an abnormal ground substance This does not form the normal collagen associated with inflammatory changes Further work is contemplated, involving ascorbic acid and allergies

III *Infection*

A difference was found between acute and chronic infection suggesting the steps leading to collagenous healing The acutely infected membrane showed the extensive infiltration of acute inflammatory cells, edema and areas of granulation tissue showing new capillary formation The large accumulation of mucopolysaccharides was impressive in the areas of capillary formation and among the concentrations of the large mesenchymal cells These, characteristically, showed foamy and granular cytoplasm and a large nucleus and nucleolus

The chronic infection showed the presence of increased ground substance mucopolysaccharides, interspersed with dense collagenous fibers The total amount of ground sub-

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The chronic infection showed the presence of increased ground substance mucopolysaccharides, interspersed with dense collagenous fibers The total amount of ground substance was less than in the acute stages It presented a vivid picture of the outcome of chronic infection with collagen formation scarring and decreased vascularity The decreased ground substance activity results in the eventual inability of the tissues to respond to the stress of infection

IV Pregnancy

The most striking feature in the vasomotor rhinitis of pregnancy was the bulk of mucous glands which appeared to fill practically the entire section This was associated with numerous large cavernous areas With toluidine blue, the slides appeared to be "washed-out" having practically no areas of metachromasia even in the blood vessels They appeared more like normal tissues which had been subjected to hyaluronidase The colloidal iron stains substantiated this impression There was a definite decrease in the amount of ground substance The basement membrane was either atrophic or non-existent All the cases were from the fourth to the seventh month of pregnancy, a period associated with increased estrogens To understand these changes, the gonadotrophic hormone levels must be considered

V Thyroid Dysfunctions

It was difficult to obtain turbinate specimens in clear-cut cases of glandular dysfunction which had not already been treated Four cases were studied two hypothyroid (one with true myxedema), one thyrotoxicosis and one case with complete ablation of pituitary activity by Synchrotron irradiation The most persistent finding in all cases was the hypertrophy of the mucous glands The hypothyroid states, including the dyspituitary, showed a marked increase in ground substance with the colloidal iron stain

Turbinate tissue from the patient with pituitary ablation showed the most striking "puddling" of amorphous material, obliterating the normal lamina propria in many areas There was almost complete absence of the normal cellular infiltration The majority of cells present were the large mesenchymal cells associated with formation of ground substance Even the normally flat endothelial cells of the capillaries showed large nuclei with prominent nucleoli very similar to those of the mesenchymal cells Since theoretically, no thyrotropic hormone is secreted after pituitary irradiation, this suggests the relationship of other hormones to ground substance activity The increase in ground substance of the other two hypothyroid cases was not as dramatic, but was distinctly more than normal The thyrotoxicosis case showed less than normal ground substance as evidenced by the visibility of the collagenous material in the lamina propria and the basement membrane

The increased turgidity of the turbinates in the hypometabiloc states was reflected in an increase in amount and polymerization of the ground substance Associated with this was a mucous gland hypertrophy, explaining the excessive nasal secretions Both pregnancy and hypometabolic states are associated with hyperactivity and hyperdevelopment of mucous glands The relationship of the mucous glands to these various states deserves further study

VI Atrophic Rhinitis

The most prominent change in the three cases was the metaplasia into stratified squamous epithelium The absence of mucous glands, the arteritis, and the collagen deposits in the walls of the arterioles were striking There was an increase in the amount of mucopolysaccharide of the ground substance associated with an increased amount of collagen This was paralleled by a marked increase of mesenchymal cells of the large primitive type and increased numbers of small capillaries There was no lack of vascularity to explain this lesion Of particular interest was the presence of a large number of mast cells In selected areas, the picture of a blood vessel associated with collagen, active fibroblasts and mast cells was reminiscent of juvenile angiofibroma[16]

Treatment with corticoids caused a remission in clinical symptoms and showed a dramatic change in the turbinate There was diminution in all elements of the connective tissue The ground substance appeared less dense There were fewer mesenchymal cells present and those remaining appeared smaller and more typically mature There was diminution in mast cells as well as in the density of inflammatory cell concentration At least part of the therapeutic effect of the corticoids[166-168] can be seen to be due to their inhibiting action upon the connective tissue components and corroborates the experimental work cited in the literature

VII Granuloma

Lastly, a case of laryngeal granuloma was studied to compare a naturally occurring granulation tissue with those experimentally produced and reported in the literature The findings were essentially those described by other workers[9,60,84,93 98,99] and helped corroborate the authenticity of our own histochemical stains and interpretations The laryngeal granuloma was quite typical histologically Histochemically, the greatest amount of ground substance appeared in the greatest area of growth activity This was the junction between the amorphous fibrinous debris and the invading capillaries and mesenchymal cells The same typical, large fibroblasts were seen and again appeared to be related to the formation of the acid mucopolysaccharides There was practically *no evidence of mast cells in significant* numbers

DISCUSSION

Evidence has been presented that connective tissue is an anatomical, functional and biochemical unit Since the connective tissue matrix is all pervasive, it must eventually be involved in all body processes

Attention has been directed to the characteristic response of connective tissue in a variety of diseases and functional states This response consists, primarily, of a series of physico-chemical changes in the acid mucopolysaccharides of the ground substance and is related to the activity of the mesenchymal cell This intricate process is outlined in a hypothetical diagram in the accompanying chart (see Chart 2)

Experimental data is presented which suggests, at least within the limits of a preliminary survey, that many of the pathologic states of the nasal turbinates are reflected in connective tissue changes. Vasomotor rhinitis, to name one, is more clearly understood in the light of these changes. This was predicted on theoretical grounds.[11] The close interrelationship of the nasal tissues to other body functions is also demonstrated on the histologic level.

It is felt that these studies may have clinical application. There is evidence that a variety of materials affects the ground substance in general. These range from the chemical to the hormones and vitamins. It would be expected that some of these should exert a similar effect upon the nasal ground substance and may provide suggestions for the utilization of new therapeutic agents. The histochemical picture presents evidence of functional changes in the tissues and may be used to study the pharmacological actions of the agents. This was done in one case of atrophic rhinitis treated with prednisolone.

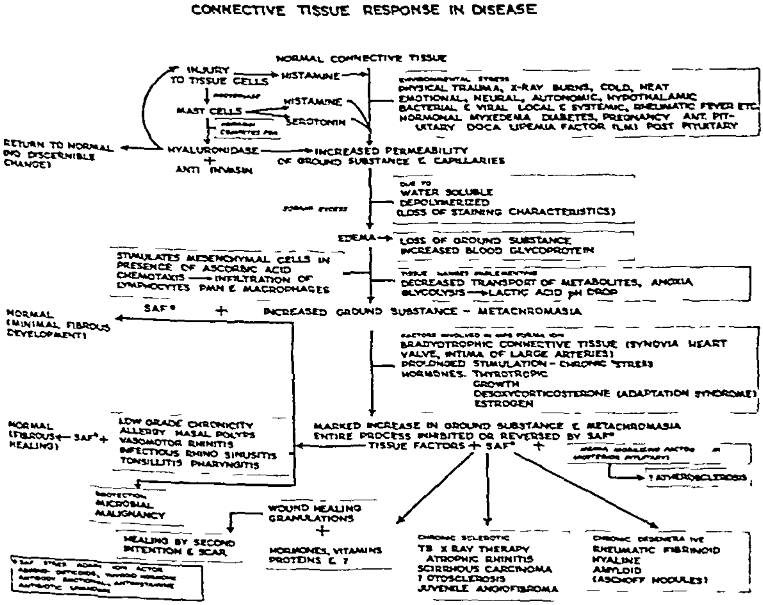
CONNECTIVE TISSUE RESPONSE IN DISEASE
NORMAL CONNECTIVE TISSUE
INJURY TO TISSUE CELLS
HISTAMINE
MAST CELLS
HISTAMINE
SEROTONIN
HYALURONIDASE + ANTI INVASIN
RETURN TO NORMAL (NO DISCERNIBLE CHANGE)
PHYSICAL TRAUMA, X-RAY BURNS, COLD, HEAT
EMOTIONAL, NEURAL, AUTONOMIC, HYPOTHALAMIC
BACTERIAL & VIRAL LOCAL & SYSTEMIC, RHEUMATIC FEVER ETC.
HORMONAL MYXEDEMA DIABETES, PREGNANCY ANT. PITUITARY DOCA LIPEMIA FACTOR (LM) POST PITUITARY
INCREASED PERMEABILITY OF GROUND SUBSTANCE & CAPILLARIES
WATER SOLUBLE
DEPOLYMERIZED
(LOSS OF STAINING CHARACTERISTICS)
EDEMA
LOSS OF GROUND SUBSTANCE
INCREASED BLOOD GLYCOPROTEIN
STIMULATES MESENCHYMAL CELLS IN PRESENCE OF ASCORBIC ACID
CHEMOTAXIS → INFILTRATION OF LYMPHOCYTES PMN & MACROPHAGES
DECREASED TRANSPORT OF METABOLITES, ANOXIA
GLYCOLYSIS → LACTIC ACID pH DROP
SAF* + INCREASED GROUND SUBSTANCE – METACHROMASIA
NORMAL (MINIMAL FIBROUS DEVELOPMENT)
BRADYOTROPHIC CONNECTIVE TISSUE (SYNOVIA HEART VALVE, INTIMA OF LARGE ARTERIES)
PROLONGED STIMULATION – CHRONIC "STRESS"
HORMONES. THYROTROPIC
GROWTH
DESOXYCORTICOSTERONE (ADAPTATION SYNDROME)
ESTROGEN
MARKED INCREASE IN GROUND SUBSTANCE & METACHROMASIA
ENTIRE PROCESS INHIBITED OR REVERSED BY SAF*
TISSUE FACTORS + SAF* +
? ATHEROSCLEROSIS
LOW GRADE CHRONICITY
ALLERGY NASAL POLYPS
VASOMOTOR RHINITIS
INFECTIOUS RHINO SINUSITIS
TONSILLITIS PHARYNGITIS
NORMAL (FIBROUS HEALING) ← SAF* +
MICROBIAL
MALIGNANCY
WOUND HEALING GRANULATIONS + HORMONES, VITAMINS PROTEINS & ?
HEALING BY SECOND INTENTION & SCAR
TB X RAY THERAPY
ATROPHIC RHINITIS
SCIRRHOUS CARCINOMA
? OTOSCLEROSIS
JUVENILE ANGIOFIBROMA
RHEUMATIC FIBRINOID
HYALINE
AMYLOID
(ASCHOFF NODULES)

Chart 2

The ear, nose and throat are readily studied since specimens may be obtained in a functional state at surgery and from biopsies[11,163,168,169] This contrasts with other investigations which have used animal experiments or post mortem material

SUMMARY AND CONCLUSIONS

1 A review of the literature presents evidence that connective tissue behaves as an anatomical and functional unit

2 The ground substance is the most important component It responds characteristically to a wide variety of conditions The degree and site of response depends upon tissue specificity

3 The mesenchymal cell is considered to be the ultimate source of the acid mucopolysaccharides, the active factor in the ground substance

4 Histochemical investigation of turbinate biopsies in a number of disease states reveal connective tissue changes that correlate well with the experimental findings of investigators in other fields

5 The connective tissue changes observed add to a further understanding of the processes involved in the vasomotor rhinitis of allergy, pregnancy and endocrine dysfunction

6 Histochemical methods were suggested to evaluate therapeutic agents One case of atrophic rhinitis, treated with prednisolone, was presented

7 A new approach to the study of such connective tissue diseases as laryngeal nodules and otosclerosis is suggested

ACKNOWLEDGMENT

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THE FRACTURED MANDIBLE

A Method of Treatment for the Otolaryngologist[*][†][‡]

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INTRODUCTION

Today, because of fast cars and super highways, many disastrous accidents occur in rural areas, and the small community hospital becomes responsible for the total care of the critically injured patient. The attending otolaryngologist may be among the first called to treat the patient with injuries of the face, nose or even the mandible. Although the fractured mandible is usually reserved for the oral or maxillofacial surgeon, these specialized services may not be readily available, and under these circumstances the otolaryngologist must have a basic knowledge of the problem and a satisfactory method of treatment. Since most otolaryngologists have no experience with these cases during their residency training, they are unfamiliar with the principles of good treatment and often do not know where to begin.

Our objective has been to study this subject and from the abundant information in the literature and from our personal experience distill a method of treatment of the fractured mandible which can be applied by the otolaryngologist trained in head and neck surgery. I believe that the otolaryngologist, using easily available equipment, working with the local dentist, can satisfactorily treat the majority of fractured mandibles.

SURGICAL ANATOMY

The surgical anatomy of the mandible can be divided into

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the mandible proper, with its nerves, blood vessels and muscles, and the teeth

The Mandible Proper

The mandible consists of a condyle, neck, coronoid process, ramus, angle, body and symphysis[1] The bony structure varies in the different areas, and this is clinically significant The condyle or head is broad, and it is separated from the mandibular fossa of the temporal bone by the cartilaginous meniscus and is surrounded by a fibrous capsule Fractures within the capsule involving the head of the condyle are rare[2] The neck is slender and when under stress will fracture preventing injury to the middle cranial fossa[3] The external pterygoid muscle tendon inserts into the anterior margin of the capsule and partially into the neck The coronoid process develops from the pull of the temporal muscle similar to the development of the mastoid bone from the pull of the sternocleidomastoid muscle, and it is seldom fractured Two plates of compact bone, separated by a core of cancellous bone, make up the ramus It is covered on its lateral surface with the masseter muscle and medially by the internal pterygoid Because of this double muscular covering, the ramus when fractured seldom becomes displaced As the ramus broadens into the thicker body there is a line of structural weakness along which fractures occur[3] The body or horizontal ramus which lies anterior to the angle is thicker than the descending ramus The two halves of the mandible are united in the midline, but a blow to the chin may separate this symphysis The tooth bearing or alveolar portion is weaker than the main section of the body, and it may be fractured alone, without a fracture through the body

There are two openings in the bone the mandibular foramen, which is the entrance for the inferior alveolar nerve and artery, and the mental foramen which is the exit of the mental nerve, a cutaneous terminal branch of the inferior alveolar nerve Except for an anesthesia of the lower lip, injuries to the mental nerve have little clinical significance Within the ramus and body of the mandible lies the mandibular canal, through which passes the inferior alveolar nerve and artery The nerve innervates the teeth and the mandible The artery nourishes the bone and the teeth, and with the periosteal collateral circulation the mandible is quite vascular Fragments of bone which have an attached periosteum should be preserved since the circulation may be adequate to maintain viability, however, if the blood supply to a tooth is disturbed, the pulp will die, and it is usually best that such teeth be removed before infection develops

The muscles have a double significance Their normal action provides extensive motility to the bone and when the mandible is fractured, they are important in the displacement of the fragments The internal and external pterygoid muscles produce a side to side or chewing motion of the jaw When a fracture occurs anterior to their insertion, the posterior fragment will be pulled inward and upward The masseter and temporal muscles participate in the upper displacement of the posterior fragment These four muscles of mastication or the posterior group, interact to produce a general pull which is upward and toward the midline The anterior group, or the accessory muscles of mastication, consisting of the genio-hyoid, mylohyoid, digastric and genioglossus muscle, have a combined tendency to pull the anterior fragment downward and inward

Because of the action of the muscles on the fragment, the fracture line can be classified as favorable or unfavorable[3] In the unfavorable fracture, the fragments are kept separated by the muscular tension When favorable, the fragments are aided in apposition by the muscles The fracture line can be in a horizontal or vertical plane The favorable type of fracture is partially immobilized by the muscle action and requires less surgical fixation to produce rigidity

The Teeth

The teeth are important surgically for several reasons Their presence in the jaw, and this is particularly true of the third molar and the cuspid in the adult, furnish a potential line of weakness In the child, the presence of unerupted permanent teeth weakens the structure of the mandible[3] but this appears to be compensated by the increased elasticity of the bone

The upper and lower dental arches articulate with the opposing facets of the teeth The dental occlusion must be considered in the proper reduction of the fracture The normal articulation of the teeth serves as a guide in the alignment of the fragments, and fixation is made restoring the patient's normal bite If this is neglected in the process of reduction, the patient will become a dental cripple requiring considerable rehabilitation in the future The surgeon should familiarize himself with the normal variations in dental occlusion and be aware of the fact that a patient's normal bite may be an abnormal one, such as a crossbite

The teeth also serve as pegs for interdental and archbar wiring There is a difference in the solidity of the deciduous and permanent teeth, and the primary teeth do not withstand stresses as well as the permanent teeth and may loosen or shift in position This presents a special problem of handling a fracture in a child When possible the permanent teeth should be used for immobilization and these erupt at different ages

The edentulous patient, because of the lack of the precise articulating surfaces of the teeth, presents a different situation Although some degree of variation in alignment may be compensated for by new dentures, the normal distance between the upper and lower dental arches should be maintained during the reduction of the fracture This will prevent strain upon the temporo-mandibular joint and avoid future pain in this area

CLASSIFICATION OF FRACTURES

The general classification of fractures can be applied The fracture may be *simple*, a linear crack with no external communication and usually seen between the condylar head and angle It may be *compound* either through the tooth bearing area or with a facial laceration The *comminuted* fracture is the result of a violent force and frequently is also compound

The *complicated* fracture usually involves the inferior dental nerve and vessels The impacted fracture is rare, and there may not be any noticeable clinical movement The surgical neck of the condyle may present a *greenstick* fracture When a fracture occurs with minimal trauma one must be suspicious of a generalized or local skeletal disease causing this *pathological* fracture

A special description of the mandible fracture can be based on the anatomical site of the injury

1 Condyle
2 Neck
3 Coronoid process
4 Ramus
5 Angle
6 Body
7 Symphysis
8 Alveolar process

The fracture may be

1 Single unilateral
2 Double unilateral
3 Bilateral
4 Multiple

Kazanjian has devised a useful classification based on the presence of teeth in the fragments [4]

1 Class I—teeth are present on either side of the fracture

2 Class II—teeth are present on one side of the fracture

3 Class III—no teeth are present on either side of the fracture

Classifying the fracture will crystallize the salient features of the injury and will help in determining the proper therapy

THE HEALING PROCESS

When the mandible is fractured, bleeding occurs between the ends of the fragments The blood irrigates the wound and then clots, protecting the injured ends Within 48 hours new tissue starts forming from the mitosis of the fibroblasts and endothelium, and these cells invade the blood clot By the third day the fibroblasts have produced collagen fibers, and capillary sprouts can be identified microscopically[5] As part of the reparative process, phagocytes enter the injured area soon after trauma occurs and begin to remove the cellular debris, blood pigments and fibrin The proliferation of the fibroblasts and capillaries continues, and a highly cellular granulation tissue results After the first week, this tissue starts to immobilize the bony fragments, however, they can still be moved with pressure and any necessary correction in alignment can be made[6] The fibroblasts develop a basophilic ground substance which surrounds the cells and as this callus is formed, the union between the fragments becomes firmer and greater forces are needed for realignment The healing process continues with the absorption of bone by the osteoclasts and replacement by osteoblasts Trabeculae are formed, shaped by muscle stresses until the fully differentiated bone is produced The total process may take a year, but the mandible is rigid enough for function in about six weeks

When comminution of bone is present, the fragments are united by this process and are incorporated in the final structure A bone graft which replaces destroyed tissue, is invaded by the fibroblasts and in time becomes an integral part

The lacerated epithelium, either the skin or oral mucous membrane, heals by proliferation from the epithelial edge Injured muscle is replaced with fibrous tissue and if enough muscle remains it continues to function normally The damaged blood vessels are replaced by collateral circulation The severed nerves undergo degeneration of the distal end but if carefully anastomosed, there may be some return of function although it may take over a year

Rapid and uncomplicated healing of the fracture depends on several factors *1* the fragment ends must be in close apposition The further the fragments are separated the longer it will take to fill in the defect and a large defect may not be united by bone but by fibrous tissue, producing an unstable union *2* The fragments must be immobilized as completely as possible. A minute amount of motion at the fracture line may stimulate proliferation of bone[2] but any additional motion will either cause shearing of the granulation tissue and injury to the circulation across the healing zone or it will produce unwanted tensions at the healing line which prevent the laying down of bone. A third factor is the presence of infection in the wound. It may reach the fracture site through several channels. Fractures through the tooth bearing area are frequently compounded into the mouth, and the fracture will be influenced by the amount of infection present there. Bacteria can be brought in through a skin laceration or by infected teeth which are adjacent to the fracture. The blood stream and lymphatics have a lesser role in infecting the area. Suppuration produces necrosis of the soft tissue and bone, preventing proliferation and bridging of new bone. In this environment, the healing process is slowed down and the dead bone or sequestrum must be removed before healing can continue. Foreign bodies also will act as sources for reinfection and will delay healing until removed.

When there has been a large loss of tissue, either soft or bony, infection is usually present. This type of injury heals by secondary intention. Granulations develop in the base of the wound and gradually will fill in the defect. Healing may be complicated by an osteomyelitis of the bone and the final union in this type of injury frequently is fibrous and unstable functionally. The solution is the elimination of infection with the removal of the sequestrum, and when this has been accomplished, the fibrous union must be replaced by a bone graft for stability.

The patient's nutrition is important for healing. Vitamin C is essential in the production of collagen and vitamin D is needed for the completion of calcification. Bone cells which are actively proliferating require generous supplies of oxygen, organic calcium salts, amino acids and proteins. Parathyroid hormone, thyroxine and estrogens are also needed.

PRELIMINARY EXAMINATION AND EMERGENCY CARE

The mandible is fractured under many circumstances. A child may fall while skating or riding a bicycle, adults will get into arguments or fall down stairs and today, one of the most common causes is the automobile accident

Since there are numerous reasons for the injury, the problem is presented to the doctor in several ways The patient may arrive, with a minimum of pain, with no lacerations or bleeding, complaining only of difficulty in opening and closing the mouth Or they may be brought into the emergency room in severe distress The severely injured patient must be examined promptly and given emergency care The preliminary examination should survey the situation and determine what life saving immediate care is needed The most urgent problems are respiratory obstruction or severe hemorrhage Both may be present and perhaps complicated by unconsciousness

Airway obstruction can occur for a variety of reasons and must be corrected immediately When the patient is unconscious and supine, the tongue falls backward If we add the additional displacement of soft tissues that occurs with the fractured mandible, the airway is closed Salivary secretions, vomited food or blood clots accumulated in the pharynx will cause further obstruction A broken denture may be impacted there An examination to determine the cause of the obstruction must be done The tongue is grasped with a hemostat or sponge holder and pulled forward The pharynx is inspected, and any foreign bodies which are present are removed Secretions, vomitus and blood are aspirated If the patient can be safely turned, and put in the prone position or on the side, this will help in maintaining an open airway The jaw and tongue will fall forward and open the hypopharynx When the patient cannot be moved, an oral airway or nasopharyngeal tube must be inserted If these measures do not relieve the obstruction and it continues, the larynx must be examined Food or a piece of denture may be lodged between the vocal cords and must be removed Occasionally, the patient, in addition to a fractured jaw, may also have a crushing injury to the larynx The edema or a hematoma of the tissues may be causing an obstruction and must be recognized The airway may be established by inserting an endotracheal tube, a bronchoscope or by doing a tracheotomy[8] If there is no evidence of laryngeal obstruction as the cause of respiratory distress, the chest must be examined A pneumothorax, produced by a broken rib puncturing the lung must be recognized and relieved quickly A 50 cubic centimeter syringe with a No 16 needle can be used to withdraw sufficient air from the pleural space to allow expansion of the lung For more lasting decompression, underwater drainage must be used, until the lung continues to remain expanded The conscious patient with respiratory obstruction is apprehensive and uncooperative from hypercapnia[9] Oxygen by face mask or a nasal catheter must be given to relieve the distress No time can be lost in treating patients with respiratory difficulties since a few minutes of cerebral anoxia will produce irreversible brain damage[10] When the patient's airway becomes progressively obstructed from edema, a tracheotomy should be done The tracheotomy facilitates the removal of any aspirated material, and by passing a catheter into the trachea the patient can be given a high concentration of oxygen The tracheotomy is valuable in handling the patient with a pulmonary injury or the aged patient with diminished pulmonary reserve[11, 12] Doing the procedure early enough may prevent future complications such as atelectasis and hypostatic pneumonia[13]

Severe bleeding must be treated quickly or it will exsanguinate the patient If conscious, they will try to remove the blood from the pharynx and mouth by coughing and expectorating If unconscious, the blood will accumulate in the pharynx and enter the larynx Profuse bleeding is frequently associated with the compound fracture, the tongue and the floor of the mouth If the soft tissues are intact, the bleeding may stop as the hematoma enlarges, otherwise, the bleeding vessels must be clamped and ligated The abraided areas require local pressure to stop the oozing Occasionally, a hematoma may form at the base of the tongue or in the vallecula and produce respiratory obstruction These are incised and drained when detected The patient's blood loss is estimated by the hematocrit, and until it is replaced intra-

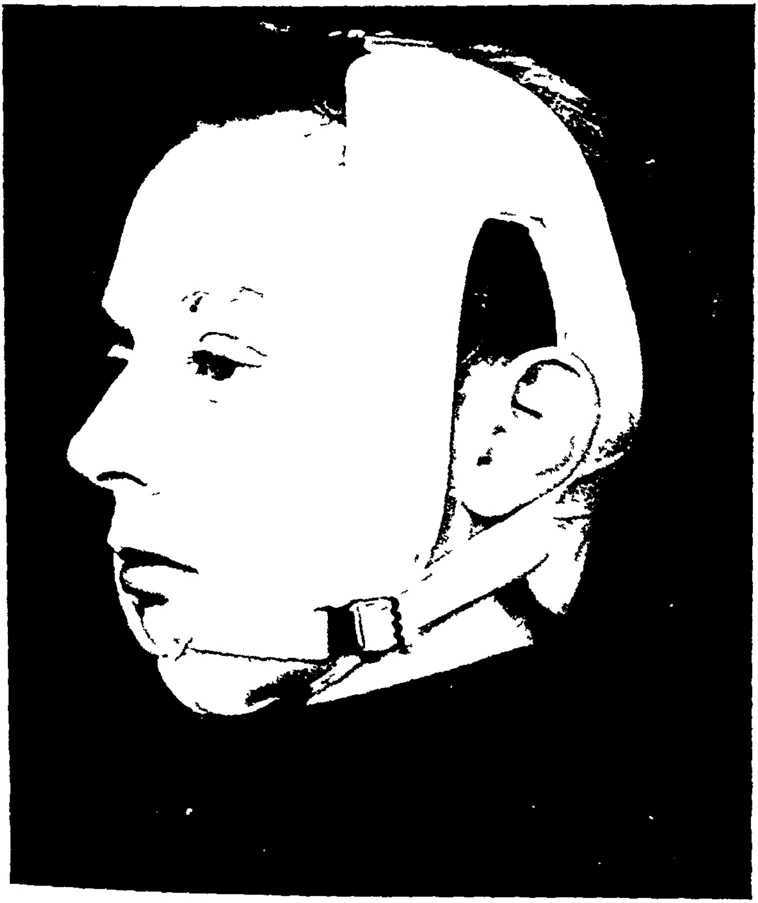
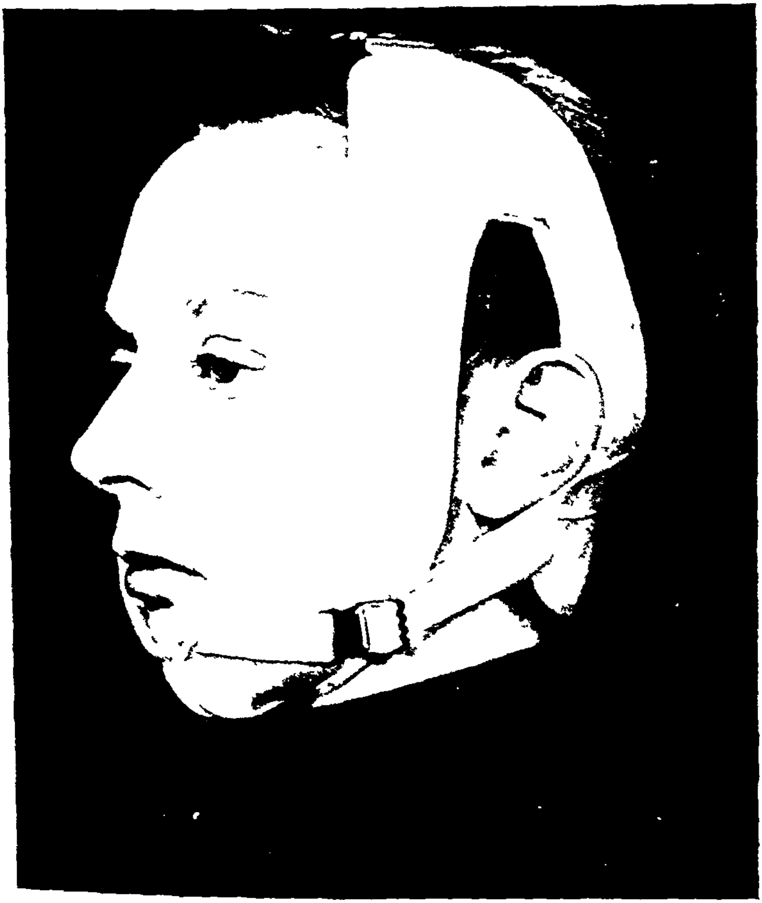


Fig 1 The fixation halter is created by modifying the cervical traction halter It can be made in three sizes for an accurate fit

venous saline or plasma should be given[14] When in shock the vasoconstrictor drugs neo-synephrine hydrochloride and Levophed Bitartrate are given by intravenous drip In the aged patient with arteriosclerosis a sudden blood loss may be fatal, and therapy must be started quickly

Pain is relieved by the injection of demerol or morphine, however, when there is respiratory obstruction or a brain injury, drugs which depress respiration such as morphine or barbiturates should not be used airway may be established by inserting an endotracheal tube, a bronchoscope or by doing a tracheotomy[8] If there is no evidence of laryngeal obstruction as the cause of respiratory distress, the chest must be examined A pneumothorax, produced by a broken rib puncturing the lung must be recognized and relieved quickly A 50 cubic centimeter syringe with a No 16 needle can be used to withdraw sufficient air from the pleural space to allow expansion of the lung For more lasting decompression, underwater drainage must be used, until the lung continues to remain expanded The conscious patient with respiratory obstruction is apprehensive and uncooperative from hypercapnia[9] Oxygen by face mask or a nasal catheter must be given to relieve the distress No time can be lost in treating patients with respiratory difficulties since a few minutes of cerebral anoxia will produce irreversible brain damage[10] When the patient's airway becomes progressively obstructed from edema, a tracheotomy should be done The tracheotomy facilitates the removal of any aspirated material, and by passing a catheter into the trachea the patient can be given a high concentration of oxygen The tracheotomy is valuable in handling the patient with a pulmonary injury or the aged patient with diminished pulmonary reserve[11 12] Doing the procedure early enough may prevent future complications such as atelectasis and hypostatic pneumonia[13]

Severe bleeding must be treated quickly or it will exsanguinate the patient If conscious, they will try to remove the blood from the pharynx and mouth by coughing and expectorating If unconscious, the blood will accumulate in the pharynx and enter the larynx Profuse bleeding is frequently associated with the compound fracture, the tongue and the floor of the mouth If the soft tissues are intact, the bleeding may stop as the hematoma enlarges, otherwise, the bleeding vessels must be clamped and ligated The abraided areas require local pressure to stop the oozing Occasionally, a hematoma may form at the base of the tongue or in the vallecula and produce respiratory obstruction These are incised and drained when detected The patient's blood loss is estimated by the hematocrit, and until it is replaced intra-



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Cases with compound fractures should receive 3,000 units of tetanus antitoxin or if they have been previously immunized, one cubic centimeter of tetanus toxoid

A survey should be made of the patient's associated injuries The severe accidents frequently produce multiple injuries and they may not be readily detected The abdomen as well as the skull, spine and extremities should be carefully screened

Until a careful evaluation can be made of the fracture, the mandible should be immobilized The Barton bandage may be used, however, the fixation halter can be applied (see Fig 1) It is a modification of the halter used for cervical spine traction, and is made by substituting a buckle in place of the rings Three sizes, small, medium and a large, can be prepared in advance and kept ready for use With gauze padding in the chin for comfort, it maintains immobilization until time for surgery In addition, the fixation halter is used in postoperative immobilization and can be substituted for the plaster chin bandage described by Schaefer[15] The halter has the advantage of being easily removed for dressings and can be changed when soiled

During the preliminary examination the injury to the mandible is put in its proper perspective, and the more serious injuries are given priority

THE EVALUATION

After the patient has received the necessary emergency care, the total problem must be evaluated This requires detailed consideration of several factors

The type and extent of injury to the mandible must be accurately established Considerable information can be obtained by careful inspection and palpation The examination starts with the region of the temporo-mandibular joint Pain elicited on pressing over the joint may indicate a fracture of the head or neck of the condyle Displacement of the condyle anteriorly indicates a dislocation, which may occur without a fracture The ramus is palpated for crepitus, since displacement in fractures of this structure are rare The contour of the angle of the mandible can be palpated between thumb and forefinger and any changes detected The body and symphysis are examined along the inferior border for any break in continuity When a suspicious area is located, it can be further examined by holding the fragmented ends between thumb and forefinger of both hands and gently trying to produce movement The patient is asked to open his mouth and any limitations or deviations of motion should be noted A displaced fracture involving the neck of one condyle will produce a deviation of the jaw to that side on opening the mouth The gingival and buccal mucosa are examined for lacerations Fractures of the body are frequently compounded into the mouth and may be evidenced by a laceration of the gingival mucosa The contour of the mandible is palpated within the mouth, on both the buccal and gingival surfaces, starting as high on the ramus as can be reached Occasionally a severe facial swelling will disguise a fracture which can be detected manually on an intra-oral examination

The dental arches are examined and loosened, broken or absent teeth should be noted and charted The graphic method of charting used by dentists should be learned The remaining teeth are palpated for rigidity to assess their value as anchoring pegs for wiring The presence or absence of dentures is noted, and if absent the patient is asked whether they are available since the dentures, even if broken, are of value in determining the dental occlusion The normal dental relationship must be determined, since this is the most valuable guide in the alignment of fragments, and all methods of reduction are based on the reconstruction of the patient's normal occlusion

The mandible should not be examined as an isolated organ, but must be considered in its relationship to the maxilla Both structures are involved in some of the methods of treatment and must be considered as one unit If the mandible alone is fractured, the maxilla will serve as a splinting arch for wiring, therefore, in the thorough evaluation of the

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The complete evaluation includes a radiographic study Although the physician may feel confident of his findings on physical examination and believes that no additional information can be obtained radiologically, radiographs should be made Some impacted fractures may not be evident clinically but will be detected on X-ray Radiographs make possible a progressive study of the case and the early detection of complications If litigation occurs a valuable record will be available

The study requires several views The postero-anterior (nose-forehead) position will visualize the condyle and the ramus The occipital-mental (nose-chin) position will bring into view the coronoid processes and is also valuable for the maxilla and malar bone The straight lateral view silhouettes the profile of the upper jaw, but because of the superimposition of the two sides of the mandible it may mask a fracture of the body This fault can be corrected by tilting the head and plate 30 degrees from the horizontal and photographing each side The condyle and the ramus will be exposed This same 30 degree lateral position with the head rotated 15 to 30 degrees upon the spinal column, will give an excellent view of the body of the mandible from the angle to the canine tooth depending on the degree of rotation Fractures of the alveolar ridge, particularly in the region of the incisors, may be clarified by using intra-oral films Further views may be suggested by the radiologist for a specific case[16] The radiographs should be considered an aid in diagnosis and should not be interpreted isolated from the patient

After the local pathology has been evaluated, consideration is given to other factors The type of anesthesia to be used is considered, and the less severe fractures can be handled with a local anesthetic This may be given in the form of an inferior alveolar nerve block, unilaterally or bilaterally[17] If the fracture is in the midline, supplementary infiltration of the mucous membrane in the area may be necessary If the wiring of the upper dental arch is required, the gingiva can be infiltrated with the anesthetic agent Novocain 2 per cent with 1 100,000 epinephrine or Xylocaine 2 per cent with 1 100,000 epinephrine is used The severe injuries will require a general anesthetic, and it must be determined whether the patient's medical condition will tolerate it Many of our mandibular fractures are associated with multiple injuries, and some of these may be of more serious consequence to the patient Surgery on the jaw should be deferred until the patient has recovered consciousness although a tracheotomy is valuable in removing accumulated pulmonary secretions This concept was first developed by Galloway[18] in treatment of bulbar poliomyelitis and subsequently recommended by others[19, 20] If the patient is in shock from blood loss, replacement therapy is given and the blood pressure restored and stabilized When more than one surgical procedure is required, interdepartmental planning will prevent the repetition of anesthesia The orthopedist frequently can reduce a fractured femur, while the mandible is being repaired Although the choice of the anesthetic agent is left to the anesthesiologist, the one most frequently used is intravenous pentothal and succinylcholine with endo-tracheal oxygen and nitrous oxide[21]

The technique of administration will vary When the patient has had a preliminary tracheotomy, the endo-tracheal tube can be passed through the opening The surgeon will have to decide on the need of a tracheotomy postoperatively, particularly when wiring has been used and the jaws are closed The use of the tracheotomy will prevent the postoperative asphyxiation from vomiting, which may occur before the wires or rubber bands are cut At other times the patient can be spared a tracheotomy The fixation of dental archbars can be done under general anesthesia, and later when the patient has reacted, the jaws can be fixed together with wires or elastics[22] When the surgeon is in doubt as to the need of the tracheotomy, it should be done[23] The risk of sudden asphyxiation is much greater than that of the surgery. The patient who has concomitant injuries of the chest and who will have difficulty postoperatively in handling secretions will benefit from a tracheotomy[11]

The patient's general medical condition will need to be evaluated with a careful history and physical examination The elderly patient may have a cardiac, pulmonary or metabolic disorder which will require special attention from the internist These conditions need to be controlled before surgery can be safely performed

The evaluation of the patient extends beyond the pathology of his mandible, and plans must be made for the entire hospitalization

METHODS OF TREATMENT

A review of the literature of the past 50 years reveals how much thought and ingenuity has been devoted to this subject It also becomes apparent that interest was stimulated by specific circumstances, the wars, the development of inert metals and the use of antibiotics The methods of therapy changed as new developments occurred, and this process will continue

Although many techniques of reduction and fixation have been devised, only two basic methods are involved first, the use of the teeth for fixation either with direct wiring, archbars or dental splints, second, the direct approach to the bone with wire, plates, pins or external appliances The experience of many surgeons has shown that no single technique is applicable in all situations because of the variety of injuries After reviewing the methods, it appears that 80 to 90 per cent of the mandible fractures can be satisfactorily treated by either interdental or archbar wiring[2,3 24,25 26 27 28,29] The remaining cases because of the absence of teeth or the location or displacement of the fracture must be handled by another method of fixation, and opinions differ as to the best approach In skillful hands many of the techniques can produce equally satisfactory results

In evaluating the techniques for the purpose of this paper, they had to fulfill special requirements

1 The equipment should be simple to use

2 A minimum of specialized laboratory facilities should be required

3 The methods must be safe and reliable

Each technique was evaluated and accepted or discarded

Interdental Wiring

This is one of the most useful methods of all Although this technique was developed early,[24 30 31] with modifications,[32,33]

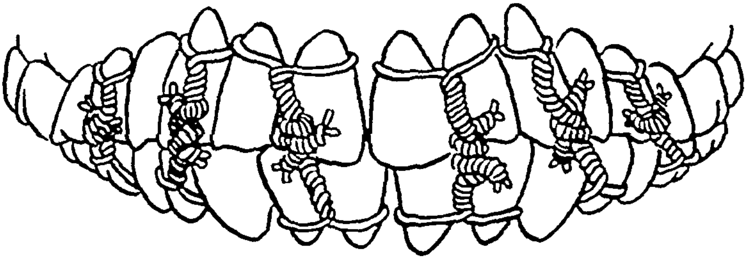

Fig 2 The superior and inferior interdental wires are tightened with the teeth in normal occlusion producing intermaxillary fixation

it can be applied successfully in many cases At first brass was used and later, stainless steel wire Stainless steel wire is flexible, strong and can tolerate twisting without breaking The finer grades such as No 30 may serve, but No 26, a heavier gauge, is more satisfactory The wire is placed around the neck of the tooth and twisted firmly The incisor teeth are best treated in pairs and the rest individually[4] The attached wires on the opposing teeth are drawn together bringing the dental arches into occlusion, and tightened By firmly tightening several wires on each side of the dental arch, the jaws can be rigidly immobilized (see Fig 2) Many variations of anchoring the wires to the teeth have been developed, and one of the most useful is the Ivy loop[24] This method overcomes the objection of having to wire the jaws together at the time of placing the wires around the teeth By using the Ivy loop technique, the teeth are first wired, and later the dental arches can be brought together with wire or elastics Kazanjian has recommended the use of the button technique, and it has been used successfully for many years[4] The twisted portion of wire is rolled into a small button which is then used as a peg for holding elastic bands

Interdental wiring is also used across the fracture line as a preliminary procedure to bring the fragments together, before the dental arches are wired together When the mandible and the maxilla are wired together, the result is intermaxillary fixation

Arch Bar Wiring

This technique is second to interdental wiring in usefulness The original arch bar of German silver has been varied to include loops (Jelenko splint) or buttons (Winter splint), or it may be made in a continuous row of malleable Monel metal (Erich splint) which can be cut to the necessary length The Jelenko and Winter splints are semi-rigid, but they can be bent to fit the dental arch and shortened with a cutting pliers The Erich splint is more flexible and can be molded to the shape of the dental arch by finger pressure Both types of splints serve a special purpose The rigid splint acts as an orthodontic appliance and gives greater stability to the fracture line The flexible splint is useful in producing intermaxillary fixation with rubber bands or wire and is easier to apply

The application of the splint consists of bending its shape to fit the dental arch and then wiring it to the available teeth Number 26 gauge stainless steel wire is used for this purpose Refinements in the fixation of the splint to the teeth have been developed[2 3,4 28 29]

The arch bar is most valuable when one dental arch has only a few teeth present It can be wired to the opposing teeth directly or to an opposing arch bar Frequently both arch bars are brought together with elastic bands (see Fig 3)

Dental Splints

There are three main varieties of dental splints which are in common use One of the earliest, and of orthodontic origin, is the Angle splint It consists of a gold band to which is soldered a gold bar The band is placed around a tooth and fastened securely with a screw and a bolt The bar is bent to the contour of the dental arch and then wired to the teeth[4] The bands are made in various sizes to fit the different teeth The Angle splint is useful when only one tooth is present on the posterior fragment, however, the same result can be accomplished by the proper application of a rigid arch bar

The dental cap splints are used by those who apply the dental technique for all mandibular fractures[2] Their use

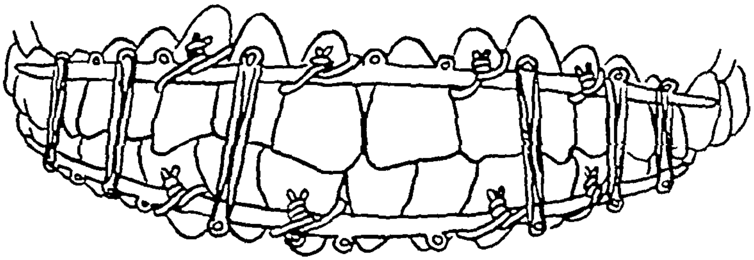

Fig 3 The opposing arch bars are brought together with elastics or wire

requires dental training and experience As soon as possible after the injury, impressions are taken of the patient's teeth and from these the normal dental arch is reconstructed, usually in two sections The splints are cast to fit the impressions and are then cemented to the teeth The sections of the splint are fastened together with screws An important prerequisite for the use of this technique is the availability of special laboratory facilities to construct the splint When these facilities are not immediately available, time will be lost Their application also requires the special knowledge of dental techniques, and this is not part of the otolaryngologist's training Besides this, there are two other objections to these splints the caps are cemented to the teeth, and this alters the patient's normal bite by the thickness of the cap Over the period of weeks which would be needed for the fracture to heal, the teeth may be adversely affected by the pressure In other instances, the cement may loosen, and the splint will come off It can be wired to the teeth for added fixation, but this is a duplication of effort Most of the problems that would be handled by a cap splint can also be handled by the arch bar or interdental wiring

The Gunning splint is used in association with alveolar or circumferential wiring[2,3,4,28,29] It is made of an acrylic plastic and is lined with gutta percha It can be prefabricated and kept ready in various sizes When used, the splint is placed over the fractured dental arch, and by tightening wires placed circumferentially around the body of the mandible or through a hole drilled in the alveolar bone, immobilization can be given to the fragments At least two wires are needed on either side of the fracture line This splint without intermaxillary fixation does not always furnish complete rigidity, and a fracture behind the last molar tooth, cannot be immobilized with this method unless other measures are used, such as the head cap with traction[34] The Gunning splint has its greatest usefulness in the small child, where injury to the unerupted teeth must be avoided[35,36,37,38] It is held in place with circumferential wiring until healing occurs

A modification of the circumferential wiring technique is used to immobilize a fractured edentulous maxilla to the mandible A hole is drilled through the alveolar process of the maxilla, and the stainless steel wire which is passed through this hole is firmly anchored to the teeth of the mandible The same process can be applied to the mandible if it is edentulous The wires are removed when the fracture is healed

Other variations in dental splints have been devised for specific purposes, but they do not alter the basic principle[39,40]

Interdental or arch bar wiring can handle most of the cases treated with dental splints, and the remainder can be handled by other less involved techniques

Internal Pin Fixation

The use of the transfixion pin or wire of the Kirschner type to hold the fragments together has been described by various authors.[41,42,43,44,45] Heavy Kirschner wires, bicycle spokes or round stainless steel pins have been recommended. In this technique, a wire is passed through the bony fragments with a drill. This produces fixation in one plane. The fragments, however, may still rotate or move laterally on the single wire. A second wire is usually applied and this eliminates the rotation, however, there may be some motion along the long axis of the pins, and by using interdental or arch bar wiring this can be prevented. This method has been used in a large variety of jaw fractures[43,45] including those of the condyle.[44] An objection to this method is that it must be combined with one of the other techniques for the most accurate alignment of the fragments, also, since one end of the wire is left protruding through the skin for the several weeks required to heal a fracture, there is a potential channel for infection to enter.

External Skeletal Fixation

The method of external skeletal fixation introduced by Anderson[46,47] was applied to the fractured mandible by Converse and Waknitz[48] in 1942. This method received considerable evaluation during the last war[49,50,51,52,53,54] and again more recently.[55,56] Many variations of the basic appliance have been devised,[57,58,59,60,61] but the fundamental principle is the application of pins or screws into the bone which are then connected externally to a rigid bar. When the screws are fastened securely to the bone and to the bar, the fragments are immobilized, however, the screws have a tendency to work loose from the bone, and the immobilization will fail. The opening through the skin is an invitation to infection, and patients have complained that they cannot properly clean their faces and that they keep hitting the appliance. An important surgical objection is the difficulty in producing and retaining an extremely accurate alignment of fragments.

Open Reduction with Interosseous Wiring

Since this method was first included in the treatment of jaw fracture its use has progressively increased.[27,62,63,64,65,66,67,68,69,70,71,72,73,74,75,76,77,78,79] A change has occurred from the time

Dunning[72] (1934) advised against the use of wire sutures in bone to the opinion of Gordon[65] (1947) and Plumpton and Crawford[73] (1955) who use this method in most of their problem cases

This technique requires an exposure of the fragments, drilling small holes through them and fastening the pieces together with wire The fragments can be aligned with great accuracy and by using several loops of wire, rigidity can be obtained Earlier, No 24 gauge brass wire was used,[63] then tantalum,[64,79] and now stainless steel wire[67,70 73] is preferred

The fracture can be approached from either of two directions Orally, the gingival membrane is incised, the periosteum elevated, and the ends of the fragments are exposed Holes are drilled through the alveolar portion, wires are passed through and tightened and the periosteum and gingival membranes sutured [28 64] The wires are permanently located beneath the gingival membrane, and they may prevent a comfortable fit with dentures

The more familiar and preferred approach is through an upper neck incision A 3 5 cm incision is made a finger's width below the lower edge of the mandible over the fractured area By separating the tissues horizontally with blunt dissection, injury to the mandibular branch of the facial nerve is avoided Its location, described by McCormack,[80] is constant The periosteum is exposed, incised and elevated from the fractured bone for a distance of one centimeter on either side of the fracture line The fragment is held firmly with a clamp and while protecting the soft tissues under the fragments with a flat metal retractor, holes are drilled It is important to remember the location of the inferior alveolar canal and to stay beneath this level The No 26 stainless steel wires are passed through the holes and tightened (see Fig 4) The periosteum is closed with 3/0 chromic catgut suture and the skin with 3/0 black silk without drainage Fractures in the condylar area can also be treated with open reduction and wiring either preauricularly[81 82 83 84] or endaurally [85,86]

When there is a fracture compounded, externally or orally, this technique can still be used If a comminuted fracture, those fragments with attached periosteum may still be viable and should be preserved A thorough cleansing of the soft tissues with removal of all foreign bodies and debris is imperative A soft rubber tissue drain is put into the area of the fracture, the periosteum is sutured and the skin closed The drain is left in until all drainage stops

The wires which are inserted are left in permanently When the tissues have been thoroughly cleaned and antibiotics used, removal of the wires because of infection is uncommon[64 65 73] The frequent reason for removal has been the electrolytic action and this will be discussed later Gibson[76] has devised a technique of removal of wire sutures which he recommends for compound fractures The surgeon, using open reduction methods, should not become complacent because of antibiotics The resurgence of resistant staphylococci emphasizes the continued need of obeying good surgical principles of debridement and cleaning

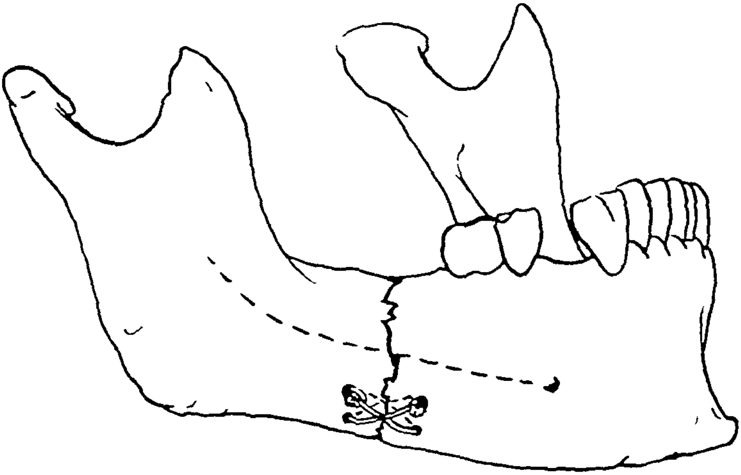

Fig 4 Interosseous fixation is produced by drilling holes on either side of the fracture and then immobilizing the fragments with No 26 gauge stainless steel wire The mandibular canal must be avoided when drilling the holes

Open Reduction with Plating

This procedure is a development of the previous one Instead of wire being used to approximate bone fragments, larger pieces of inert metal are fastened to the bone by wire or screws (see Fig 5) The use of metal plates for fractures of long bones had been previously described[6] and vitallium plates were used to replace bone removed in surgery of the mandible[87,88,89,90,91] Various authors[92,93,94,95,96,97] have described the treatment of the jaw fractures with open reduction and plating, but it must be recognized that this technique is not used for the routine reduction of the mandible fractures but only in special circumstances Severe fractures of the mandible with a loss of bony tissue, to anchor a bone graft, to make the mandible rigid in the combined fracture with the maxilla, these are indications for using the metal plate All of the precautions mentioned under interosseous wiring apply to the use of the metal plates, however, they have been safely used in compound fractures immediately after the injury,[88] but temporary drainage is used in all of these cases If there is no evidence of gross infection, the incision can be closed immediately after the insertion of the plate

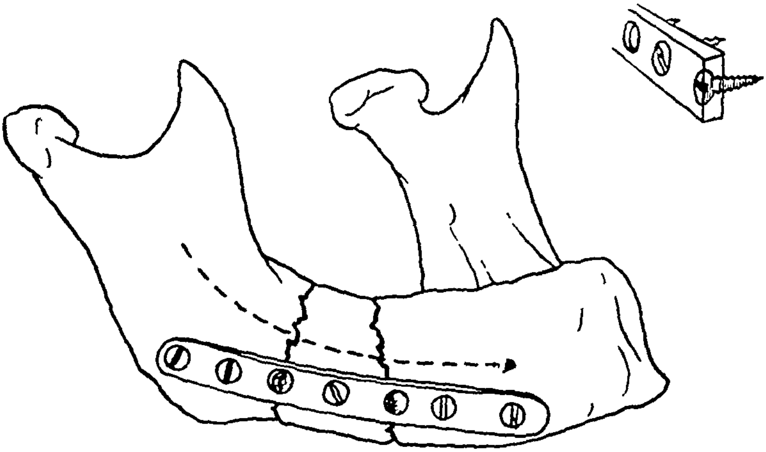

Fig 5 The vitallium plate is fastened to the fragments with screws producing a rigid mandible

The use of open reduction with wiring or plating makes possible the treatment of the majority of mandibular fractures which cannot be satisfactorily reduced and immobilized by interdental or arch bar wiring

The question arises as to whether there is any harm in the permanent retention of this metal

TISSUE REACTION TO METALS

With the acceptance of the open reduction technique, surgeons became interested in the cause of their failures Reductions which were free of infection, were unsuccessful because the plates would not hold The bone around the metal would break down, the screws would loosen, and the plate would slip out of position Venable,[98] interested in this problem, determined that electrolysis was the principal cause of failure of metal appliances in bone Dental research has also shown that metals in the presence of saliva act as a galvanic cell with erosion of the metal and that tissue changes result[3] Metals vary in the electrolytic effect, and in a few this effect is minimal,[99, 100, 101, 102] therefore, the maximum success in retaining metals in tissue occurs with the use of the most inert metals These are divided into two groups the alloys and the pure metals

In 1926 the present 18-8S Mo type of stainless steel alloy was developed In addition to iron, it contains chromium, nickel, silicon and molybdenum This alloy can be machined, cast into plates and drawn into wire It also has maximum corrosion resistance and a minimum of electrolytic action Recently it has been further improved producing the 19-9S Mo alloy[101] In small quantities, as is used in interosseous wiring, the effect on bone is minimal, but in the larger amounts of plating the electrolytic effect is increased

A more inert metal was needed This was developed by the Austenal Laboratories in 1929 and named "Vitallium" This alloy of cobalt, chromium, molybdenum, manganese, silicon and carbon is the most inert available and causes no appreciable electrolytic action[99] It has no stimulating or retarding effect on the healing of bone[102] and in several cases, infected wounds healed around the metal.[93] Venable, in a review of 1,227 cases, found that it can be left in the body indefinitely with no danger of late tissue damage.[99] Where it has been removed many years later, the metal and screws are bright and untarnished and the surrounding tissues are normal. Vitallium when introduced into the tissues immediately forms a chromium oxide surface film, and this prevents corrosion. Its remarkable inert qualities are slightly offset by its hardness, which requires that all plates and screws be cast, also, it cannot be drawn into wire and has only a small tolerance to bending.

Tantalum, a basic element, has a minimal electrolytic action. It is strong, tough, malleable, can be drawn into wire or machined, but it cannot be cast, however, it is available only in wire and skull plates.

The electrolytic effect is increased when two types of metals are used together. Chromium plated steel plates are unsuccessful, particularly when fastened with steel screws. Vitallium plates should be immobilized with vitallium screws and not stainless steel screws or wires. Although the silver alloy arch bar (Jelenko or Winter) is fastened to the teeth with stainless steel wire, a lesser electrolytic effect is obtained between a stainless steel arch bar and wire, and when applicable they are preferred.

The ideal metal for the purposes of the mandibular fracture would be as ductile and tough as stainless steel and as inert as vitallium or tantalum.[100] Research in metallurgy will someday achieve this goal.

REPAIR OF SOFT TISSUES

The healing process begins soon after the injury, and repairs should not be delayed. Unless the soft tissues are approximated, granulations will fill in the defect and produce distortions and disfigurement.

Lacerations of the face overlying a fractured mandible are frequently ragged and dirty. There may be a large amount of debris ground into the wound, and the severe compound fracture may resemble a war injury[illegible] The longer the contamination is permitted to remain, the greater the danger of infection[103] If the surgeon, after his evaluation, decides to proceed immediately, the cleansing of the injured area should be deferred until the patient is under general anesthesia When the patient is unconscious and the major surgery is delayed, the laceration is irrigated promptly with warm saline and all obvious debris is removed The tissues are approximated with a few loosely tied silk sutures, rubber drains are inserted and a dry dressing is applied The reduction of the fracture is then deferred until the patient's head injury has been evaluated and treated The conscious patient, whose reduction is postponed for some reason, should have the laceration repaired promptly Irrigation is used and if debridement is necessary, the area is infiltrated with 2 per cent novocain with 1 100,000 epinephrine It is preferable to do these procedures in the operating room, rather than in the emergency room

The repair of the laceration, whether done with the reduction or alone, begins with cleansing[104] The skin is washed with Phisohex or Septisol and foreign bodies which are imbedded are diligently picked out to prevent tatooing The laceration is separated and irrigated with copious quantities of warm normal saline Every hidden corner is inspected for a foreign body The debridement should be conservative, although devitalized tissue must be removed Ragged skin edges are made smooth and bleeding vessels are ligated[105] Frequently the fracture is exposed and can be inspected Devitalized bone fragments are removed, but if there is periosteum attached they are retained If, in the judgment of the surgeon, interosseous wiring or plating will be required, it can be done at this time The periosteum is important in healing, and effort should be made to preserve it In the closure, the tissues are approximated with chromic catgut suture One or more rubber tissue drains are inserted, and the skin closed with small interrupted dermalon sutures

Unless noted on examination, oral lacerations may be overlooked. Occasionally they are more extensive than those on the skin surface. When the buccal mucosa or periosteum is detached from the mandible, it is replaced as accurately as possible. The edges are approximated with chromic catgut, but space is left between the sutures to permit drainage.

Antibiotic or antiseptic solutions are no longer used to irrigate the wound because of their tendency to sensitize or destroy the tissue. Infection in the injured area is prevented by the use of systemic antibiotics.

FRACTURES OF THE MANDIBLE

Condyle

Condylar fractures are among the common fractures of the jaw. They are of two types the intracapsular, with injury to the head of the condyle, are rare while the extracapsular or those that occur to the neck are frequently seen.[2,3] Among the neck fractures, we have those due to minimal trauma and no displacement, the greenstick type, and the fracture with displacement. The insertion of the external pterygoid muscle on the head, produces a displacement of the fragment when the fracture at the neck is complete. A dislocation of the condyle can occur without a local fracture, and when present there frequently is a fracture in another part of the mandible.[77] Thoma[82] and MacLennan[106] have thoroughly described all the varieties of fractures that may occur.

There are two opinions for the treatment of the condyle fracture. First that all condylar fractures should have an anatomical reduction with immobilization.[81,82 83 84,85,86,107] Unless this is done, the occlusion will be altered, and eventually symptoms will arise in the injured area. The condyle is restored to its normal alignment by open reduction and wiring of the fractured ends,[81,82 83 84 85 86] the use of the Kirschner wire[41,45] or by plating.[23 97] The second opinion is treatment with immobilization alone for all types of fractures.[29 108 109] The mandible is immobilized by intermaxillary wiring or elastics, with the teeth in normal occlusion. A fibrous union occurs at the fracture site, which will function as a joint, even if the fragments are displaced and not in alignment Between these two views lies the contemporary opinion[23, 72, 110] The majority of fractures can be handled adequately by intermaxillary wiring, a few will require an open reduction or a condylectomy

A non-displaced, non-painful fracture of one or both condyles may need only a liquid diet and restriction of chewing for a month A painful fracture either without displacement or a greenstick variety can be made comfortable with intermaxillary wiring or elastic band fixation Sometimes the wearing of the fixation halter for one week will relieve the pain without interdental immobilization The halter immobilization is useful in the edentulous patient with one fractured condyle A unilateral displaced fracture of the neck, in a patient who has some teeth, is best treated by intermaxillary fixation Either interdental wiring or arch bar wiring with elastic immobilization is used The jaw is immobilized for four to six weeks in adults and three to four weeks in children The intracapsular fracture with comminution of the head, is treated in the same way When there are two fractures of the mandible with one at the neck, the condylar fracture is treated in the same manner, by immobilizing the mandible in normal dental occlusion after the other fracture has been reduced and immobilized The majority of condylar fractures will be satisfactorily treated by this conservative technique, however, special circumstances may require an open reduction and this should be approached with caution

In a bilateral condyle neck fracture with displacement, the mandible will be pulled posteriorly, and this will markedly distort the occlusion If this cannot be permanently corrected by intermaxillary fixation, open reduction and interosseous wiring of one condyle as described by Shapiro[72] will prevent this deformity It is used in conjunction with intermaxillary fixation When the condyle fragment is so displaced that the mouth cannot be opened or closed, even after manipulating the fragments, open reduction is needed When the fragment can be reduced, interosseous wiring is used, if the alignment does not hold then a condylectomy is done[110]

The condyle can be approached from three directions The preauricular incision described by Thoma,[81 82 84] Dingman,[83] and Richardson[23] directly exposes the condyle and the joint Its main disadvantage is the proximity of the branches of the facial nerve which are consistent [80] If the nerves are not severed by the surgery they may be injured with retraction Henny[111] has described the submandibular approach, through an incision under the angle of the mandible The masseter muscle is separated from the bone, and the condyle is exposed under the muscle Visibility for interosseous fixation or condylectomy is good The main branches of the facial nerve are avoided, since they lie superficially to the masseter muscle The location of the mandibular branch of the facial nerve must be appreciated and avoided in the incision Recently Rongetti[86] and Davidson[85] have described the anterior endaural approach to the condyle, and those otolaryngologists familiar with endaural surgery may prefer this technique Open reduction and interosseous wiring of the fracture should be combined with some form of mandible immobilization preferably interdental or arch bar wiring The use of plate fixation as described by Stuteville[97] is not needed, and the use of the Kirschner wire or external skeletal fixation has the disadvantages previously described

Coronoid Process

Fractures of the coronoid process are rare [112 113 114] Since the coronoid lies protected beneath the zygomatic arch, these fractures are seen combined with fractures of the arch and the malar bone The fractured coronoid has minor clinical significance since the function of the detached temporal muscle is compensated for by the masseter and pterygoids The most common symptom is pain rather than interference of function

It is treated with intermaxillary fixation by applying arch bars and rubber bands or in the edentulous patient a head bandage of the halter type When an open reduction is needed, and this would be a rare occasion, the submandibular approach described by Henny[111] would give a safe exposure to the area

Ramus

Although these fractures occur more frequently than those of the coronoid they are still uncommon[112,113,114] Laterally a large area of the ramus is protected with the masseter muscle, medially, the internal pterygoid The weakest point in the transmission of the blow to the jaw is at the neck of the condyle, and this fractures first When fractures of the ramus occur, they are not displaced because of the overlying muscles The treatment consists of using either dental fixation or a head bandage for immobilization Open reduction is rarely necessary, however, the approach described by Henny[111] is excellent

Angle

Where the ramus joins the body of the mandible, there is a line of structural weakness, and here fractures occur frequently[3] The presence or absence of posterior molar teeth, the direction of the line of fracture, the displacement from muscle pull have been responsible for a variety of methods of reduction and immobilization, however, the problem centers around one important point and that is the presence of displacement of the fragments

The fracture which is horizontally and vertically favorable with no displacement of fractured ends, is satisfactorily treated by immobilizing the jaw with intermaxillary fixation in occlusion If there is an associated fractured condyle on the opposite side, this will also be treated at the same time When the fracture is unfavorable, either horizontally or vertically, intermaxillary fixation alone is not adequate since the posterior fragment is displaced

It has been in treating this displaced fragment that many techniques have been devised for maintaining the reduction[2,3,4,28,29,43,48] Among them is the use of open reduction and interosseous wiring[68,72,73,74] This approach has simplified the problem and makes possible the treatment of all types of displacement with one method The technique is simple and safe A 3 cm incision is made a finger's width below the angle of the jaw adjacent to the fracture area The skin and platysma are separated and the periosteum exposed This is incised and elevated from the bone on either side of the fracture line Two small holes are drilled through the bone on both sides of the fracture, and 26 gauge stainless steel wire is then passed through the holes and tightened, bringing the fragments together with great accuracy (see Fig 4) The incision is closed without drainage, and the teeth are fixed together in occlusion to complete the immobilization In a bilateral angle fracture this procedure is done on both sides, and the wire is left in permanently

This method is especially applicable in the edentulous mandible If adequate immobilization cannot be obtained with the use of the wire alone, the head halter can be used to supplement the fixation

Body

Fractures of the body are of two types those with teeth on the posterior fragment, and those without The latter type can be considered as being the same as the angle fracture and is easily treated with open reduction and interosseous wiring The anterior fragment is then immobilized with intermaxillary fixation If there are teeth in the posterior fragment, interdental wiring across the fracture line combined with wiring to an arch bar, followed by immobilization by intermaxillary fixation is usually satisfactory A double fracture on one side, if there are teeth in the central fragment is treated in the same way

In children, a non-painful and non-displaced fracture can sometimes be treated by using the fixation halter

The edentulous mandible, with a single fracture can frequently be satisfactorily treated with interosseous wiring and immobilization with the fixation halter The patient should be kept on liquids for three to four weeks This type of reduction is extremely useful in the aged since early restoration of function occurs When there has been marked comminution of fragments, and these cannot be firmly realigned with wiring, plating with a vitalium plate across the fracture line gives firm support to the separation When combined with

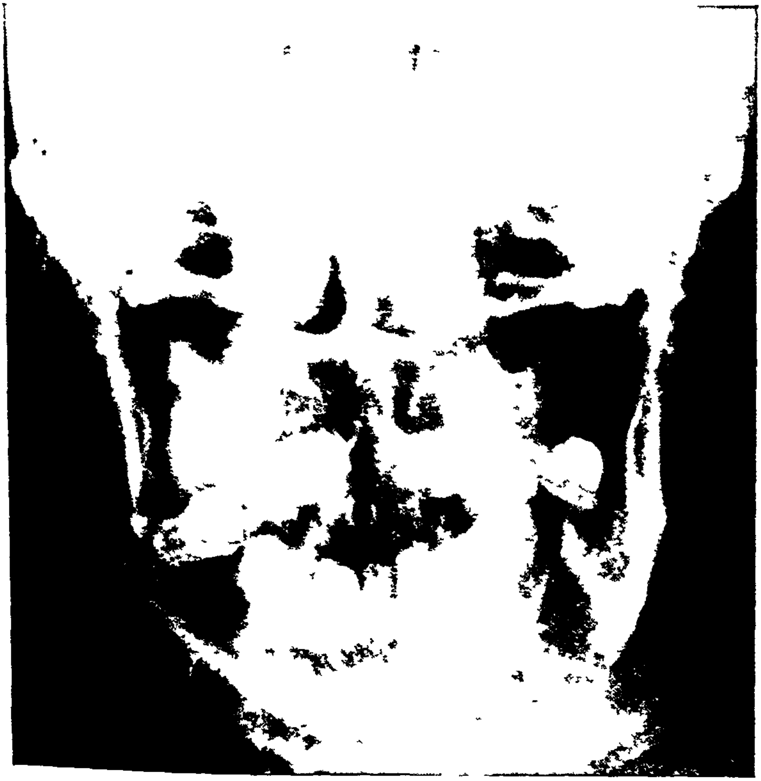

Fig 6 A displaced fracture of the right angle is combined with a comminuted fracture of the left body

the bandage early restoration of function occurs Bilateral fractures are also treated with wiring or plating as necessary and supplemented with intermaxillary wiring or halter (see Figs 6 and 7).

Symphysis

These fractures may be a true separation in the midline or they may be oblique When teeth are present on either side of the fracture, interdental wiring across the fracture line and then fixation to the maxilla will give good immobilization If only a few teeth are present then wiring to an arch bar with intermaxillary fixation will serve

In the edentulous patient the use of open reduction with

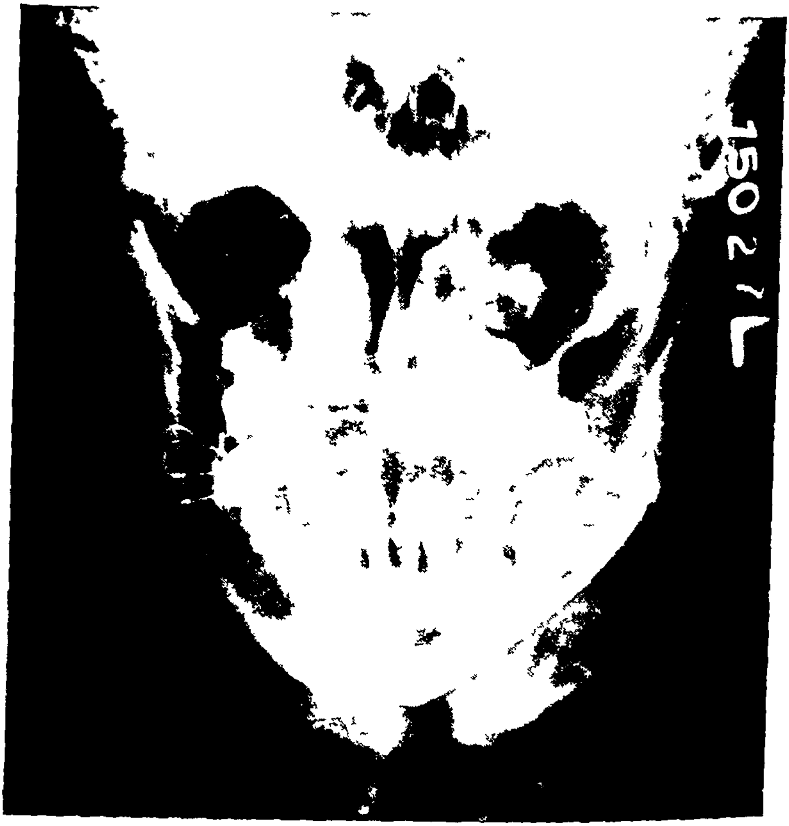

Fig 7 The angle fracture is reduced and immobilized with interosseous wiring and is combined with plating of the body fracture The mandible is then immobilized to the maxilla with intermaxillary wiring

interosseous wiring, through a submental incision is satisfactory With a severely comminuted fracture, plating may be needed

Alveolar Process

When there is a fracture through the alveolar process with displacement of teeth, an attempt should be made to save the teeth if possible The alveolar bone with the teeth attached, should be reduced to the normal occlusal position and retained by wiring to an arch bar If the teeth have been avulsed by the blow, the alveolar bone should be inspected for roots and these should be removed by the dentist When the alveolar bone is rough due to the fracture an alveolectomy is

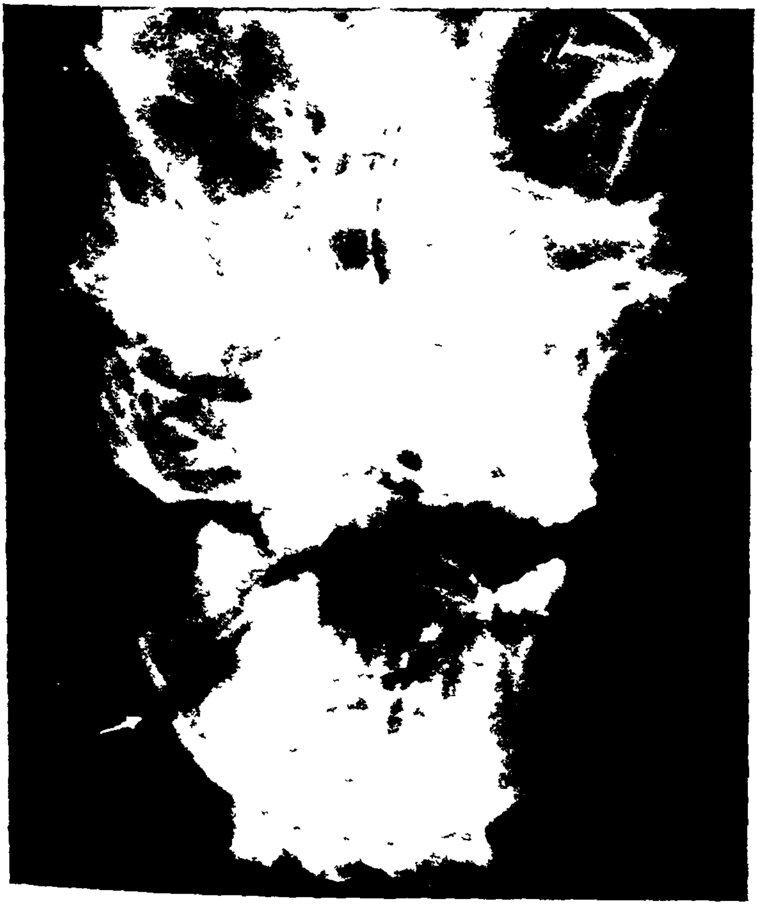

Fig 8 A fracture of the mandible is combined with a displaced fracture of the maxilla (LeFort I)

done This procedure is performed by the consulting dentist at the time of surgery and with experience can be done by the otolaryngologist

Fracture of the Mandible and Maxilla

This type of injury is a more complicated problem than has been previously discussed When the mandible alone is fractured, the maxilla can serve as a splint, to which the mandible is fixed[28] When both the maxilla and mandible are loose neither can serve as a splint unless one is made rigid The

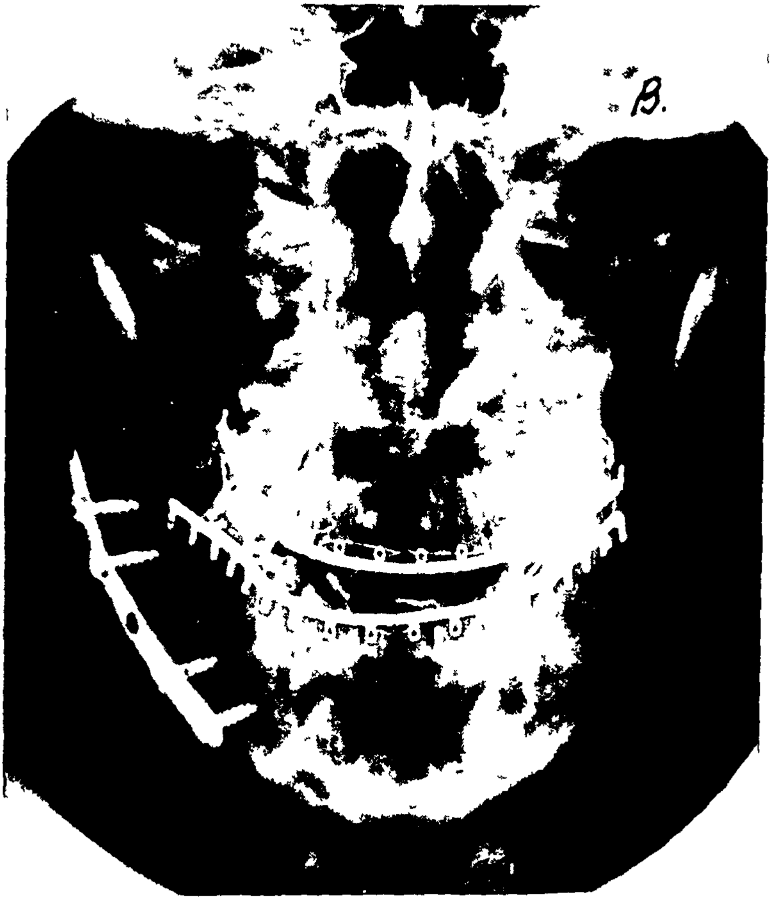

Fig 9 The mandible is plated for rigidity and by applying arch bars with intermaxillary wire fixation, the maxilla is immobilized

maxilla can be anchored by open reduction and wiring to the rim of the orbit or to the zygomatic arch as described by Adams and Walden[66] As an alternative method, the mandible is created into a rigid splint, and the maxilla is then fixed to the mandible[71] As long as good dental occlusion is maintained, the functional result will be satisfactory

When the maxillary injury consists of a depressed fracture of the anterior wall without motion or displacement of the dental arch, intermaxillary fixation with arch bars will be adequate to fix the mandible If the patient has an edentulous

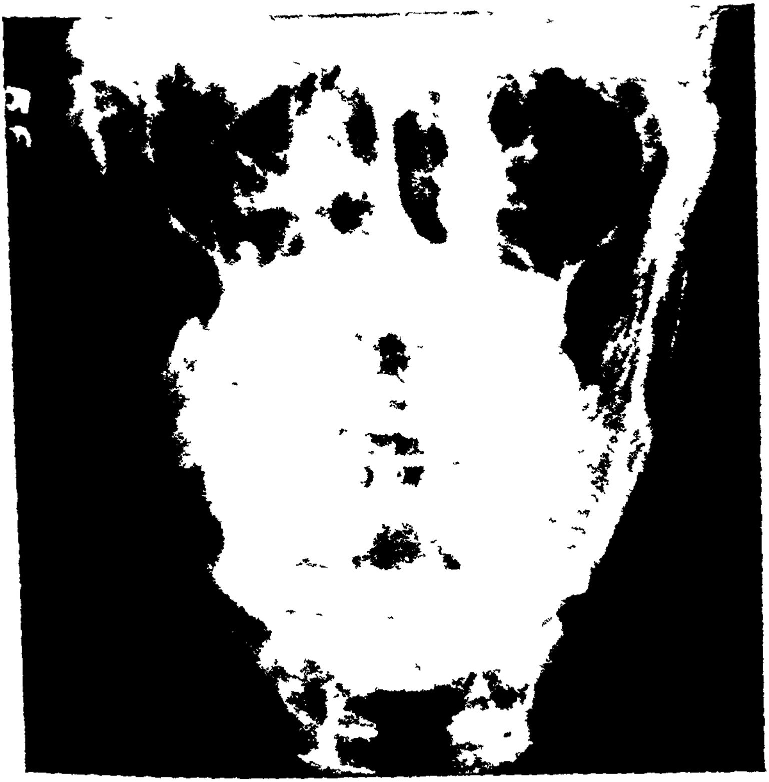

Fig 10 A bone graft taken from the iliac crest is used to fill in the defect and is held in position by the vitallium plate offering immediate rigidity to the mandible

maxilla the mandible is immobilized by using the pyriform crest as described by Thoma[115] and Fordyce[116] or the nasal spine[117] When the maxilla is displaced or freely movable it is then necessary to convert the mandible into a splint This is done with the use of open reduction and either interosseous wiring or plating (see Figs 8 and 9) If the mandible can be made rigid with the use of interosseous wiring rather than plating, this is preferred Although the plates may be left in permanently without harm, it is preferable to use as little metal as is necessary to produce rigidity

Fractures with a Loss of Bone Tissue.

Severe fractures of the mandible, particularly those which are compound, may involve a loss of bone tissue After the soft tissues have been cleaned, the bone fragments are inspected Those which have no attached periosteum will probably die and act as a sequestrum unless removed All those which are attached to the periosteum should be saved, since they may unite If a loss of bone tissue has occurred and there is a space between the fragment ends, the final union will be fibrous and unstable and in these circumstances the defect must be filled in with bone for a satisfactory union Several methods have been described using bone chips to fill in the defect [1,28] The mandible is first immobilized to the maxilla with intermaxillary fixation and then through a submandibular incision the fracture site is exposed and the defect filled in with bone chips

The vitallium plate is useful in this type of problem Fig 10 illustrates the use of the vitallium plate to hold in position a bone graft from the iliac crest until bony union has been achieved The plate is still in place and has not caused any difficulties requiring removal The plating was supplemented with intermaxillary fixation

The bone graft is not inserted while there is evidence of infection in the wound The laceration is allowed to heal while the fragments are retained in the occlusal position by interdental wiring When there is no evidence of suppuration, induration or pain, the area is reopened with a submandibular incision, and the bone graft is inserted The patient is maintained on antibiotics during the period of healing, before and after bone graft is done

SUMMARY OF TREATMENT

Fracture	Treatment
Condyle—	
1 A non displaced and non painful fracture	Liquid diet and restriction of chewing
2 A painful fracture with no displacement or a greenstick variety	Intermaxillary wiring or rubber band fixation or halter immobilization in the edentulous patient
3 Unilateral displaced fractures in patient with teeth	Intermaxillary fixation with wires or elastics for 3 4 weeks (child) or 4 6 weeks (adult)

Fracture	Treatment
4 Bilateral displaced fractures in patient with teeth	Intermaxillary fixation with wires or elastics If unsatisfactory occlusion results then an open reduction and interosseous wiring of one side is used in combination with intermaxillary fixation
5 Unilateral displaced fracture—edentulous patient	Replace dentures in mouth and immobilize with halter fixation
6 Bilateral displaced fracture—edentulous patient	Halter fixation If the jaw motion is restricted or marked displacement is present then use a unilateral open reduction and interosseous wiring combined with the halter
7 Condyle combined with a fracture in another site in a patient with teeth	Reduce and immobilize other fracture by interdental wiring or open reduction and then use intermaxillary wiring to immobilize condyle
8 Condyle combined with a fracture in another site—edentulous patient	Other fracture is immobilized with open reduction and interosseous wiring and the jaw is immobilized with the halter
Coronoid Process—	
1 Patient with teeth	Intermaxillary fixation by arch bars and elastics
2 Edentulous patient	Head halter for immobilization
Ramus—	
1 Patient with teeth	Intermaxillary fixation by arch bars and elastics
2 Edentulous patient	Head halter for immobilization
Angle—	
1 No displacement of fragments—patient with teeth	Intermaxillary fixation by wires or elastics
2 Displacement of fragments (horizontally or vertically unfavorable) in patient with teeth	Open reduction and interosseous wiring combined with intermaxillary fixation by wires or elastics
3 Displaced fragments in the edentulous patient	Open reduction and interosseous wiring or plating (if comminuted) combined with halter immobilization
4 Bilateral angle fractures	Open reduction and interosseous wiring combined with intermaxillary fixation by wires elastics or head halter if edentulous
Body—	
1 With teeth on the posterior fragment	Arch bar immobilization and intermaxillary fixation with wires or elastics
2 No teeth on the posterior fragment	Open reduction and interosseous wiring combined with intermaxillary fixation with wires or elastics
3 Edentulous mandible	Open reduction with interosseous wiring or plating combined with halter immobilization

Fracture	Treatment
4 Severe comminution of fragments with or without teeth	Open reduction with interosseous wiring or plating combined with intermaxillary fixation by wiring or elastics or if edentulous, halter immobilization
5 Double unilateral fracture with teeth in fragments	Arch bar wiring and intermaxillary immobilization
6 Double unilateral fracture — edentulous fragments	Open reduction with interosseous wiring or plating Immobilization with the head halter
Symphysis—	
1 Teeth present on both sides of the fracture line	Interdental wiring or arch bar wiring across the line of fracture combined with intermaxillary fixation with wires or elastics
2 Teeth present on only one side of fracture or if edentulous	Open reduction and interosseous wiring combined with intermaxillary elastic or halter fixation
Alveolar Process—	
1 Teeth present in fragment	Immobilize the teeth to an arch bar
2 Edentulous patient	If periosteum is attached retain the fragment, if not, then do an alveolectomy
Mandible and Maxilla—	
1 Teeth present in mandible and maxilla	Mandible is made rigid with arch bar wiring and then maxilla is immobilized to the mandible with intermaxillary wiring
2 Teeth present in maxilla edentulous mandible	Mandible is made rigid with open reduction and wiring or plating and maxilla is fixed to mandible by use of alveolar process circumferential wiring of the mandible
3 Teeth present in mandible with an edentulous maxilla	Mandible is made rigid with arch bar wiring and maxilla is fixed to mandible with alveolar process circumferential wiring
4 Edentulous mandible and maxilla	Mandible is made rigid with open reduction and plating or wiring and the maxilla is fixed to the mandible with alveolar process circumferential wiring with the dentures between the arches to maintain the proper spacial relationship
Fracture with a Loss of Bone Tissue—	
1 Fragments contain teeth	The dental arch relationship is retained by intermaxillary wiring of the fragments until a bone graft is inserted
2 Fragments are edentulous	The arch relationship is maintained by bridging the gap with a metal plate until the bone graft is retained in proper position by interosseous wiring or plating

FRACTURES IN CHILDREN

The incidence of the fractured mandible in children is low It is estimated that they occur in two to five per cent of all mandible fractures[35] and less than one per cent are in children under six years of age[36] Since by the twelfth year the mandible closely resembles that of the adult except for the third molar tooth,[35] we consider this age to be the upper limit of the childhood fracture

The child with the broken jaw presents a somewhat different problem from that of the adult, starting with the examination The fear and lack of cooperation usually prevents a thorough physical examination and X-rays assume a greater importance Although many fractures in the adult can be treated with local anesthesia it is almost always necessary to give the child a general anesthetic[37]

The mandible of the child presents special characteristics The bone is more elastic and softer, and this appears to predispose to a greenstick type of fracture, frequently of the neck of the condyle[36] The presence of tooth follicles in the bone produces a potential weakness and may account for the widespread fractures that occur The fracture lines in children may involve two to 14 teeth and a larger proportion of the bone than in the adult[35] The presence of a growth center in the condyle, which may be injured, can lead to a deformity in the development of the jaw,[36 118] and this has been shown experimentally by Sarnatt[119] Interference with the tooth follicles will prevent the normal development of the dentition

The treatment of the fracture is based on the principal of using a method which will give minimum interference with the tooth follicles Although some[118 120] believe that adult methods are applicable in most cases, many authors prefer special methods[15 31 35 36 37 121 122] When the permanent teeth are present, intermaxillary fixation with arch bar and elastics is preferred The arch bar is wired to the available secondary teeth as in the adult (see Fig 3) The deciduous teeth, because of their shape do not lend themselves to the dental wiring methods or to the use of cemented cap splints, and because of the presence of the secondary tooth follicles, some authors do not recommend the use of external skeletal fixation or open reduction with interosseous wiring [15,36,38,121] Others believe that open reduction can be done when needed, but the unerupted teeth should be retained and the interosseous wiring be placed in the cortical bone without interfering with the developing teeth [35,120] A fracture of the body behind the last molar tooth, the angle and the ramus can be safely wired by open reduction when necessary, as in the adult Most surgeons recommend that condyle, neck and head fractures in children be treated conservatively by intermaxillary dental fixation or by a head bandage The fixation halter described earlier serves adequately A displaced fracture will need the specialized dental technique in the construction of a Gunning type of splint or an acrylic splint The fractured segments are aligned under the splint, and the splint is then fixed to the mandible by circumferential wiring as described by Thoma[28] or Erich [29,34] The mandible should be immobilized by fixation to the superior dental arch or by immobilizing the jaw with the fixation halter for additional rigidity

FRACTURES IN THE AGED

Medical progress in extending the life span has increased the number of aged patients Kulowski[123] in his series of 661 automobile accidents reported that 11 per cent of his casualties were 60 years or older We can expect that this group will continue to increase

The patient who is 65 years old presents several interesting characteristics The mandible may be edentulous and as a result of the absorption of the alveolar bone, rather narrow When this is combined with the increased brittleness of the bone and the exposed position, it is surprising that it is not fractured more often The older patient frequently has another illness for which he is or should be receiving therapy A coronary sclerosis, hypertension or a pulmonary emphysema can significantly alter the prognosis of a fractured jaw

The evaluation of the fracture must consider two aspects the local problem, and the patient's general condition Occasionally it may be necessary to compromise a perfect surgical result for safety to the patient The surgery should be deferred until the patient has had a general medical evaluation and any necessary treatment The choice of an anesthetic must be carefully considered and when possible local is used The presence of excessive secretions in the airway of the aged patient may require a tracheotomy[12]

Kennedy[124] in discussing the management of injuries in the aged has advised that function be restored as soon as possible Older patients have a small tolerance for cumbersome external appliances and head caps Because of these various factors the treatment of mandible fractures in the aged is made as simple as possible When teeth are present intermaxillary wiring is used and if this cannot be easily or safely done, open reduction with interosseous wiring or plating is done This latter method gives a rapid restoration of function The postoperative course frequently requires special attention to the patient's nutritional and mental state A high vitamin, protein and caloric liquid diet is prescribed, and the agitated patient with cerebral arteriosclerosis is made comfortable with the use of tranquilizers Early mobilization will prevent complications and the patient should be permitted to sit in a chair as soon as possible

POSTOPERATIVE CARE

The nutrition of the patient is an important adjunct in the therapy A high protein, calorie and vitamin liquid diet is prescribed and it can be taken by a straw or from a spoon Where complete immobilization is not required, as in certain impacted fractures, soft solid foods such as cooked cereals, creamed soups and ice cream can be added In children and in the aged the dietary requirements need additional attention The child's weight should be checked periodically to be sure that growth needs are being met The old patients may require dietary regulation for diabetes, cholecystitis or other medical conditions

The use of antibiotics has made the open reduction technique safe and uncomplicated Fractures in the areas with teeth are usually compounded into the mouth The same occurs with lacerations of the face which lie over the fracture Both types of injuries can heal with primary intention when the antibiotics are used When face lacerations are present the area is drained with a soft rubber drain for 48 to 72 hours to remove the accumulated blood and serum Procaine penicillin 300,000 units and 5 grams of streptomycin are given twice a day for one week or until the soft tissue lacerations are healed If there is suppuration, the drain is left in and the patient kept on antibiotics until it stops

Mouth care is important in the treatment Warm saline rinses after and between meals help to keep the area clean, especially when lacerations are present The use of hydrogen peroxide or perborate solutions may produce undue tissue damage The antibiotic solutions may sensitize the patient and are not recommended The use of elastics for fixation enables a periodic examination of the mouth and cleansing of the teeth when indicated

The postoperative care does not end with the patient's discharge from the hospital They should be seen for intermittent follow-ups for several months which makes possible an early recognition of any delayed complications If the patient has suffered dental injury he should be suitably referred The surgeon's judgment will develop from observation and comparison of his results over a period of years

SPECIAL EQUIPMENT

The special equipment needed to apply the techniques outlined is easily available

1 Stainless steel wire (alloy 18 8S Mo) in No 26 and No 28 This is available through surgical suppliers

2 Wire cutter and pointed pliers The types obtained from electrical supply houses are satisfactory

3 Jelenko or Winters arch bars are available as a package unit from dental supply houses or directly Jelenko Arch Bar J F Jelenko & Co Inc, New York 19, New York, Winters Arch Bar Baker & Co, Inc 113 Astor Street, Newark, New Jersey

Small elastic bands for immobilization are included or they can be easily made from rubber tubing

4 Erich Monel arch bar is available in a roll from N I R O 280 West State Street, Westport, Conn

5 Vitallium plates and screws are obtained from Austenal, Inc 224 East 39th Street, New York 16 New York

Vitallium Bone Plate No 6913 Mandibular 3" straight

Screws, No 6912—7/64"

Venable Screwdriver—7/64"

Vitallium Finger Plates, No 6315—¾′ and 1" with four holes

Screws, No 6349

Screwdriver, No 6884-3

6 The drill for the interosseous wiring and plating can be either a Zimmer or Stryker drill and is usually available in hospitals A drill bit size No 55 or 052′ is used

SUMMARY

The subject of the fractured mandible has been discussed as a problem to the otolaryngologist, the pertinent literature has been reviewed and various techniques described, evaluated and applied

A method of treatment has been presented which can be satisfactorily applied by the modern otolaryngolgist trained in surgery of the head and neck

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THE INFLUENCE OF DEAFNESS ON THE CREATIVE INSTINCT*

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It has been said of the creative artist that his work, be it prose or poetry, painting or sculpture, music or drama, is the product of an inner compulsion whose only standard is of his own making, and that the desire to please others in no way influences or even stimulates his compositions, which, no doubt, accounts for the fact that many creative artists never live to see their work appreciated.

Bach in his own lifetime was scarcely known outside his own country, while Van Gogh sold only one picture, and that for but a few francs. A genius is not concerned with his impact upon society. He can only obey the creative impulse which sooner or later leads to immortality. It is interesting, therefore, to speculate upon the extent to which the outpourings of a man of genius can be influenced by his mental and his physical state.

The Pathetique Symphony of Tchaikowsky, first performed but three weeks before his death, expresses, particularly in its final movement, the tortured and perhaps over-dramatized despair of the neurotic, while the macabre and often malevolent heroes of Dostoevsky are reflections of his own character and circumstances.

My purpose here is to show how deafness may be said to have influenced the creative instinct of artists in three quite different fields: a writer, a painter, and a composer. In each I believe the cause of the deafness was different; in two it must surely have left its effect on their work, but it may well be disputed whether it had any effect on the output of

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the third, but as I shall hope to show you there may well be a good reason for this

Man, as we know, is a social animal He likes to talk, and he is occasionally prepared to listen, so that if he is deprived of his hearing he feels left out, and indeed he gradually gets left out of things He becomes withdrawn and even suspicious, and one of my patients once told me that "deafness takes all the colour out of things", which leads me to my first patient—the Spanish painter, Goya, who became suddenly and totally deaf in 1793 at the age of 47 as the result of a sudden and severe illness, which for a time affected his vision as well This tragedy overtook him not long after he had been appointed Court Painter to Charles IV of Spain, at a time when he was in great demand as a portrait painter, though his reputation as a painter was founded on his compositions of Spanish life, all in the most delicate, though at the same time striking, tints of lemon, red, yellow and pale green These colourful scenes were for the most part full of action, life and gaiety, and in this period of his activity one cannot but be impressed by his genius for depicting the pleasant side of Spanish town and country life

Soon after he was overcome by deafness, Goya's artistic output changed, both in colour and in character Much of the colour left his pictures, in fact the walls of his house, the "Quinta del Sordo" were covered with what, because of their sombre tints, are known as the "Black Paintings" Most of these have been preserved and can now be seen in the Prado Museum in Madrid, where they provide such a striking contrast to his colourful and gay earlier paintings, many of which are also to be seen in the Prado Not only have they lost their colour, but they have also lost their gaiety Instead, they dwell on the macabre and even at times the disgusting side of life His series of etchings on the Horrors of War and the Caprichos are curiously unpleasant Though they may be true of the times and certainly of wartime, they are entirely different from what he produced in his earlier and healthier days His social and political conscience seems to have developed to the extent of influencing his artistic output only after he became deaf

At one time I wondered whether the unusual disorder that ushered in his deafness could have affected his appreciation of colour, but this surely was not the case, because he could on occasion conjure up the old colours, as can be seen in his last painting, "The Woman of Bordeaux," painted 25 years after his illness when he was approaching 80 years of age, or the famous though heartrending "Night of May 3rd", or again the amazing and revealing Group of Charles IV and his family

What then was this illness that for a time affected his vision and left him totally deaf? Of course, syphilis has been blamed, and there can be no doubt that, as the statistician would put it, he was often "at risk" Though happily and fruitfully married he had many love affairs and one of his models, who also was his mistress, by her beauty attracted the attention of the Prime Minister Godoy, who took her over from Goya in return for forgiving a political exile Godoy himself was the lover of the Queen whose two youngest, and certainly as portrayed by Goya, brightest children, are said to have been fathered by Godoy

Goya was also for a time lover of the Duchess of Alba, who, it has been said, was the model for his two paintings, the clothed and the unclothed "Maja"

No matter how hard he tried to acquire or avoid a specific infection, it is unlikely that this would suddenly attack vision and hearing and also, no doubt, balance at the age of 47, allowing full vision to return after a few months

To me it sounds much more like a curious and little known affection of the pigmented areas in the head and neck in which temporary patches of white hair and white skin are accompanied by temporary affection of the uveal tract, and alas permanent loss of hearing and often of vestibular function Known as the Vogt-Koyanagi syndrome, I have seen three full-blown cases and two other partial ones In my cases the deafness was almost complete, and so far as I know, permanently leaving the patients in a sorry plight I can well imagine what a profound and shocking effect this might have on a sensitive and gregarious artist To be cut off from

his fellow creatures would encourage his creative instinct to turn towards the sorrows and even the horrors of life instead of its pleasures, and in those times of a corrupt court, invasion and domination by Napoleon Bonaparte, there were plenty of horrors to depict

Let us now go back almost a century to study the case of another creative artist, this time the writer Jonathan Swift, who died at the age of 78 in the year 1745, just a year before Goya was born Poet, wit, satirist, political pamphleteer, friend of kings and queens, Dean of St Patrick's Cathedral, Dublin, but never, to his mortification, to become a bishop He also was famous as a platonic lover whose correspondence with Vanessa and with Stella has entertained posterity more than it satisfied his languishing correspondents, who both died spinsters within a few years of one another and more than 25 years before the object of their unrequited or at any rate unsatisfied affection

His introduction to political and court life came early as secretary to Sir William Temple, who played an important part in cementing the friendship between Holland and England and who had helped to bring William of Orange as joint occupant of the English throne with Mary

Swift leaned towards the Tory side of politics which was under the guidance of Edward Harley, after whom Harley Street was named, and who later became the Earl of Oxford The Tories were opposed to Marlborough and his wars, and Swift was their most potent and most strident pamphleteer

His two most famous works, "The Tale of a Tub" and "Gulliver's Travels" were brilliant but biting satires on the times in which he lived, and later in his prose and his verse, he became at times venomous and even disgusting One cannot help comparing some of his later works with some of Goya's drawings, both of them dwelling, almost harping, on the worst side of human nature and the times

Towards the end of his sad life, Swift's days were darkened by deep depression which was regarded as madness, though there is no real evidence to show that he had lost more than two of his senses, those of hearing and balance.

For this unfortunate man suffered from bilateral Meniere's Disease and was subject from early manhood to bouts of giddiness and deafness which pursued him throughout his life. Let us hear what the patient himself has to say about it all:

"I got my giddiness in 1690 (at the age of 23) by eating 100 golden pippins at a time at Richmond. Four years later at a place 20 miles further on in Surrey I got my deafness, and these two friends have visited me one or other every year since, and being old acquaintances have often sought fit to come together."

It is, however, probable that he was hard-of-hearing in one ear before the giddiness started because he wrote to his friend, Sheridan, as follows:

"In England, before I was 20, I got a cold which gave me a deafness that I could never clear myself of. Although it came but seldom and lasted but a few days, yet my left ear has never been well since, but when the deafness comes on, I can hear with neither ear, except it be a woman with a treble or a man with a counter tenor."

Clearly he was describing bilateral Meniere's Disease with severe deafness in the left ear and fluctuating low tone deafness in the right ear.

A sympathetic friend, Mr. Forde, gives us some idea of the view held then by patients of doctors who were trying to cope with a disorder which defied their efforts:

"These sort of disorders provoke the Physicians everywhere, and they are merciless dogs in purging and vomiting to no purpose when they know not what to do."

Poor Swift continues to protest against the purgings and bleedings ordered by his doctor, maintaining that his trouble lay neither in his belly nor in his blood, but in his head, and in this, of course, he was quite right.

After he died in 1745, the skull and contents were examined, and it was stated by a Dr Delany that the brain was loaded with water This, in view of our present knowledge of Menière's Disease, was not very far from the truth

Here then we have a great writer, plagued all his life with giddiness and deafness from recurring bouts of Menière's Disease a depressing prospect for anyone, but for a sensitive creative and ambitious man, who better than any of his contemporaries could see and portray the follies of his time, and who was frustrated in his ambition to become at least a bishop, it is no wonder that his genius was soured Whether his attitude to women was influenced by his disorder is a matter of conjecture There can be no doubt that he enjoyed their company and that they were his for the asking, but he never asked I have known many sufferers from this and from other forms of deafness who never married, and though I have never asked them outright, I have the impression that they did not want to saddle a partner with having to share such an affliction

Here then are two great creative artists, each gregarious and each shut off from his fellow creatures by a severe form of perceptive deafness In each the artistic output was tinged with pessimism and even despair, and I suggest that it was their deafness that played a large part in their gloomier outpourings

Now we come to our third patient, the composer Beethoven, who was born in 1770 and who, despite his deafness must surely be the greatest composer the world has known

Our first knowledge of his affliction comes from the Heiligenstadt Testament which was in fact his Will and was only opened and read after his death

During the summer of 1802, Beethoven was in the small village of Heiligenstadt not very far from Vienna, engaged in composing his Second Symphony He was 32 years of age at the time and his Will and testament which he wrote during this summer included the following remarks about his deafness

"*But only think that during the last six years I have been in* *a wretched condition, rendered more by unintelligent Physicians, decreased from year to year with hopes of improvement, and then finally forced to the prospect of lasting infirmity"*

Here then is the patient's own account of his deafness which he had noticed for at least six years—at the age of 26—and no doubt it had been coming on slowly before that

Is there any wonder that Beethoven was a difficult man, one who could claim to be the very Prince of Sound and yet he could not hear himself?

All the deaf are sensitive about their affliction, but how much more sensitive a musician must be to the sly laugh or grin that his defect would be bound to evoke from the less charitable of his acquaintances, yet despite this great and progressive handicap he composed 100 more works after writing this Heiligenstadt Testament

Towards the end of his life he was extremely deaf He conducted the first performance of his Ninth "The Choral" Symphony, and as was the custom in those days, he was seated in a revolving chair As the symphony finished he waited, still facing the orchestra, for the applause There was dead silence He looked at the orchestra, shaking his head, the tears pouring down his cheeks Then they turned him around to face the audience who were cheering and clapping in a frenzy of applause which he could not hear

After his death in 1827 his skull and temporal bones were examined but nothing definite was found, though as usual that etiological whipping boy of those days, syphilis, was evoked

Like Swift, and possibly for the same reason, Beethoven was unmarried, though there is ample evidence to show that he was attracted to women I think that he, too, preferred not to share his affliction with someone else, though I also think that both he and Swift would have been happier men if they had

As regards the cause of Beethoven's deafness, I cannot help agreeing with my friend Dr Tremble and many others, that the most likely cause of a progressive deafness starting in early adult life is otosclerosis

Another point in favour of this is that as he became more deaf, he used to listen to the piano as he was playing by holding one end of a short stick between his teeth, the other end being applied to the piano top Surely this is evidence of good bone conduction such as one would expect with otosclerosis He also used hearing aids, though of course in those days they were non-electrical, but the mere fact that he even tried hearing aids suggests that he had a conductive deafness which, in the absence of any history or evidence of suppuration, was almost certainly due to otosclerosis

Why then, it may be asked, did not Beethoven's deafness have any noticeable effect on his artistic output? Why was his music, if the argument is to be logical, not gloomy or even macabre?

So far as his everyday life is concerned it seems that he behaved in the conventional way of the hard-of-hearing He was difficult, quarrelsome, suspicious and withdrawn but his artistic output does not appear in any way to have been influenced by his deafness His eighth symphony is as gay and light as his second, and it is difficult, nay impossible, to believe that any of his compositions could have been improved upon

I think that one reason that his deafness did not affect him more seriously is that his was a conductive deafness He did not have to suffer the distortion and discord of the fluctuating deafness of Meniere's Disease when the very sound of music can be intolerable, moreover, he was not like Goya, suddenly cut off from all outside sounds His was a gradually increasing deafness which did not become profound until the pattern of musical expression was well established in his mind After that, what was important was what he created in his mind and what he could write down, not what he heard For him the supreme tragedy was that he could not hear what he had composed Though he knew it was right, he did not have what must be the consummation of musical experience, the ability to hear his own work played back to him by a full orchestra

No doubt many other examples of the influence of mental *and physical* disorders can be adduced, but equally there can *be no doubt that in all* three patients I have mentioned, deafness was a great burden, and that in at least two of them it *affected their artistic* output, *whilst in the third it robbed him* of the fullest appreciation of his own genius

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VIIeme INTERNATIONAL CONGRESS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY

The Seventh International Congress of Otology, Rhinology and Laryngology will be held in Paris, July 23-29, 1961, at the New Faculte of Medicine, Rue des Saints-Peres The program includes *1* The Recent Acquisitions, Pathology and Therapeutics of Otosclerosis by Profs Sourdille, Sercer and Kimpotic, Weber, Ardouin, Larroude, Ferreri and Shambaugh *2* Indications and Five-Year Results in Surgery and Radium in the Treatment of Cancers of the Larynx and Hypopharynx—Profs Pietrantoni and Agazzi Alonzo, Casadesus, C L Jackson, Leicher, Leroux-Robert and Ormerod *3* A Report on Allergy and Infections of the Nose and Bronchus—*Profs* Van Dishoeck, *Clerici, Hlavacek, Voorhorst,* Mayer, Laskiewicz and Terracol

Tours and entertainment for guests and their ladies have been arranged President Dr Maurice Aubry, Secretary General Henry Guillon For further details write Dr Guillon, 6 Avenue McMahon, Paris, France

AURAL CHOLESTEATOMA EXPERIMENTAL OBSERVATIONS*†‡

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Several theories have been proposed to explain the appearance in the middle ear of stratified squamous epithelium and subsequent formation of cholesteatoma Clinical and histopathological evidences indicate, however, that in most cases, the squamous epithelium originates in the epidermis of tympanic membrane and/or skin of external canal The extensive literature on this topic can be found in the publications of Nager,[1] McKenzie,[2] Lindsay,[3] Teed,[4] Day,[5] Ojala and Saxén,[6] Ruedi[7-9] and Friedman[10]

Although the immigration theory has been accepted by most investigators, not all opinions agree regarding the mechanism underlying the ingrowth of epidermis It is accepted that the epidermis may immigrate through a perforation in Shrapnell's membrane or a marginal perforation in pars tensa, however, since Nager[1] stated that "no cholesteatoma can arise with a central perforation," the possibility that the epidermis of the pars tensa can grow inwards through a central perforation has been frequently overlooked

The controversy has been mainly about the conditions predisposing to and causing attic cholesteatoma and also whether a perforation is necessary for ingrowth of epidermis The aural cholesteatoma following necrotizing otitis media had been disregarded since the widespread use of chemotherapy and antibiotics has reduced its incidence considerably

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‡From the Section of Otolaryngology of the University of Chicago

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Clinical observations indicate that attic cholesteatoma may develop in two ways[11, 12]: in one, there may be no history of recurrent otitis media, and Shrapnell's membrane presents a small perforation but little or no retraction. Histologic findings in experimental animals[11] suggest that this cholesteatoma may originate from epidermis which immigrated through the perforation. The other type observed clinically is preceded by repeated episodes of acute middle ear inflammation leading to formation of a retracted pocket. The development of this pocket seems to be due to cicatricial retraction of inflammatory tissue contained in the epitympanum. If a subsequent acute middle ear inflammation produces swelling, exudation, and exfoliation of epidermis with retention of debris in the pocket, then cholesteatoma may develop either with or without a perforation of the retracted Shrapnell's membrane. Further development of the cholesteatoma may sometimes be averted by prompt removal of the debris. As formerly[12] pointed out, the previous explanation applies as well to those cases of cholesteatoma which develop by way of the posterosuperior quadrant of the pars tensa. In these cases the inflammatory process destroys the membrana propria (or dense fibrous tissue layer). That part of the membrane is then devoid of its supporting middle layer. As adhesions form and later contract the unsupported part of the membrane is retracted beneath the annulus, thus the conditions for development of cholesteatoma during a subsequent acute episode are established as in the case of retracted Shrapnell's membrane.

The clinical assumption that the epidermis may ingrow in the absence of a perforation is supported by histologic studies in both man and experimental animals.[6, 7, 8, 9] According to Ojala and Saxén,[6] Shrapnell's membrane is provided with a loose, connective tissue beneath the epidermis as a continuation from the skin of the external canal. This layer, known as the corium, prevents invasion of basal cells into Prussak's space and the attic, however, inflammatory processes may produce clefts or openings in it through which the stimulated basal cells may grow as finger-like projections. Depending upon the inflammatory conditions in Prussak's space and attic, the invading basal cells extend over the surface of the damaged lining or underneath the mucosa or through granulation tissue. Cornification of the center of the finger-like projections of basal cells eventually may take place, thereby developing a cholesteatoma without perforation of Shrapnell's membrane. Ruedi[8,9] emphasized the potentialities of basal cells to proliferate and that under suitable conditions these cells may form epithelial cones which can invade the epitympanal space in the absence of perforation.

The study to be reported here will describe alterations in the middle ear of experimental animals which may have a bearing on the pathogenesis of aural cholesteatoma. The investigation illustrates the histologic picture of the epidermis invading the middle ear through a marginal perforation, through a central perforation and from an apparently intact eardrum. In addition, the questions of how the migratory epidermis progresses and the clinical implications of the findings are discussed. The observations to be reported here have no bearing on the pathogenesis of true cholesteatoma which sometimes involves the petrous pyramid, nor do they contribute evidence on the question of metaplasia of middle ear cuboidal epithelium to stratified squamous epithelium.

METHOD

The investigation was carried out in 30 guinea pigs. The experimental procedure has been described in detail elsewhere.[13] Essentially, the method consisted of injecting 0.10-0.12 cc. of a 1.0 per cent aqueous solution of quinine hydrochloride or 0.8 per cent alcohol solution of quinine sulfate, slowly into the antrum. None of the guinea pigs showed signs of otitis media when the drug was administered.

Twenty-four animals were killed and fixed by intravital perfusion with Heidenhain-Susa solution at convenient intervals ranging from one to 76 days after the injection. The temporal bones were removed in one block and processed according to standard histologic technique for hematoxylin-eosin stain. The serial sections were studied under regular light microscopy.

Six guinea pigs were injected with quinine as described above for observation of immediate changes in the tympanic membrane For this purpose, the external canal was cut transversely near the eardrum and observed directly under a high power dissecting microscope (40X)

RESULTS

A Acute Stage

Direct observation of the eardrum revealed that it became vasoconstricted soon after the quinine solution touched the inner surface In one hour or less, bullae appeared in the pars *flaccida* and/or pars tensa The bullae enlarged slowly covering as much as half of the eardrum The content was evacuated in a few hours into the external canal The histologic examination showed that the bulla was formed between epidermis and membrana propria and that in the latter microscopic perforations could sometimes be observed

On the third day, the epidermis of the eardrum showed striking hyperplasia and exfoliation Perforation or necrosis of the tympanic membrane sometimes occurred The lining of the tympanic cavity presented thrombosis, hemorrhages and edema in the substratum and the epithelium was necrotic in some areas

Commentary The formation of bullae, vascular changes and hyperplasia of the epidermis of the eardrum represented a reaction to the irritating agent Other substances applied over the inner[8,9] or outer[14,15] surface of the eardrum have produced a similar hyperplastic reaction

The importance of these findings is that by means of an irritating agent the epidermis of the tympanic membrane and probably also the adjacent skin of the canal were strongly stimulated, thereby setting up a favorable condition for proliferation

B Chronic Stage

It is probable that the quinine was absorbed or eliminated in a few days, but the resulting inflammatory reactions or its

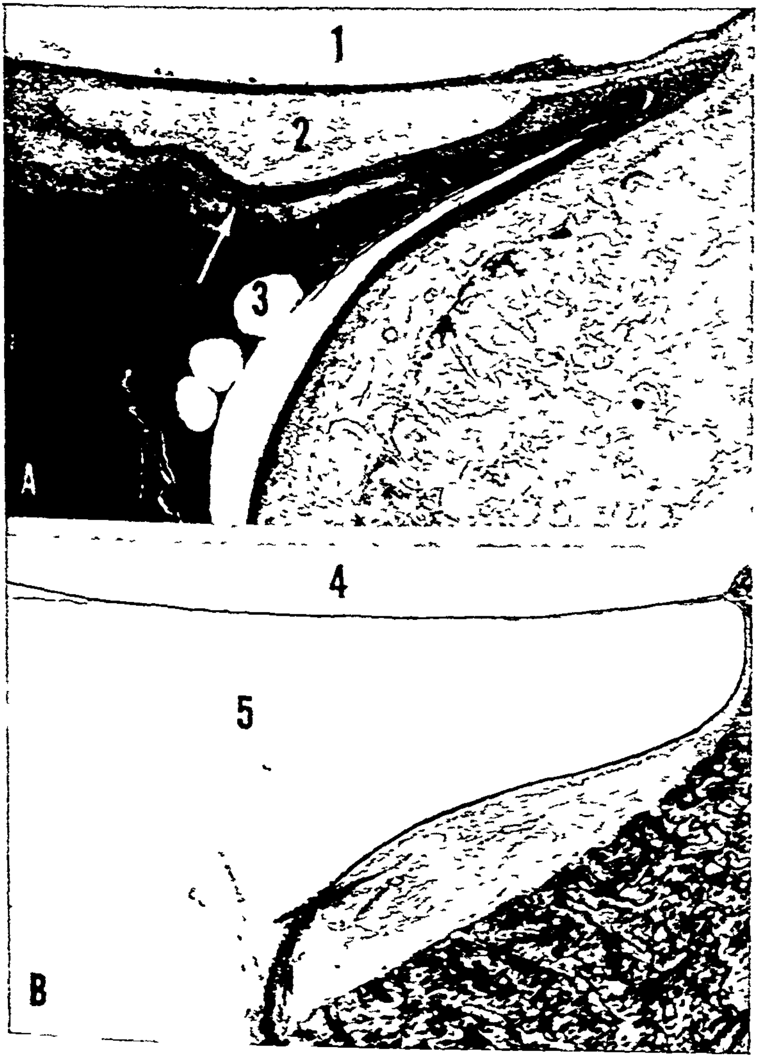


Fig 1 Guinea pig D-50 Animal sacrificed seven days after injecting quinine hydrochloride A.—Hyperplasia of epidermis of the eardrum 1 tympanic cavity 2 bulla formed by detachment of epidermis 3 external canal filled with pus and debris Arrow points to hyperplastic epidermis (H-E X44) B—Control ear 4 tympanic cavity 5 external canal At the upper left corner the epidermis was detached from membrana propria This is an artifact Compare its thickness with that of epidermis shown in upper photomicrograph (H-E X44)

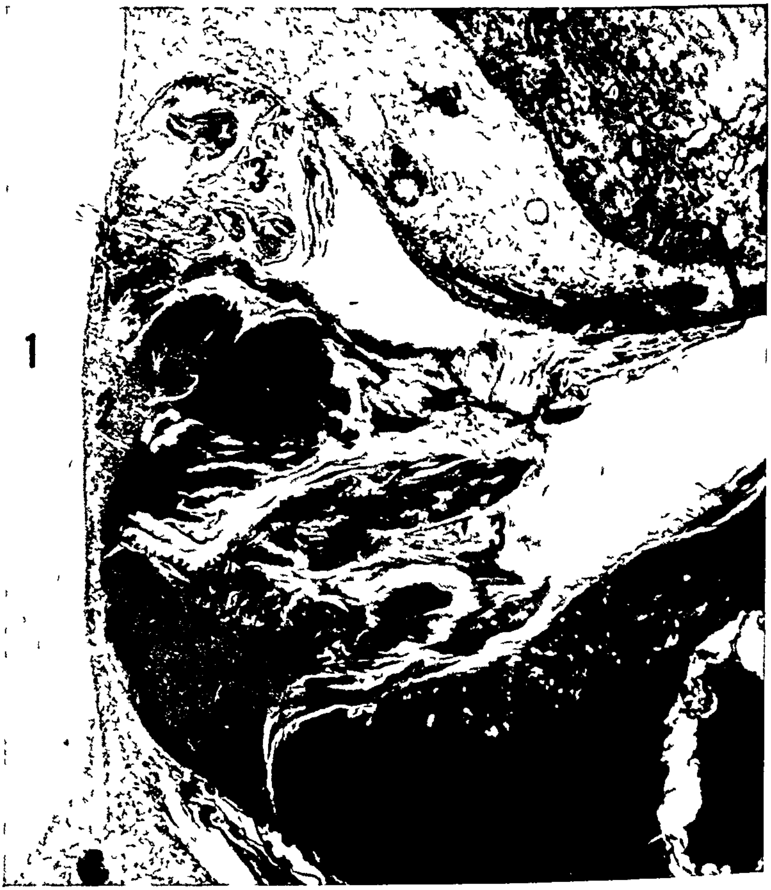


Fig 2 Guinea pig D-50 This photomicrograph illustrates hyperplasia of the epidermis of eardrum and cholesteatoma-like formation in the external auditory canal The animal was injected with quinine hydrochloride and sacrificed seven days later 1 tympanic cavity 2 epidermis of tympanic membrane 3 debris and desquamated cell layers accumulated in the external auditory canal (H-E X55)

sequelae could be traced in all animals In many cases the histologic features appeared as a mixture of inflammatory reaction to the irritant and infection, however, the latter was not determined bacteriologically

The histologic examination of these specimens showed a variety of lesions, demonstrating that the epidermis of both the skin of the canal and the tympanic membrane may immigrate into the middle ear

I Inflammatory Lesions

In about seven days, the hyperplasia of the epidermis reached a maximum thickness of 10 to 15 times that of control ears (see Fig 1)

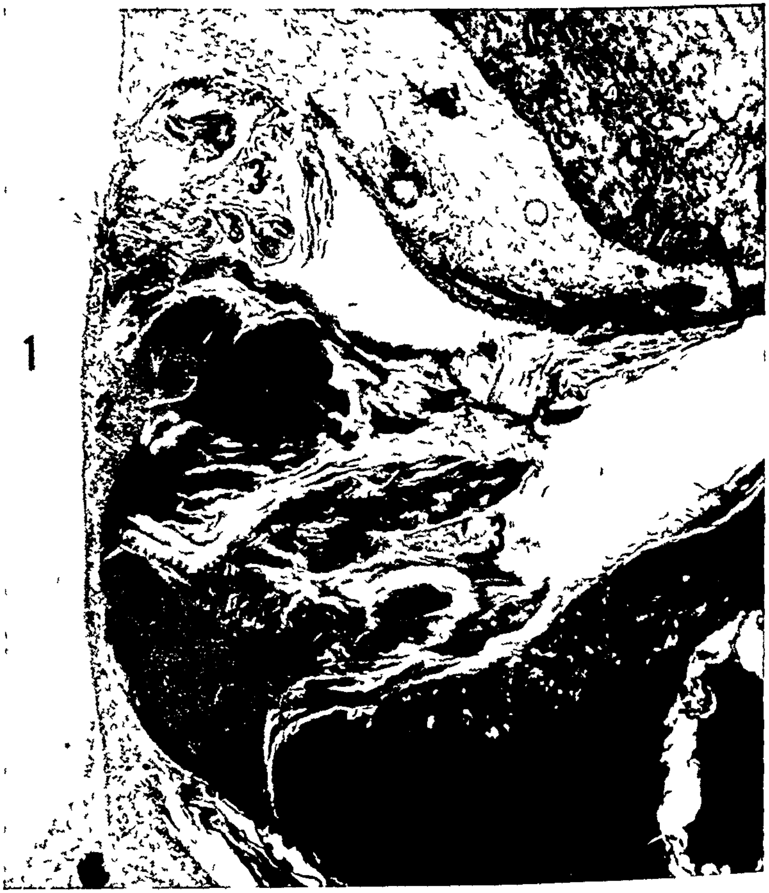
The membrana propria did not show signs of proliferation of fibers while the epithelium of the mucosa sometimes showed hyperplasia In some instances cornification and exfoliation of the epidermis of the eardrum produced a cholesteatoma-like formation in the external canal (see Fig 2)

Several ears showed necrosis of the eardrum or perforations associated with otitis media Purulent collections were found between the membrana propria and epidermis in many cases These collections were identified as having occurred in bullae as already described

Fibrous tissue and new bone, intermingled with bone resorption, proliferated extensively in the tympanic cavity (see Figs 5 and 9) This proliferative reaction sometimes invaded the cochlea through both the round window and the otic capsule Labyrinthitis ossificans, ossification of the ossicular chain and even sequestration of the cochlea were found

Commentary These observations indicate that irritative agents produce hyperplasia of the epidermis of the eardrum which may give rise to a cholesteatoma-like formation in the external canal This finding and previous investigations,[6 8 9 14 15] tend to support the opinion that irritation *per se* of the epidermis of external canal and/or tympanic membrane may play an important role in the pathogenesis of aural cholesteatoma

The overproduction of fibrous and bone tissues represents a reaction to the poison Fibrosis and ossification have been reported in muscles after injection of quinine[16] Secondary infection probably played an important role The extensive work of Friedmann[17 18] demonstrated that chronic otitis media in the guinea pig may lead to osteoplastic obliteration of the ear cavities



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II *Immigration of Epidermis Through a Marginal Perforation*

The following cases substantiated the theory that the epidermis of both skin and tympanic membrane may immigrate through a marginal perforation

Case 1 Guinea pig injected with quinine sulfate and sacrificed seven days later The histologic examination of the temporal bone revealed necrosis of the tympanic membrane and inflammation in the middle ear The epidermis of the skin had migrated into the tympanic cavity (see Fig 3) where it progressed over the hyperplastic mucosa which had lost its epithelium Arrow D points to what remains of membrana propria and arrows A, B and C to regions shown at higher magnification in Fig 4 In A, all cells appeared slightly elongated and became progressively longer and fusiform (B and C) as the leading border of the migratory sheet was approached The nuclei showed similar elongation and apparently did not present mitotic figures

Commentary This temporal bone demonstrates the initial stage of the immigration through a marginal perforation The migratory sheet apparently presents the histologic characteristics of a repairing epithelium, *i e*, reproduces the skin without the corium The photomicrographs of Fig 4, raised the question whether progression of the epithelium is by migration and/or cell division or other mechanism In this particular case, progression seems to be accomplished by migration of cells The absence of mitotic figures suggests that cell division plays no part, at least during this stage of progression. A similar mechanism has been described as occurring during repair of small wounds of the cornea and skin.[16] The possibility that other processes may also contribute to locomotion of the migratory sheet cannot be ruled out The nature of the stimulus that excites cell migration of epidermis into a region where it normally does not exist remains unknown

Case 2 This guinea pig was injected with quinine sulfate and sacrificed 76 days after administration of the drug The histologic examination of the specimen showed partial obliteration of the tympanic cavity and cochlea by new bone and fibrous tissue (see Fig 5) The eardrum and ossicular chain were destroyed and the epidermis of the skin had immigrated and covered the surface of the remaining tympanic cavity There had been complete healing in some areas as shown in Fig 5 but a study of the serial sections revealed other areas where the underlying capsule showed significant changes In some areas as shown in Fig 6, the periosteum of the otic capsule had been absorbed and the substratum of the repairing epidermis extended into the bone along blood vessel spaces and areas of osteolysis The arrows on this photomicrograph point to microcholesteatoma formations in the epidermis In another small area (see Fig 7-A) a focus of bone absorption was found in the

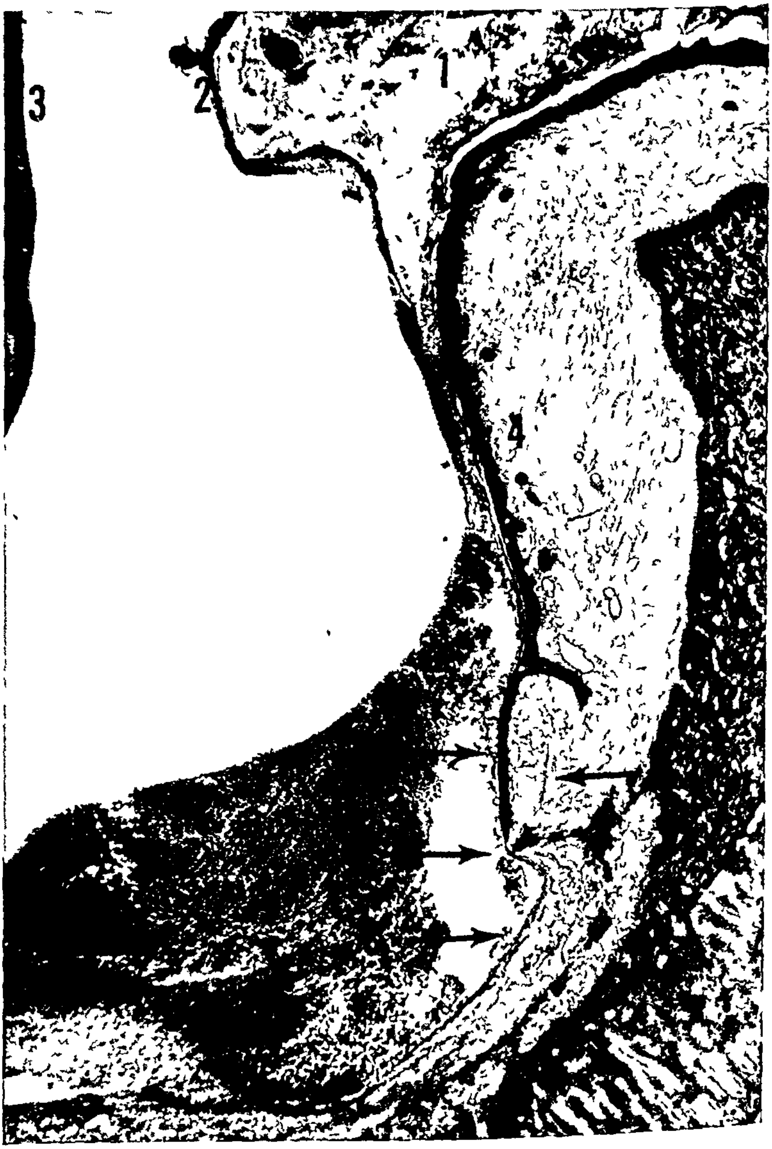

Fig 3 Guinea pig D-31 Animal sacrificed seven days after administration of quinine sulfate Initial stage of the migratory process of epidermis through a marginal perforation Middle ear epithelium almost completely absent 1 external auditory canal filled with pus, 2 malleus, 3, otic capsule 4 skin of external canal Arrows A B and C point to regions of the migratory sheet which are shown at high magnification in Fig 4 Arrow D points to remaining fibers of membrana propria Notice continuity of epidermis of skin and migratory sheet (H-E X31)

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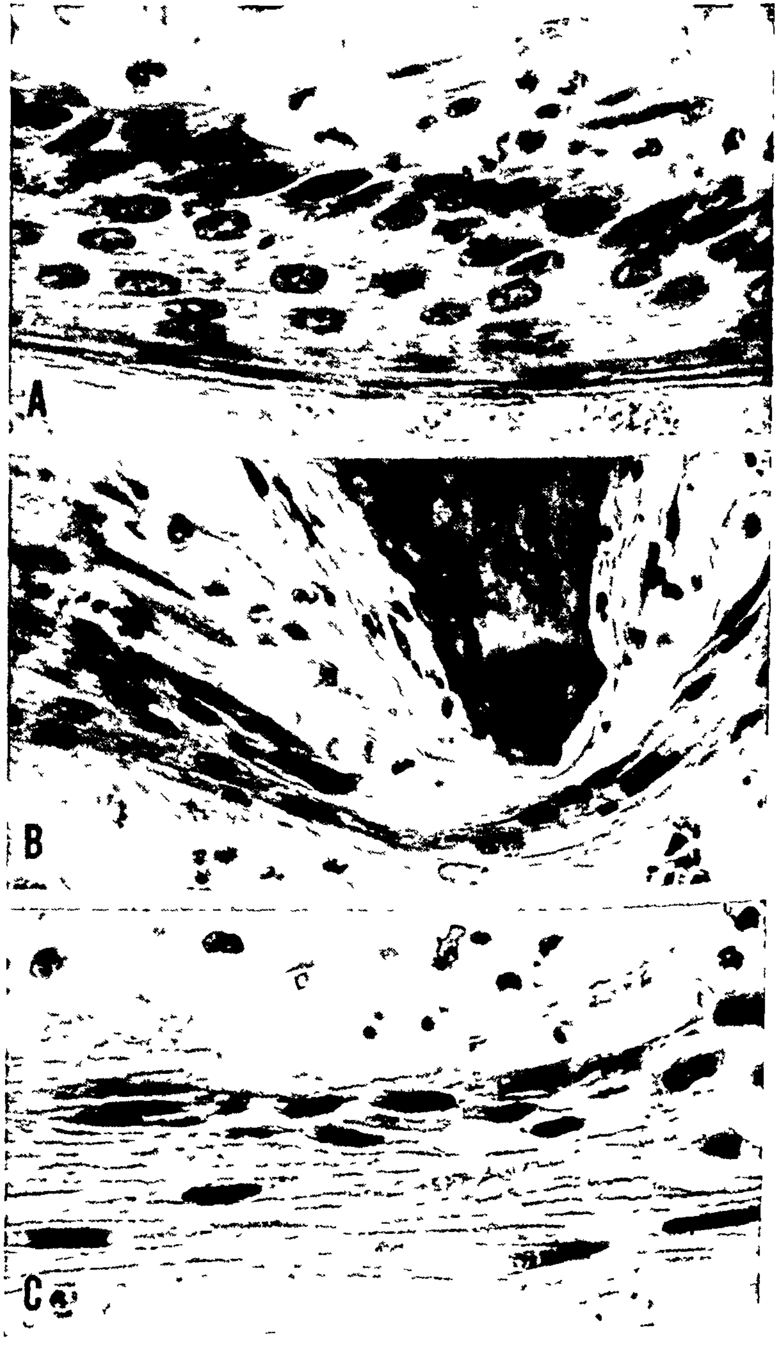

Fig 4 Histologic features of migratory sheet in the initial stage These photomicrographs correspond to regions pointed by arrows A B and C respectively of Fig 3 A —Cells and nuclei are slightly elongated (H-E X715) B —Cells and nuclei are more elongated and mitotic figures seemed to be absent (H-E X650) C —Cells and nuclei are fusiform No mitotic figures are seen (H-E X840)

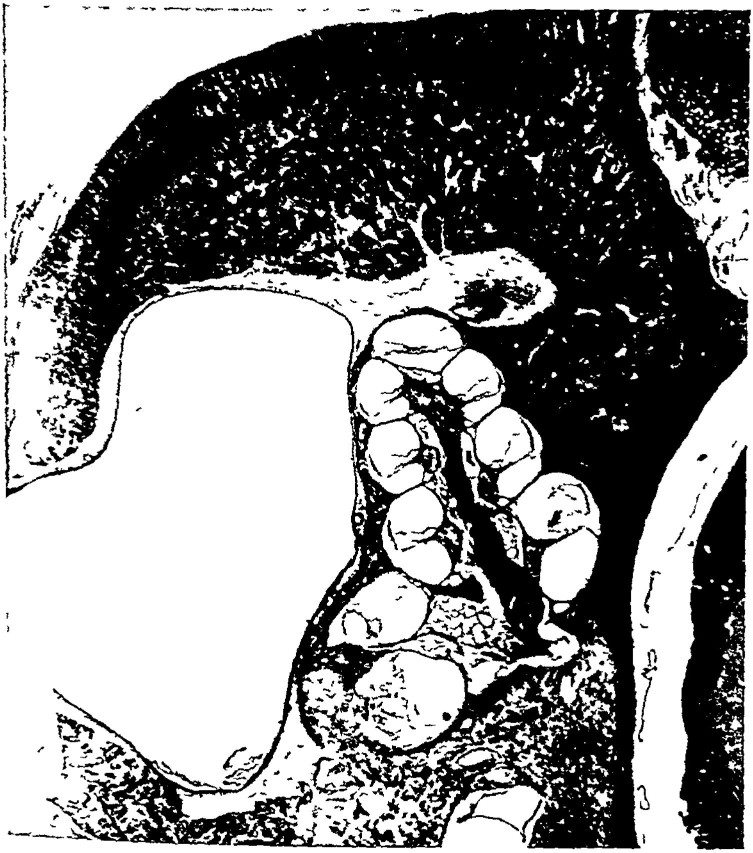

Fig 5 Guinea pig D-45 Partial osteoplastic obliteration of the tympanic cavity and first turn of the cochlea. Eardrum malleus and incus were destroyed The epidermization of tympanic cavity was complete The middle ear space appears free of infection and debris from immigrating epidermis (H-E X13)

otic capsule under a smooth epidermis The basal cells were more active than in other regions and the focus of absorption presented numerous osteoclasts (large polynuclear cells in Fig 7 B) The photomicrograph of Fig 8 illustrated another interesting area of this specimen It shows a small gap in the otic capsule due to a sequestrum (arrow A) The epidermis invaginated into the scala vestibuli forming a small pocket where debris accumulated in a cholesteatoma like formation including the sequestrum The repairing epithelium extended somewhat into the scala vestibuli (arrow B) giving a surface covering for the fibrous tissue which had obliterated the cochlear spaces

Commentary This case represents that final stage of the migratory epidermis It covered completely the remaining tympanic cavity and appeared dry and smooth except for the small region shown in Fig 8 From the histologic point of view this repairing epidermis may be considered as the matrix of cholesteatoma

The importance of Figs 5, 6 and 7 is that they may have some bearing on the following question pointed out by Lindsay,[12] "In the absence of infection, moisture and retention of debris and crust *is there any evidence* that the epithelial lining derived from the cholesteatomatous matrix causes absorption of underlying bone?" This author,[12] Baron,[10] Wolff[20] and others believed that, under these conditions, there was no evidence of biological activity of the repairing epithelium On the other hand Walsh, et al,[21] after histopathologic studies of ossicles removed from cases of cholesteatoma and also on the basis of experimental observations, believed that the matrix

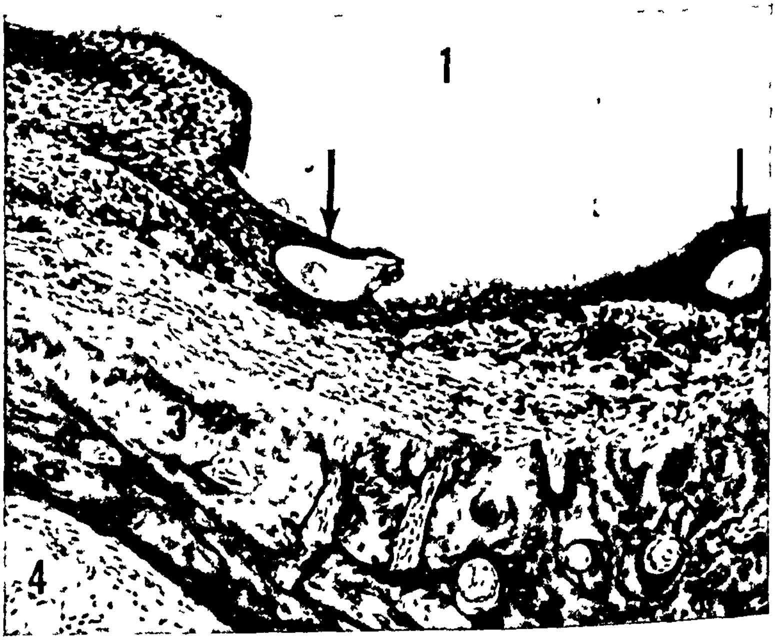

Fig 6 Guinea pig D-43 Resorption of otic capsule under an apparently smooth repairing epithelium The substratum invaded the bone through blood vessel channels and foci of osteolysis Arrows point to microcholesteatomas in the migratory epidermis 1 tympanic cavity, 2, epidermis, 3 otic capsule, 4, scala vestibuli (H-E, X130)

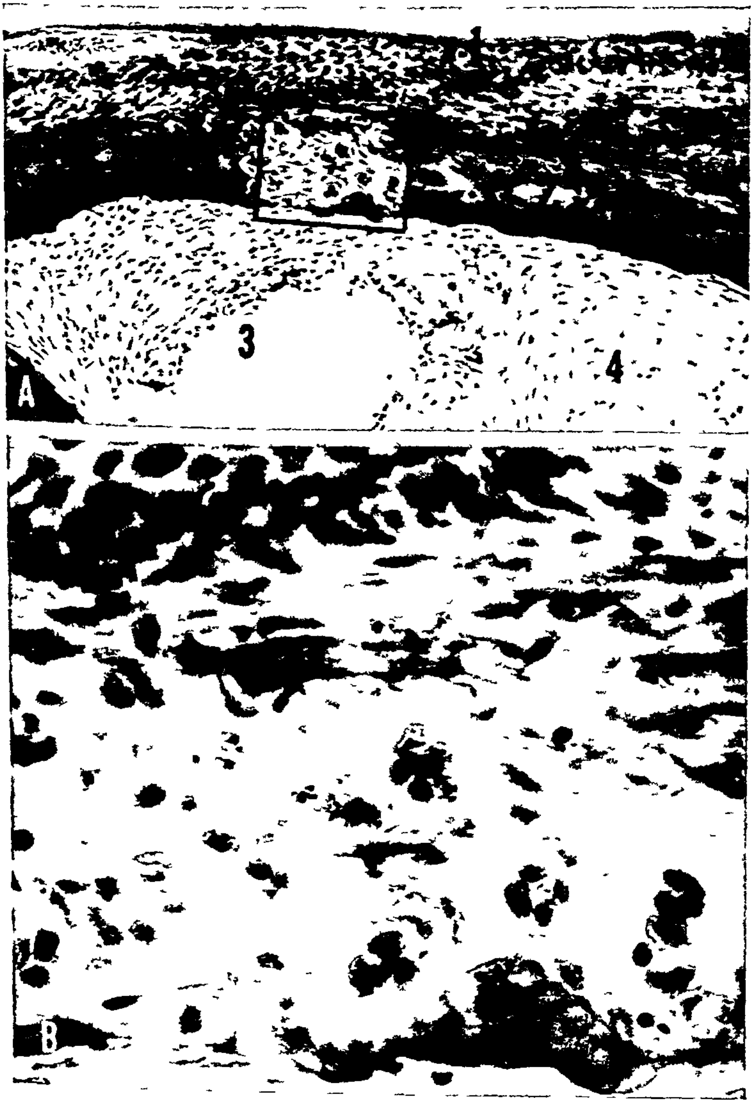

Fig 7 Guinea pig D-43 Focus of resorption in the otic capsule underneath smooth repairing epidermis A.—1 repairing epidermis 2 otic capsule 3 scala media 4 scala vestibuli obliterated with fibrous tissue The enclosed region is shown at higher magnification in the lower photomicrograph Arrow points to a microscopic purulent collection enclosed in the specimen (H-E X130) B—Osteoclastic resorption of otic capsule Numerous osteoclasts forming niches in the bone are seen (H-E X330)

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The importance of Figs 5, 6 and 7 is that they may have some bearing on the following question pointed out by Lindsay,[12] "In the absence of infection, moisture and retention of

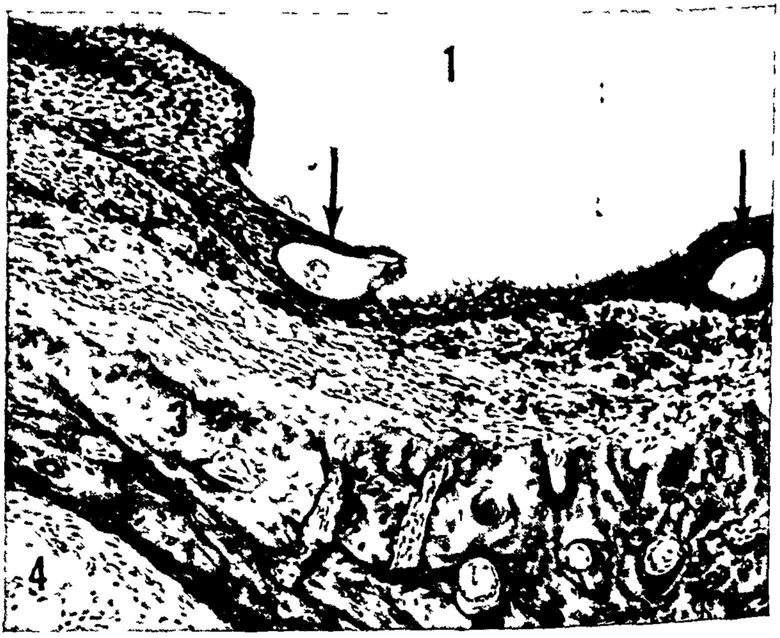

Fig 6 Guinea pig D-43 Resorption of otic capsule under an apparently smooth repairing epithelium The substratum invaded the bone through blood vessel channels and foci of osteolysis Arrows point to microcholesteatomas in the migratory epidermis 1 tympanic cavity 2 epidermis, 3 otic capsule 4 scala vestibuli (H-E, X130)

debris and crust *is there any evidence* that the epithelial lining derived from the cholesteatomatous matrix causes absorption of underlying bone?" This author,[12] Baron,[19] Wolff[20] and others believed that, under these conditions, there was no evidence of biological activity of the repairing epithelium On the other hand Walsh, et al,[21] after histopathologic studies of ossicles removed from cases of cholesteatoma and also on the basis of experimental observations, believed that the matrix instance had probably occurred during an earlier more active stage of the inflammatory reaction. The more logical interpretation is that absorption had reached its endpoint and that the reparative process was now dominant.

The photomicrograph of Fig. 8 emphasized the previous point and also that a cholesteatoma-like formation may take place in a microscopic pocket. Such a small lesion can hardly be recognized by means other than microscopic magnification.

Case 3. This guinea pig was injected with quinine sulfate and sacrificed 21 days after administration of the drug. The histologic preparations of the temporal bone showed partial obliteration of the middle ear and cochlea by new bone and inflammatory tissue. The tympanic cavity was reduced to a small pocket devoid of mucous membrane which communicated with the external ear through a narrow channel opening through a large central perforation. The hyperplastic epidermis of the eardrum immigrated through the perforation and covered the channel almost completely (see Fig. 9). The continuity of epidermis with the migratory sheet is shown in the lower photomicrograph.

Commentary. This case illustrates that under favorable conditions, the epidermis of the pars tensa has the potentiality to grow inwards similar to that of the skin. These conditions seemed to be: *1.* presence of a perforation, *2.* maintenance of a hyperplastic epidermis by inflammation and *3.* necrosis of mucosa in the tympanic cavity.

III. Immigration Without Perforation of Eardrum.

It must be clearly understood that by "perforation" is meant a discontinuity or opening which includes *all* layers of the pars flaccida and/or the pars tensa.

Clinical and histopathological evidence indicates that there are two mechanisms by which the epidermis may invade the middle ear spaces without perforating the tympanic membrane, namely, by inward displacement of Shrapnell's membrane or the pars tensa leading to a persistent retracted pocket, and by ingrowth of basal cells through clefts or openings in the corium of Shrapnell's membrane or the membrana propria of the pars tensa.

The first mechanism has been proposed several times in the past.[11,12] The progressive development of a pocket leading to cholesteatoma formation is a fact established by clinical observations. The pocket may not be perforated, at least in the

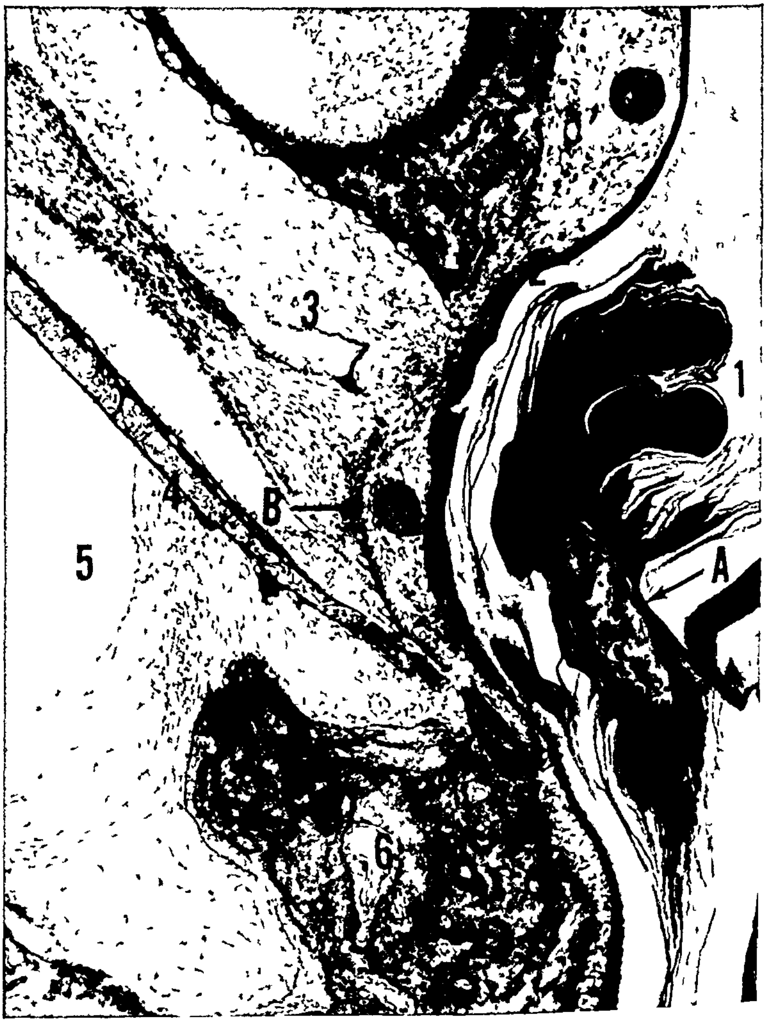

Fig 8 Guinea pig D-43 Microscopic cholesteatoma-like formation on the otic capsule 1 tympanic cavity 2 repairing epidermic 3 scala vestibuli filled with fibrous tissue 4 spiral lamina, 5, scala tympani partially obliterated with fibrous tissue 6 new bone in scala tympani Arrow A points to sequestrium of the otic capsule and arrow B to a transverse section of a cone of basal cells in scala vestibuli (H-E X53)

has a biologic activity Walsh[22] suspected that even in cases of a dry smooth cavity covered with a cholesteatoma matrix, absorption of bone may take place beneath the lining The photomicrographs of Figs 5, 6 and 7 might be interpreted as supporting the opinion of Walsh, et al,[21] and Walsh[22], however, it must be pointed out that the absorption of bone in this instance had probably occurred during an earlier more active stage of the inflammatory reaction The more logical interpretation is that absorption had reached its endpoint and that the reparative process was now dominant

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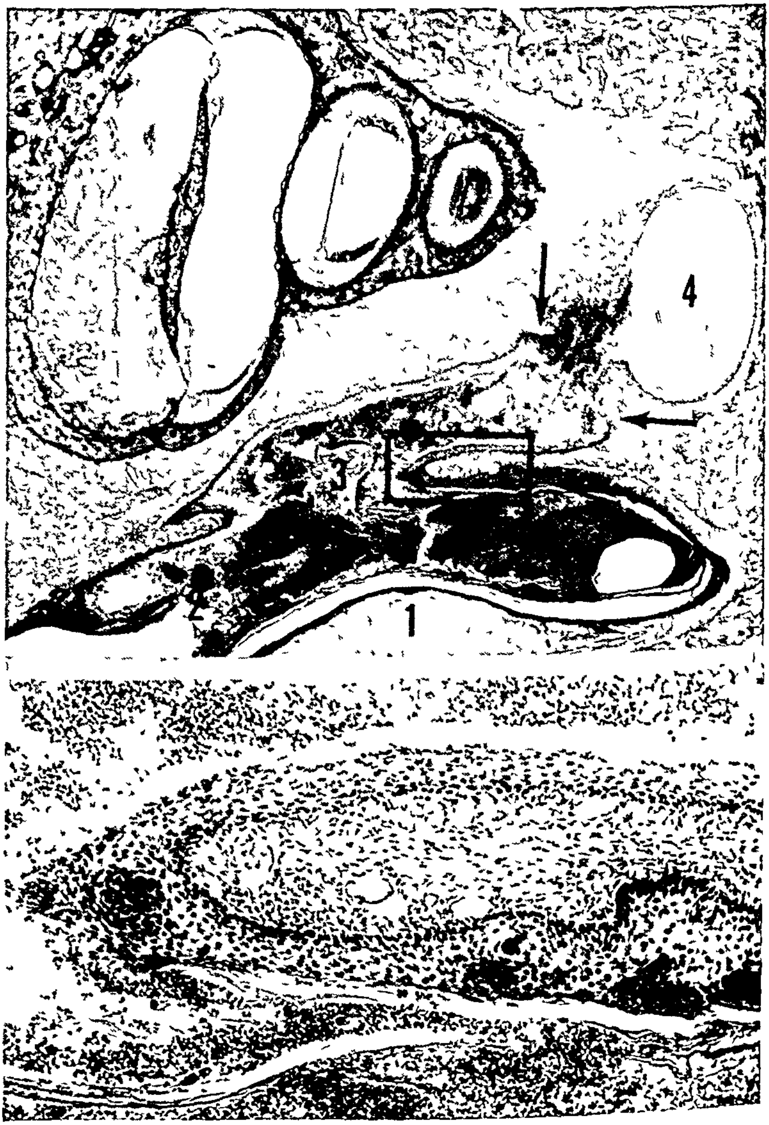

Fig 9 Guinea pig D-46 Immigration of epidermis of eardrum through a perforation Middle ear and scala tympani of first turn obliterated with fibrous tissue and new bone Middle ear epithelium is absent 1 skin of external canal 2 external canal 3 perforation of eardrum, 4 remaining tympanic cavity which communicates with the external ear by a channel lined with epidermis of eardrum Arrows point to head of immigrating sheet The enclosed region is shown at high magnification in the lower photomicrograph This demonstrates continuity of epidermis with migratory sheet (X-120)

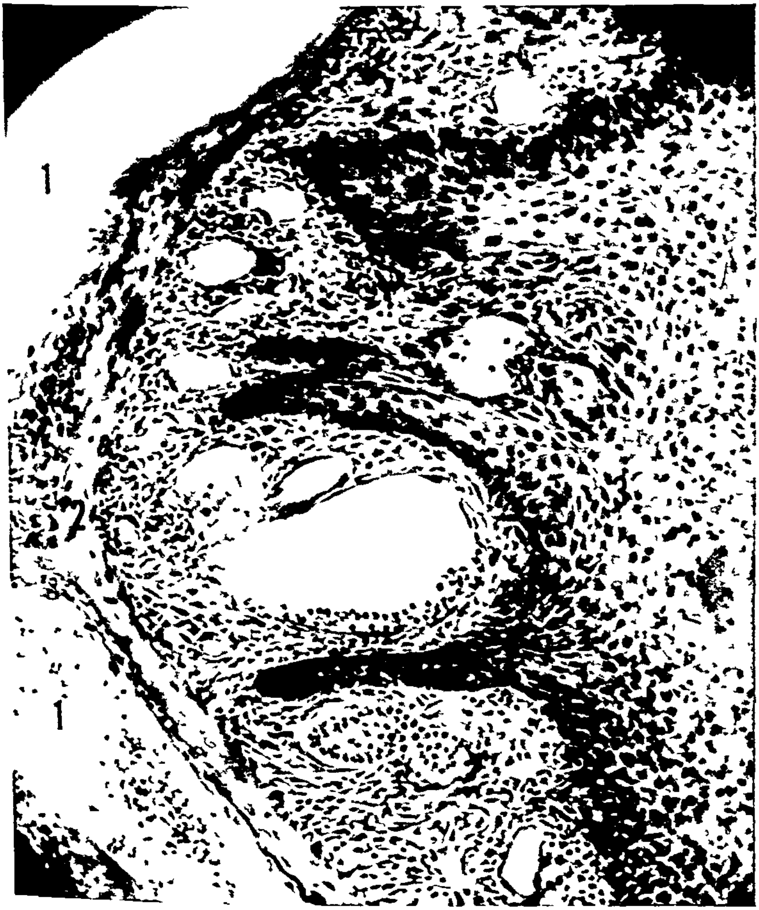

Fig 10 Guinea pig D-35 Animal injected with quinine hydrochloride and sacrificed seven days later The figure illustrates the potentialities of basal cells of eardrum s epidermis to proliferate The hyperplasia of epidermis is striking At this magnification (H-E X185) a normal epidermis is barely visible The basal cells proliferated forming long cones which extend into the hyperplastic substratum 1 tympanic cavity 2 malleus 3 hyperplastic epidermis of pars tensa

initial stages, therefore, the mechanism of this cholesteatoma is a mechanical displacement of either Shrapnell's membrane or the posterosuperior quadrant of the pars tensa The displacement is brought about by cicatricial contraction of fibrous tissue formed in the epitympanic space during the quiescent period following an acute inflammation The conversion of the granulation tissue to fibrous tissue and this to scar tissue with each acute episode, is the most characteristic feature of chronic inflammation[16] wherever this may occur. Development of a pocket should be differentiated from ingrowth of epidermis, however, when perforation occurs during or after its formation then a true immigration of epidermis may take place.

The second mechanism, the ingrowth of epidermis without perforation is attributed to proliferation of basal cells. The potentialities of basal cells to proliferate are demonstrated in Fig. 10. The photomicrograph corresponds to a section passing through the malleus. The hyperplasia of epidermis and formation of cones extending in the substratum is striking.

A defect in the corium or in the membrana propria either during or without development of a pocket may permit the ingrowth of cones of basal cells in the manner postulated by Ojala and Saxén.[6] Figs. 11 and 12 illustrate the point that the basal cells may immigrate even through a microscopic opening of the membrana propria. The specimen was taken from a guinea pig injected with quinine sulfate and sacrificed seven days after administration of the drug. The lining of the middle ear was hyperplastic, and a small amount of precipitate with macrophages was found in the hypotympanum. The external canal was partially filled with debris. The epidermis of eardrum was hyperplastic and detached from membrana propria except at the umbo. The serial sections revealed two microscopic openings of the membrana propria near its attachment to the malleus (numbers 6 and 7 of Fig. 12). The basal cells of the epidermis immigrated through both perforations into the middle ear. The migratory cells presented a fusiform shape, and their nuclei were elongated.

Commentary. This case supports the opinion of Ojala and Saxén[6] and Ruedi[7,8,9] that under an inflammatory stimulus the basal cells may proliferate and immigrate without a perforation of epidermis. The microscopic opening in membrana propria may be considered as corresponding to the cleft or opening in the corium of Shrapnell's membrane, postulated by Ojala and Saxén.[6] It is apparent that these openings are not visible in the living animal because of their microscopic size and because they are covered by a hyperplastic epidermis.

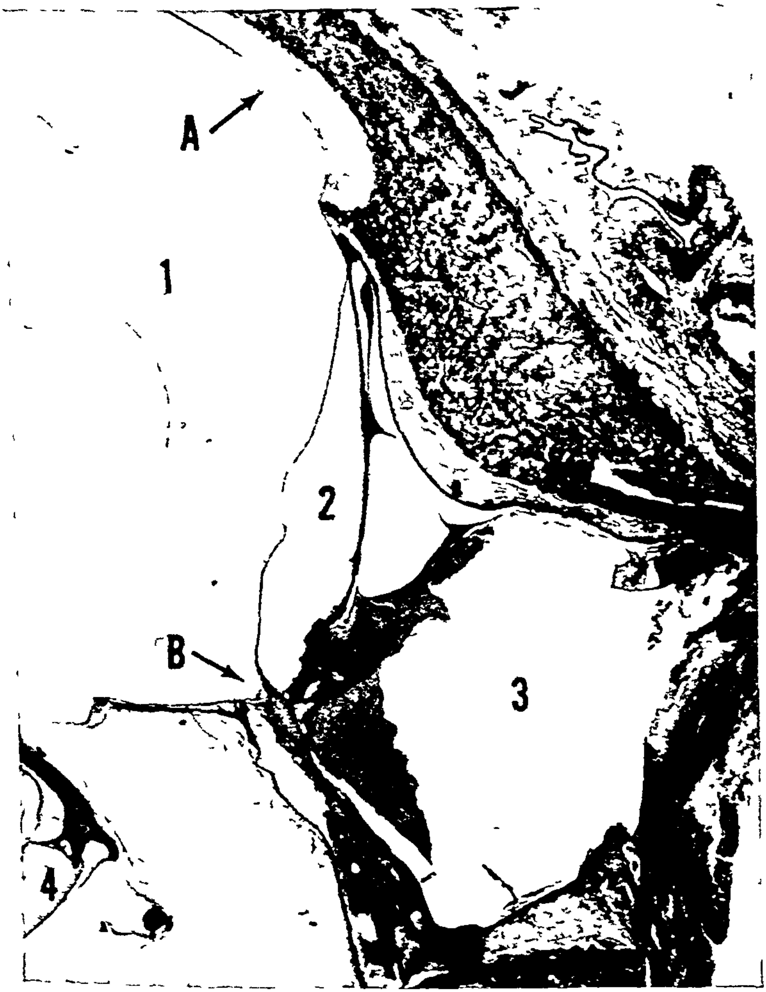

Fig 11 Guinea pig D-79 Animal sacrificed seven days after administration of quinine sulfate The specimen illustrates the initial stage of immigration of basal cells through a microscopic defect in the membrana propria of the pars tensa 1 tympanic cavity with a hyperplastic mucosa (arrow A) 2 bulla in pars tensa 3 external canal containing pus and debris 4 cochlea Arrow B points to the region of immigration This area is shown at higher magnification in Fig 12

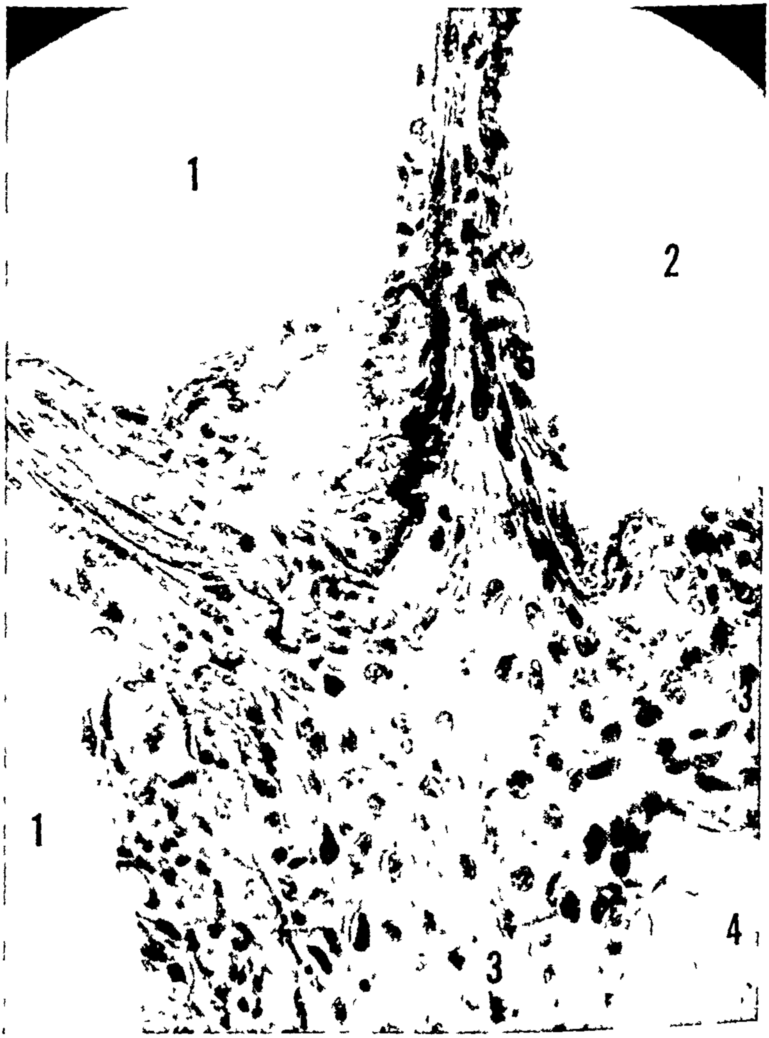

Fig 12 Guinea pig D-79 This photomicrograph illustrates that the basal cells of epidermis of pars tensa can migrate through an opening of the mebrana propria The epidermis was not perforated 1 tympanic cavity 2 bulla formed between membrana propria and epidermis 3 epidermis of pars tensa 4 external canal 5 membrana propria 6 and 7 microscopic openings in membrana propria through which basal cells of the epidermis ingrow Notice the change in shape of migratory basal cells, the cells and nuclei become elongated (H-E X385)

This temporal bone together with previous observations[6-8,9] demonstrates that the basal cells of the epidermis of a nonperforated eardrum have the potentialities to ingrow, providing the membrana propria of pars tensa and/or corium of pars flaccida are damaged It is possible that a so-called congenital or primary cholesteatoma in the attic or middle ear may develop by this mechanism The potentialities of basal cells to proliferate are demonstrated in Fig 10, but the question remains whether these cones can penetrate an intact membrana propria or corium of Shrapnell's membrane

Ruedi[9] observed in the guinea pig that an irritating substance applied over the inner surface of the tympanic membrane produced a strong stimulation of basal cells which migrated into the middle ear without perforation of eardrum In the tympanic cavity a cholesteatoma developed Ruedi[9] did not specify whether a defect in the membrana propria due to the inflammation had preceded the ingrowth of hyperplastic basal cells If this was true, then the mechanism of immigration was similar to that postulated by Ojala and Saxén[6] If there was no defect in the membrana propria, then the postulate of Ruedi implies that the basal cells can penetrate an *intact* membrana propria Similarly, the basal cells of pars flaccida might penetrate an *intact* corium In our series of animals no clear evidence of ingrowth of basal cells through *intact* membrana propria or corium could be demonstrated

The statements of Ojala and Saxén[6] and the experiments by Ruedi[8,9] are important for understanding the pathogenesis of attic cholesteatoma, because they offer an explanation for those cases which do not show an apparent perforation of the tympanic membrane

SUMMARY

Histologic observations in temporal bones of a series of experimental animals exposed to the irritative effect of a suitable quinine preparation placed in the middle ear and sacrificed at various intervals demonstrated certain behavior of the epidermis of the external canal and eardrum which is pertinent to the development of cholesteatoma

1 The epidermis of the bordering skin can migrate through a marginal perforation when the middle ear epithelium is absent

2 The epidermis of the eardrum can migrate through a central perforation to cover the middle ear mucosa when it is devoid of epithelium

3 A hyperplastic basal cell layer of the epidermis can migrate inward without an apparent perforation of the eardrum providing that the membrana propia of the pars tensa or the corium of the pars flaccida has been eroded by the inflamatory process

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PROBLEMS AND COMPLICATIONS IN HEAD AND NECK SURGERY*

GEORGE A SISSON, M D,

Syracuse, N Y

The surgeon performing radical operations for the control and cure of cancer of the head and neck must accept the discouraging fact that only 35 per cent of his patients can be cured He can prolong life in 50 per cent of his cases but do little for 15 per cent If a surgeon is unable to adjust to these grim facts he has no business in head and neck surgery Though his efforts may be sincere his judgment is impaired, and he will do more harm than good dabbling with something that someone else better indoctrinated might manage properly Unfortunately, this is the first and greatest problem in head and neck surgery Too many doctors with pessimistic attitudes are managing cancer of the head and neck in a cursory manner What often starts as a biopsy too often ends up as an insufficient excision This is particularly true in the management of intranasal and intraoral cancers We continue to see nodes in the neck indiscriminately biopsied or excised when an aspiration biopsy would be a superior technique and, after all, would offer the patient a better prospect should a radical procedure be indicated in the final analysis If the shrouds of pessimism which embrace the practice of head and neck surgery are to be removed, we, as otolaryngologists, should be the first to recognize this problem (see Figs 1-a through 1-e)

The American Board of Otolaryngology has committeed our specialty to the responsibility of managing cancer of the head and neck We must prove that we can accept this responsibility and actually lead the way towards improved cancer control

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*Read at the meeting of the Eastern Section American Laryngological Rhinological and Otological Society Inc Philadelphia Pa Jan 8 1960

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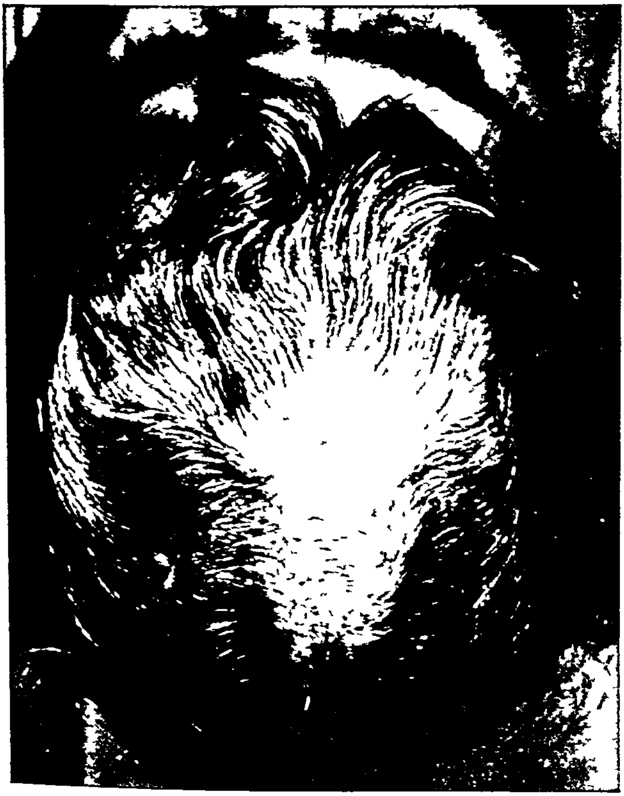

Fig 1-a Squamous cell carcinoma of scalp in 40-year-old female previously excised three times

Another problem is late diagnosis Within a three-week period I saw in consultation six cases, mostly young people, with markedly advanced lesions either at the upper limits of resectability or inoperable In each case there was a weak link in early definitive diagnosis One patient ignored symptoms, in another case two doctors were incapable of a complete examination of the ears, nose, throat, nasopharynx, etc , other organs teeth, stomach, bowel, blood stream, lungs, heart, even the psyche were thoroughly investigated while the throat and neck were overlooked Two cases were diagnosed as cardiacs with emphysema To the chagrin of those in attendance tracheostomies relieved both cardiacs In another

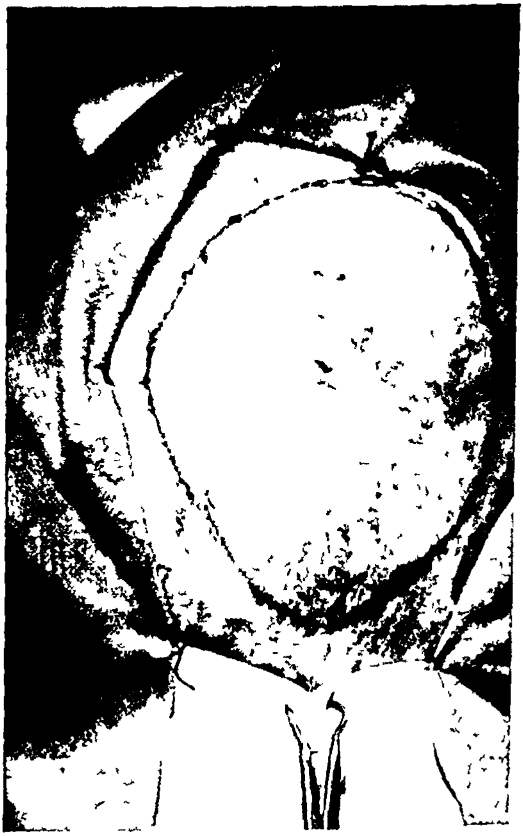

Fig 1-b Lesion in old scar demonstrating extent of resection necessary to eradicate the recurrence of carcinoma shown in Fig 1-a

case the hypopharyngeal lesion was so massive that the top was viewed easily when using a tongue depressor We must stress thorough examinations for minor and minimal complaints, and patients should be re-examined at intervals if the first examination is not completely satisfactory Emphasis upon early diagnosis should be continued for lay groups as well as for professional (see Figs 2 and 3)

Critical problems arise when selecting the borderline cases to be offered radical surgery Almost any tumor of the head and neck regardless of its stage can be resected by the experienced surgeon, however, enthusiasm for a procedure should not supplant mature judgment, experience or sensitive intuition Resectability does not necessarily mean operability

If the head and neck surgeon feels reasonably certain that surgery cannot improve, nothing should be done A self-pitying recriminatory patient does as much to harm the cancer control program as the physician who misses an early diagnosis To be considered also are the general physical and mental qualities of the patient which might defeat successful surgery Only once have I been advised by a competent internist that surgery was contraindicated because of cardiac reserve, however, I often have withheld surgery because of the patient's psychological background I have seen surgery precipitate frank psychosis on two occasions, both of these patients were young females without definite aberrations in their past medical background Conversely, I have performed

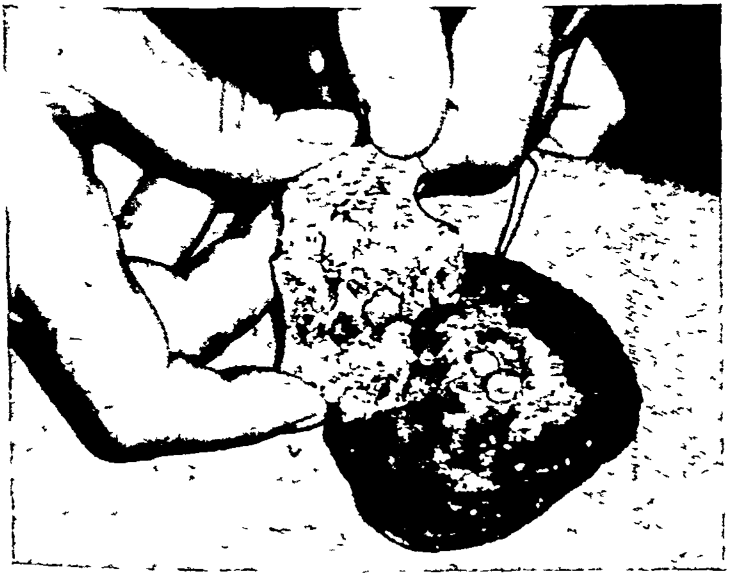

Fig 1-c Same patient shown in Figs 1-a and 1-b with specimen showing extension of cancer into cranial wall Cranial defect was replaced with a tantalum plate

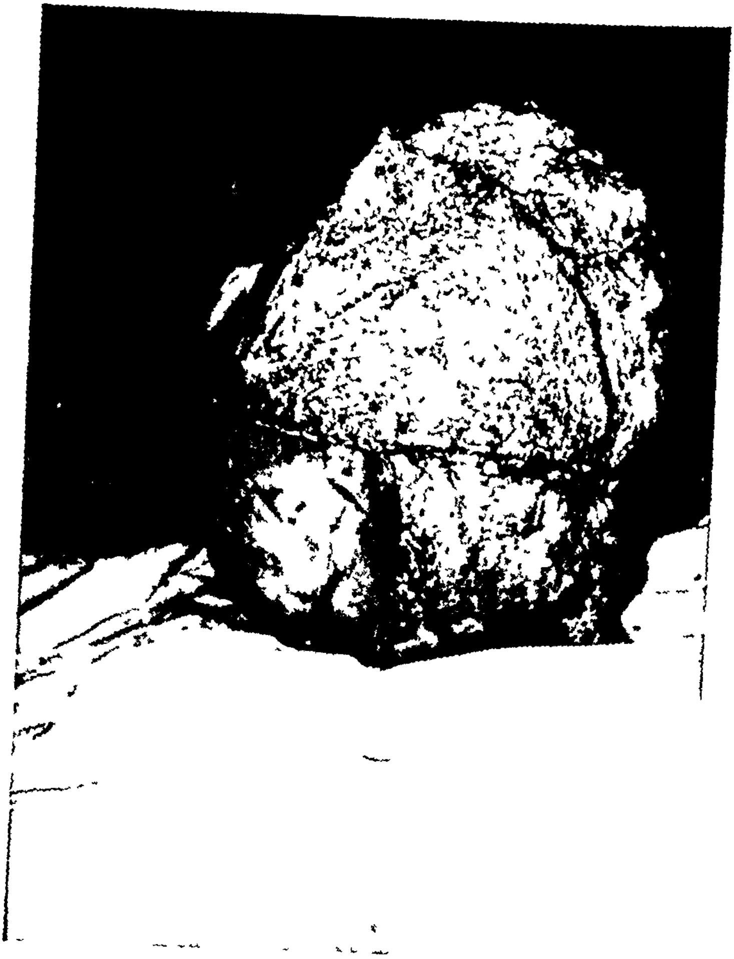

Fig 1-d Rotation and migration of pedicle flaps necessary to close original defect created by removal of cranial wall (see Fig 1-b) Left lower corner shows split thickness graft used to close donor site

radical procedures without event on highly emotional nervous individuals, young and old In many respects the added stress placed on these latter cases seemed to recruit reserves in their personality which neither they nor their families believed present

When the wrong case is selected for radical surgery the patient often causes others (relatives and friends) to feel that all such surgery is mutilating and thus negate its use where it could be beneficial It has been said that there are

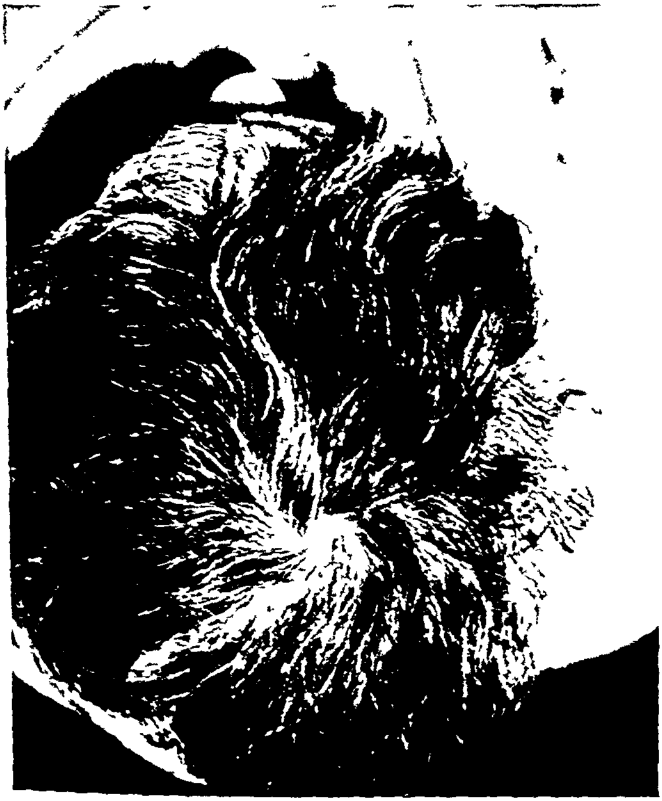

Fig 1-e Same patient shown in Figs 1-a through 1-d as she appeared nine months postoperatively

no contraindications to cancer surgery and this might also be said of palliative X-ray; however, unless the surgeon and radiologist agree that palliative X-ray therapy has a definitive purpose it may possibly end by being palliative only to the conscience and convenience of the doctor

The art of treating carcinoma of the head and neck is in the physician's ability to choose the best method of therapy whether surgery, irradiation, both or neither This ability is more difficult to acquire than the technical skill learned in the dissecting laboratory and the operating room In neck metastasis, in carcinomas closely adhering to or invading bone in irradiation failures and in salivary tumors, surgery usually is

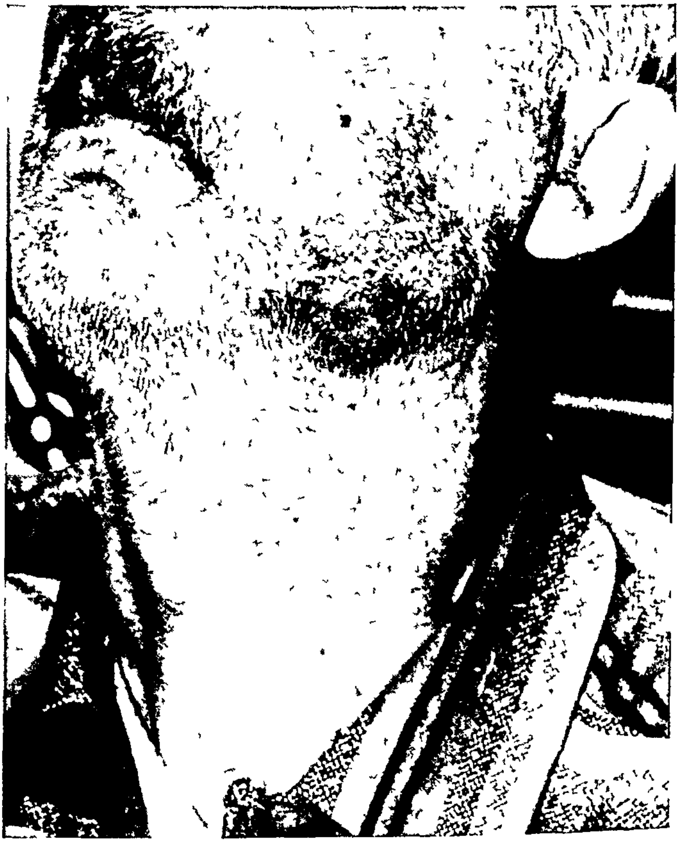

Fig 2 Far advanced inoperable carcinoma of the sinus presenting as mass in left neck

the undisputed choice of therapy In early laryngeal, nasopharyngeal and tonsil lesions irradiation often supercedes surgery In most carcinomas of the head and neck combined modalities are used for best results If irradiation is selected for the first modality the surgeon should follow the patient closely to check for recurrent disease Conversely, if surgery is the first modality, with later use of radiotherapy likely, the radiotherapist should be invited to meet the patient as soon as possible so that he too can follow the progress and offer his modality at the propitious time In controversial cases pretreatment conferences with both surgeon and radiotherapist in attendance are advisable

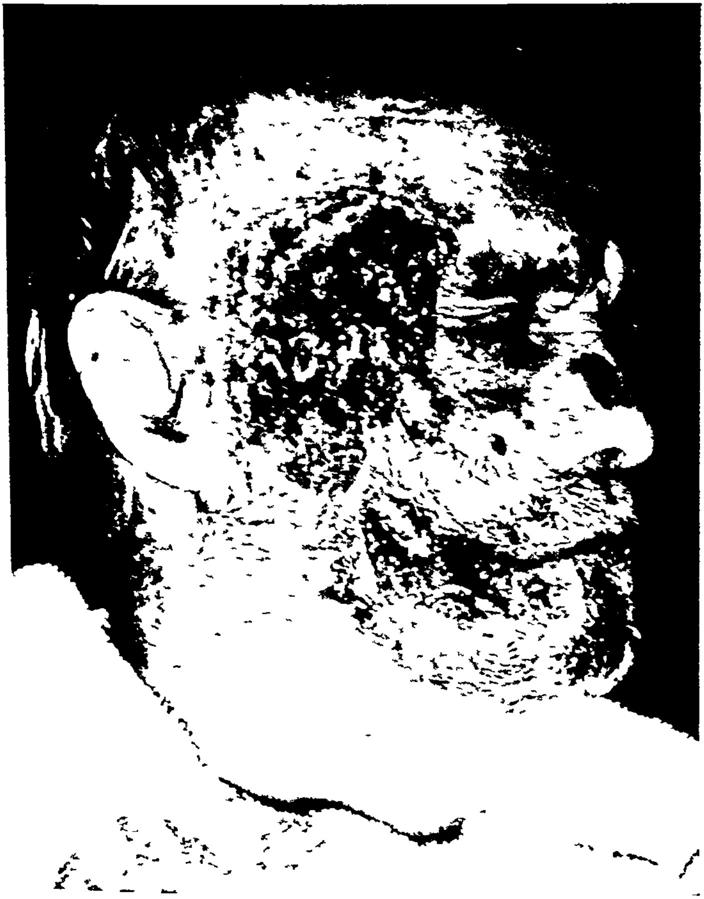

Fig 3 Far advanced inoperable multiple basal cell carcinoma improperly managed

COMPLICATIONS

The only way to avoid operative and postoperative complications in head and neck surgery is to refer these cases to someone else The surgeon accepting total responsibility must be ready to suffer numerous disappointments and frustrations. Major complications may be classified into two categories immediate and late The former come from operative risks or from immediate functional changes due to loss of anatomical structures sacrificed during surgery The latter usually are functional disabilities and unsatisfactory rehabilitation to the loss of anatomy

In the first group come those immediate and unexpected technical complications encountered at the operating table Often, after several hours of dissection, a surgeon will find the lesion beyond the limits of planned surgery, this situation occurs most frequently in tongue and sinus lesions Admittedly, head and neck surgeons deal with narrower margins of excisional safety than abdominal and thoracic surgeons For example, one cannot always make a preoperative decision as to whether or not a commando procedure will suffice Findings, after entering the deeper spaces, often necessitate the inclusion of the larynx and the base of the tongue Often, to gain another two or three millimeters, it may be necessary to remove the entire tongue

Another technical stumbling block is sudden death on the operating table During the past eight years, with a total operating experience of over 500 cases, this has happened twice In one case an air embolus was introduced when blood under pressure was allowed to run out The other case, known preoperatively to be a cardiac risk, suffered cardiac arrest from myocardial infarction We have resuscitated successfully and without incident two cases of arrest

Pneumothorax and pneumomediastinum occur in about 5 per cent of the cases, should be suspected at once and treated by underwater drainage for three to four days I have had two complications from "nicking" the thoracic duct and have removed "cottage cheese" from the flaps for several weeks This is a most aggravating complication

Sudden and extreme loss of blood can be a problem during difficult dissections of carotid body tumors or tumors adhering to the carotid bulb These rents are repaired with arterial silk sutures and are covered with tissues from adjacent areas In one case there was a severe drop in blood pressure when a tumor was dissected from the carotid artery This patient lived four days but never regained consciousness We no longer graft the carotid artery, we compress it slowly over a several-day period with the Silverstone clamp The probability of death in using this technique is about 25 per cent Recently Catlin, from Memorial Hospital of New York City, reported a survival after both carotids were sacrificed

When resecting a maxilla both suctions can plug during the crucial moments We operate with two suctions and should have three I have sacrificed intentionally and unintentionally the seventh, ninth, tenth, twelfth and phrenic nerves The seventh nerve is the most troublesome, but 25 per cent of these seem to regenerate spontaneously Massive hemorrhage can complicate a procedure Our rule is to ligate doubly all major arteries and the internal jugular Even so, on some occasions when dissecting deep in the neck I have been compelled to remove the clavicle to stop subclavian bleeding, and when resecting massive disease at the base of the skull I have encountered massive hemorrhage from the internal jugular. In one case it was necessary to transfuse 12 pints of blood in about 20 minutes while bleeding was uncontrolled The patient is alive today and free of disease. It has been five years since surgery

We now close the pharyngostoma while awaiting the reconstruction phase This procedure has spared us many immediate postoperative complications since this is certain to divert the salivary stream and prevent spit, blood and corruption from accumulating under our fresh operative flaps

Another immediate postoperative complication is the development of aspiration pneumonia because of injury to the superior laryngeal nerve If the brachial plexus is injured it will be detected on the first postoperative day, the patient will be unable to move his arm or fingers This becomes a permanent disability if the nerve is resected When the entire thyroid is removed for an infiltrating subglottic lesion we must watch for the development of tetany during the first three days postoperatively As a precaution one can perform Sulkowitch urine examinations two or three times daily for the first two postoperative days If there is any question of deficiency in the parathyroid hormone the usual anti-tetany measures are instituted at once The management of postoperative fluids and electrolytes usually does not present a problem because most head and neck patients are able to be fed through a naso-gastric tube on the first postoperative day. Intake and output, however, must still be carefully checked. The pulse, blood pressure and general clinical appearance of the patient usually alerts us to severe blood loss during the immediate postoperative period. In questionable cases we use a Foley catheter for a reliable and safe check on the urine output. Applying continuous suction to wound drains has decreased the problems of postoperative hematoma. Tracheostomy is routine in commandos, bilateral neck dissections and hemilaryngectomies. With a reliable well trained house staff in attendance, should respiratory obstructions develop, one can be more liberal in partial resections of the maxilla, radical neck dissections and localized intraoral procedures.

I have seen four naso-gastric feeding tubes break away from their moorings. Once it was necessary to do a gastrostomy to remove the tube; the others passed safely through the entire intestinal tract. We now have devised a special technique for properly securing these feeding tubes.

Massive postoperative hemorrhage is always a complication. When the patient vomits a large amount of blood, and particularly if he is on the verge of shock, it is natural for the surgeon, suspecting that operative wounds are bleeding, to consider reopening the neck. This is particularly true in the more radical intraoral procedures where a suture line is exposed to the pharynx or hypopharynx. A word of caution, however. Due consideration should be given to the possibility that reopening the neck wound will *not* solve the problem. It is not unlikely that hemorrhage is from the gastrointestinal tract and could be a complication from the stomach rather than the head and neck. We have seen ulcers bleed under stress of surgery on two occasions.

In spite of the impressive array of antibiotics and chemotherapeutic agents which we have at hand we still must be able to recognize wound infections readily. In our experience postoperative infection usually occurs the second or third day postoperatively, or late on the eighth or tenth day Meticulous technique in the operating room is extremely important, two surgical masks for each assistant are mandatory and we keep conversation to a minimum If recognized early, infection is a minor problem We purposely withhold antibiotics prophylactically as we find it more effective to order them after the body has had an opportunity to build up antibodies during an invasive period Often it is necessary to reopen the wound completely, divert the salivary stream and widely drain all pockets so unrecognized burrowing of infection will not continue and cause slow deterioration of the patient (see Fig 4)

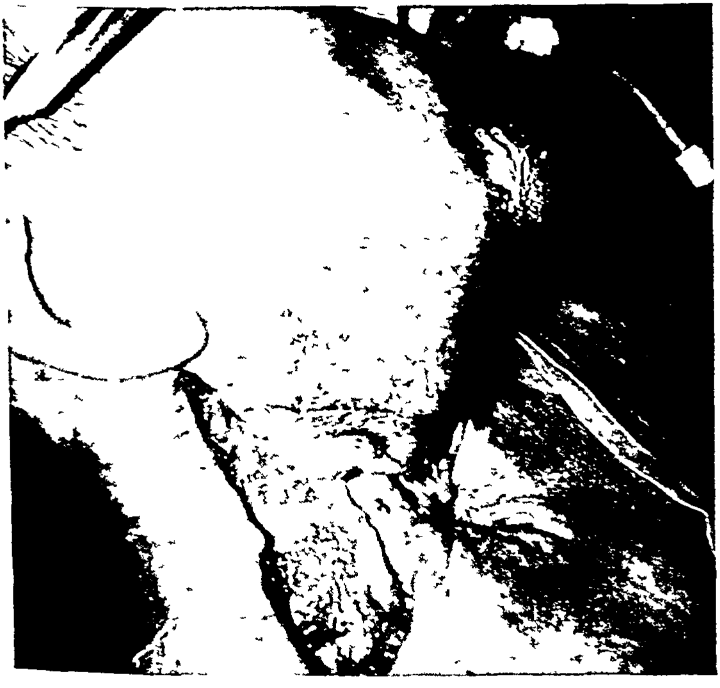

Fig 4 Case after total glossectomy total laryngectomy and radical neck dissections Wound was opened on fifth day to divert salivary stream laterally and away from the trachea. At time of photograph wound is 12 days postoperative and granulation bed is almost ready for secondary closure

Occasionally massive sloughing of skin flaps is encountered

This complication usually is found in cases that have had previous radiation or surgery, and it is particularly discouraging when the carotid artery is under the area which has sloughed The surgeon can raise a skin graft on a pedicle to solve this problem, however, thrombosis of the carotid or carotid blowout with massive hemorrhage can occur at any time and cause death We have experienced all these complications Carotid blowout, particularly if associated with infection, recurrent tumor or postradiation change, is a hopeless situation Lymphedema arises when both internal jugular veins have been ligated at the same time and usually occurs the second or third postoperative day, subsiding within six weeks in 85 per cent of the cases When working around the orbit we suture the eyelids closed to prevent corneal abrasion which can be very serious Tracheitis sicca usually occurs within the first four or five postoperative days This occurs during the winter months and is best treated with Alevaire or Turgomist although we also use pancreatic dornase, a highly active fibrolytic enzyme

The problem of recurrent disease is the most frustrating of all complications If there is any question of recurrence do not temporize, the most favorable time for surgery may be lost by procrastination or deliberation There are some people who need maximum reassurance that all is well, and it is most difficult to tell these patients if recurrence is suspected that an aspiration biopsy, further tests, irradiation or even additional surgery may be necessary

Salivary incontinence or drooling can be a late and disturbing complication If all other methods fail we advocate ligation of the parotid ducts, this seems to help the situation Unintelligible speech, dysphagia and masticatory difficulties are other long-term problems which are kept at a minimum with proper intraoral prostheses The best protheses are those made at the time of surgery by a qualified and competent prosthodontist

A distressing late complication is the poor adjustment of a patient to change in his appearance or disfigurement, most pronounced after major operations on the lower part of the face Frequently, a patient's family and friends accustom themselves to such changes, but the patient cannot Conversely, we also find that society does not always accept these people If this situation occurs it is tragic When a patient adjusts to necessary disabilities it is unbelievable that the rest of us cannot For these and other obviously related reasons we have recently organized a Cancer Rehabilitation Association in our area We hope that this association may solve some of these problems

SUMMARY

Cancers of the head and neck must be diagnosed earlier and must be treated aggressively Cancer control will advance when there are more trained otolaryngologists managing head and neck cancer Extensive experience and insight are essential when selecting borderline cases suitable for radical surgery Team effort among the referring doctor, the head and neck surgeon and the radiotherapist will improve control and reduce the problems and complications in head and neck surgery The services of a competent prosthodontist must be available in communities where radical head and neck surgery is undertaken

1200 E Genesee St

THE DE ROALDES AWARD

At the recent annual meeting of the American Laryngological Association, the DeRoaldes Award and gold medal were presented to Dr Dean M Lierle as a great physician, surgeon, educator and humanitarian and in recognition of his services to otolaryngology and to the American Laryngological Association

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COMMENTS ON TYMPANOPLASTY [*][†]

HAROLD F SCHUKNECHT, M D ,
T MANFORD MCGEE, M D ,
(By Invitation),
and
STANLEY OLEKSIUK, M D ,
(By Invitation),
Detroit, Mich

Our experience in tympanoplasty now consists of 275 operations performed during the past three years[1] For most patients the objectives of tympanoplasty have been achieved, but for various reasons, failures have occurred in about 35 per cent of the cases Some of these failures have been reoperated with success It is increasingly apparent that it is not always possible to arrest the suppurative process and to reconstruct the middle ear at the same operation, it is also obvious that some failures occurred because the nature of the tympanic pathology precluded successful reconstruction

Success in reconstructive middle ear surgery depends upon keen judgment in the selection of cases as well as the proper execution of the operation In this regard we believe that there are several relative contraindications to tympanoplasty, the term relative applying to a combination of factors concerning the nature of the pathology and the skill and experience of the surgeon

I CONTRAINDICATIONS TO TYMPANOPLASTY.

A Malfunctioning Eustachian Tube

It is probably true that reconstructive tympanic surgery can be completely successful only when the middle ear is normally ventilated during swallowing Often it is not possible to detect the passage of air through the Eustachian tube

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[*] Read at the Sixty-Third Annual Meeting of the American Laryngological Rhinological and Otological Society Inc Miami Beach Fla March 15 1960

[†] From the Department of Otolaryngology Henry Ford Hospital

Editor's Note This manuscript received in The Laryngoscope Office and accepted for publication April 6 1960

Panel Discussion on Tympanoplasty

A C FURSTENBERG, Moderator

It is my pleasure and privilege to act as moderator of this Panel Discussion on Tympanoplasty This is indeed a timely, provocative and interesting subject, it is also a complex, intricate problem which to say the least gives rise to many controversial beliefs and opinions

The scope of this subject is infinite New theories in the transmission of sound, new achievements in the technique of tympanoplasty, new investigations into the pathological changes in bone come to our attention almost daily To understand them and to know their values is frustrating to say the least, however, research in this field must go on, and I have no fear that it will not, because the inquisitive impulse of otologists today is fired up and will not stop for a period of appraisal and adjustment at this stage of the program

We are fortunate in having three panelists, each one of whom is possessed of a large and healthy curiosity, a capacity for careful observation and infinite resource I have little hope that they will harmonize national and international discord on this subject, but I am convinced that they will make an honest, non-emotional approach to the problems of tympanoplasty, direct opinions to the real issues and give us some of the answers to the problems which need to be solved today

I have the honor now to call upon Dr Harold F Schuknecht, Chief Otologist of the Henry Ford Hospital, Detroit, Mich

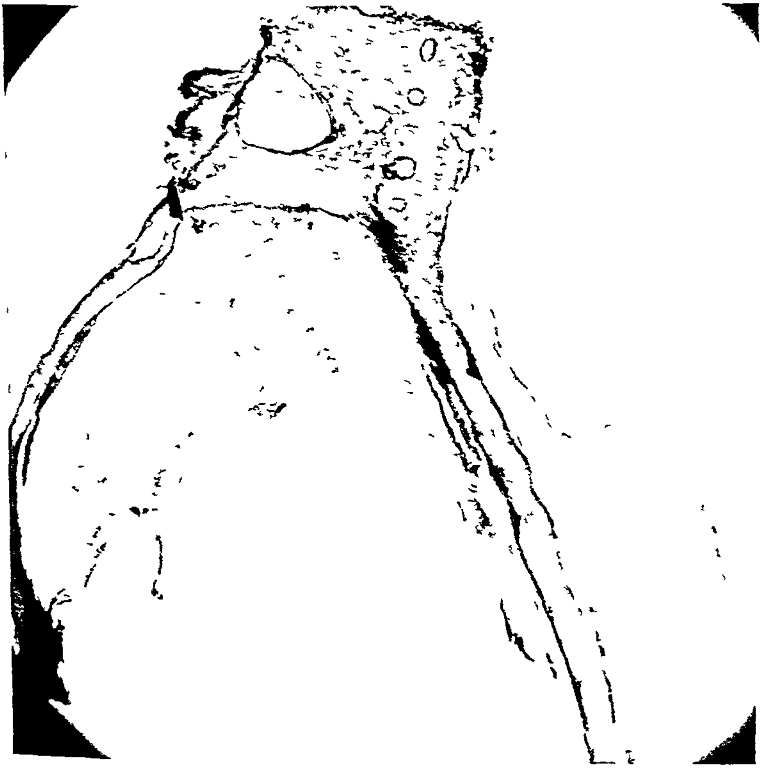


Fig 1-b Hyalinized collagen surrounding devitalized crus of the stapes

C Hyalinized Collagen Deposits (Tympanosclerosis)

This peculiar pathological condition is characterized by the deposition of enormous amounts of hyalinized collagenous material in the submucosal layers of the tympanic cavity[2] The ossicles may be imbedded in it (see Figs 1-a and 1-b) Usually there are also plaques of the material within the layers of the tympanic membrane These so-called "calcium plaques" contain insignificant amounts of calcium The diagnosis can be made preoperatively by identification of the plaques in the tympanic membrane and grayish-white nodules in the mucosa of the promontory These findings are particularly significant in the presence of a large bone-air gap which indicates ankylosis of the ossicles during swallowing because of swollen mucosa, secretions, polyps, etc , therefore, we have used calibrated politzerization and have arbitrarily considered tubal function to be acceptable when air passed through with 40 mm of mercury pressure A tympanoplasty will ultimately fail if middle ear aeration depends upon forced inflation

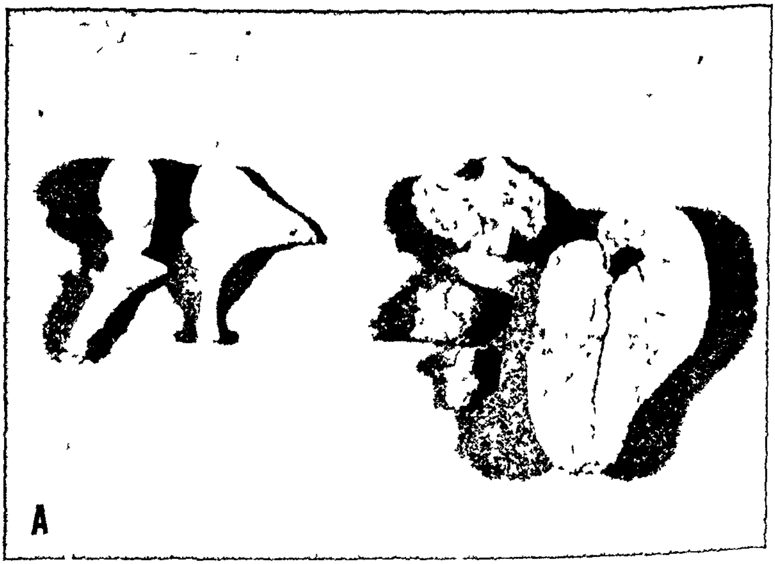


Fig 1-a Hyalinized collagen (tympanosclerosis) presenting as a large mass lying free in the middle ear Normal ossicles from another patient are shown for size comparison

B Deficient Middle Ear Mucosa

Examination under magnification (at least six power) is essential in preoperative evaluation for tympanoplasty, particularly for determining the status of the middle ear mucosa The mucosal surface area must be sufficient to provide an adequate cavum-minor Reconstructive surgery will usually fail in its objective of improving hearing if there is any destruction of mucosa in the area between the Eustachian tube and the round window Operations to graft mucosa or to maintain the size of the cavum-minor with plastic prostheses are not yet proven to be successful bleeding must be absolutely controlled, and that the graft must be approximated to its bed by packing which is firm enough to prevent collections of underlying fluid

Neither cautery nor the polishing burr should be used on the recipient site The polishing burr seems to plug vascular channels and devitalize bone by its heating effect Crushing or puncturing the graft with sharp instruments causes areas of necrosis as well as possible implantation of epidermis into the deeper layers of the graft which promotes the development of epidermoid inclusion cysts and perforations

Central graft necrosis can result when an attempt is made to bridge a large tympanic space Ideally no part of the graft should be more than 2 or 3 mm from a viable tissue bed, thus, spaces 4 to 6 mm wide usually can be bridged successfully When the entire tympanic membrane is missing and the malleus handle is missing or avascular, the central area of the graft should be placed in contact with an area of denuded promontory to ensure adequate nutrition

Chronically discharging ears should be treated before surgery to acquire a state of remission When suppuration cannot be completely controlled, it may be necessary to proceed with surgery even though the chances for success are somewhat less than for the "dry" ear.

B Late Failures (Perforation)

In our experience late graft failures are caused by either circumscribed cellulitis or epidermoid inclusion cysts

The occurrence of circumscribed cellulitis as a late graft complication is not surprising when one realizes that continuing suppuration can occur on both sides of the graft

We have examined histologically a series of 20 tympanoplasty grafts having areas of circumscribed cellulitis and/or perforations removed six months to two years after implantation All were full-thickness skin taken from the postauricular sulcus and thinned with sharp scissors

The areas of circumscribed cellulitis appeared grossly as discrete, smooth, reddish areas measuring 1 to 2 mm in size

D Age Over 60

Tympanoplasty surgery does not seem indicated in patients over 60 for improvement of hearing or for elimination of discharge from a "safe" ear Our reasons are threefold first, there is a greater incidence of graft necroses in the older patient (probably because of vascular insufficiency in the recipient site), secondly, the incidence of pulmonary, cardiac and cerebrovascular complications from surgery are greater in the aged, and thirdly, the value received for effort is less because of shorter life expectancy

Tympanoplasty may be performed for the healthy patient over 60 when middle ear or mastoid surgery is required to eliminate dangerous pathology

II CAUSES OF TYMPANIC GRAFT FAILURE

One of the most common causes for tympanoplasty failure is loss of graft substance, a complication which may occur soon or many months after surgery Our experiences correspond closely with those of Berckert,[3] Kley,[4] and Hohmann, et al [5]

Full-thickness grafts from the post-auricular area were used in 219 patients between January, 1957, and October, 1959 To this date, perforations have been found in 48 (22 per cent) A second operation now has been done on 22 of the 48 failures using split-thickness skin graft It is too early to assess the results of these revisions

A Early Failures (Necrosis)

Total necrosis of the free tympanic graft is rare Partial necrosis, which is more common, results in perforation which almost never closes spontaneously Early failure may be due to improper packing, trauma, inadequate blood supply, and infection

There is still a difference of opinion as to whether a graft should be packed into place firmly or loosely Certainly the principle is firmly established that the graft must be meticulously tailored to fit the contours of the recipient site, that and were characterized by an accumulation of chronic inflammatory cells in the dermal layer (see Fig 2) The overlying epidermis atrophies so that the rete pegs and papillae disappear Further thinning of the epidermis results in ulceration, and finally perforation Perforations occurring on the basis of cellulitis are often lined with mucosa

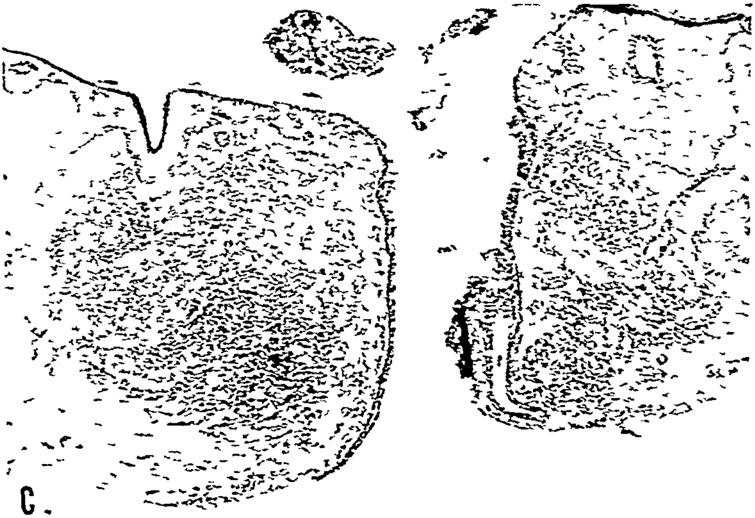


Fig 2-c Cellulitis and perforation with mucosal lining (full-thickness graft removed 26 months after operation)

A more common type of perforation is caused by formation of an epithelial inclusion cyst within the graft (graft cholesteatoma). A majority of these cysts probably arises from skin appendages (hair follicles and glands) In the process of thinning a graft it is possible to expose or cut the papillary ends of hair follicles which may result in perforation, or squamous epithelialization of the tympanic surface (see Fig 3)

Other causes for inclusion cysts are infolding of epidermis due to improper positioning of the graft, and puncturing, crushing or otherwise traumatizing the graft so as to bury epidermis into its deeper layers[6]

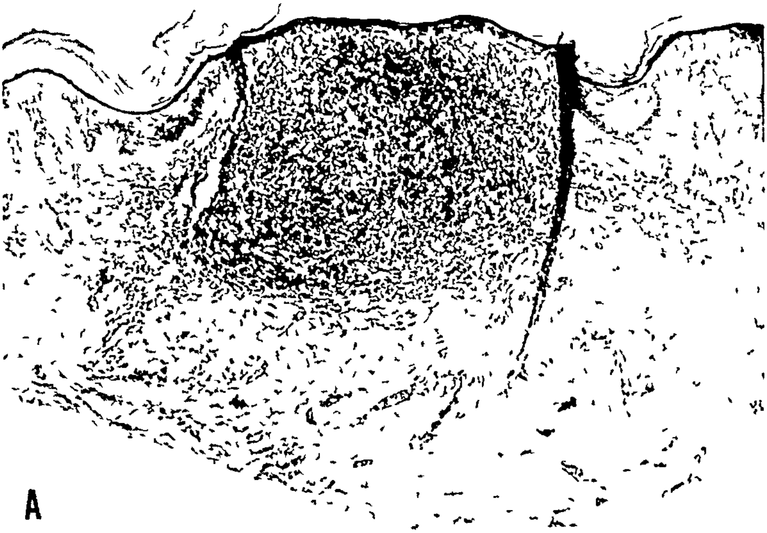


Fig 2-a Full thickness tympanoplasty grafts showing how perforation develops from circumscribed cellulitis a—Circumscribed cellulitis (full-thickness graft removed 18 months after operation)

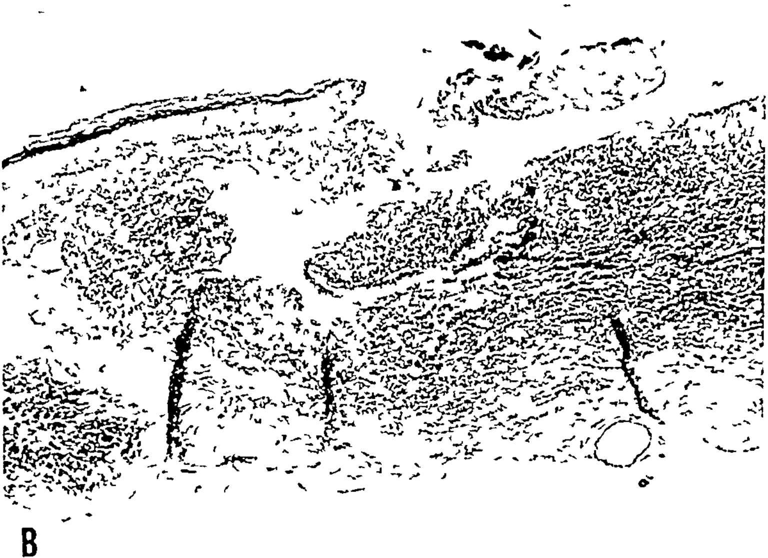
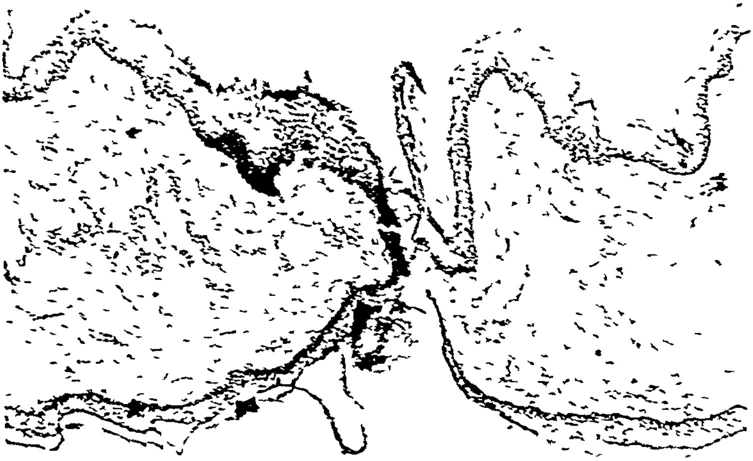


Fig 2-b Cellulitis with ulceration (full-thickness graft removed four months after operation)

We have observed the occurrence of inclusion cysts two years after an initially successful operation

The mucosal lining on the tympanic surface of the grafts consists predominantly of a fragile transitional-like epithelium which is two or three cells in thickness In the anterior mesotympanum the lining is sometimes of the pseudostratified columnar ciliated type (see Fig 4)



C

Fig 3-c Small perforation with squamous epithelial lining (removed 11 months after operation)

C Current Grafting Methods

For defects of the membrana propria which involve up to 25 per cent of the total area we now use the vein graft according to the method of Shea⁻ For defects involving from 25 per cent to 75 per cent of the membrana propria we follow the suggestion of Dr William House,[8] using full-thickness skin from the anterior wall of the auditory canal (also advocated by Plester[9]) This appears to be excellent graft tissue because it is free of hair and contains few glands These skin grafts are not large enough ordinarily to close complete drum de-

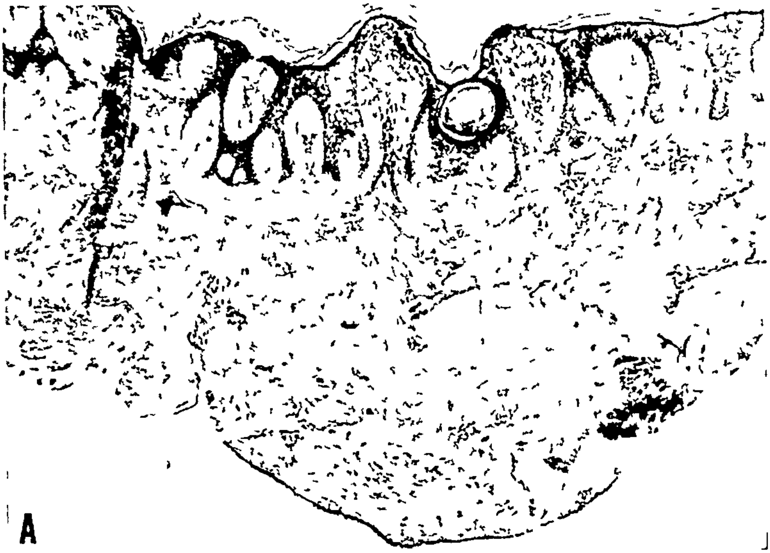


Fig 3-a Full thickness tympanoplasty grafts showing how perforation develops from epidermoid inclusion cysts a—Small inclusion cyst in the epidermis (full-thickness graft removed 18 months after operation)

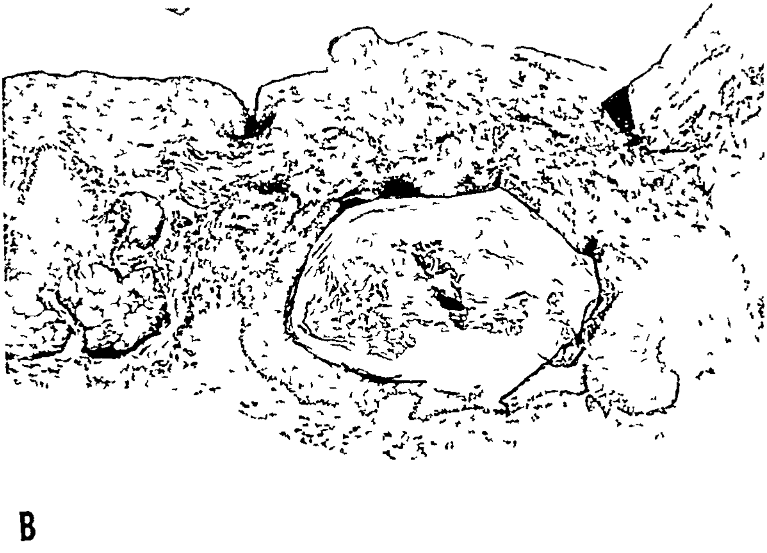


Fig 3-b Inclusion cyst deep within the wall of the full-thickness graft (Removed 18 months after operation)

fects. For reconstructions requiring larger grafts, we now use split-thickness skin (.018 inch) taken from an area with the least hair. The donor site is selected from the arm, chest, or thigh with the aid of magnification.

CONCLUSIONS

Reconstructive middle ear surgery is usually unsuccessful in the presence of the Eustachian tube malfunction, hyalinized collagen deposits, and deficient mucous membrane; ordinarily it should not be performed for patients over 60 years of age.

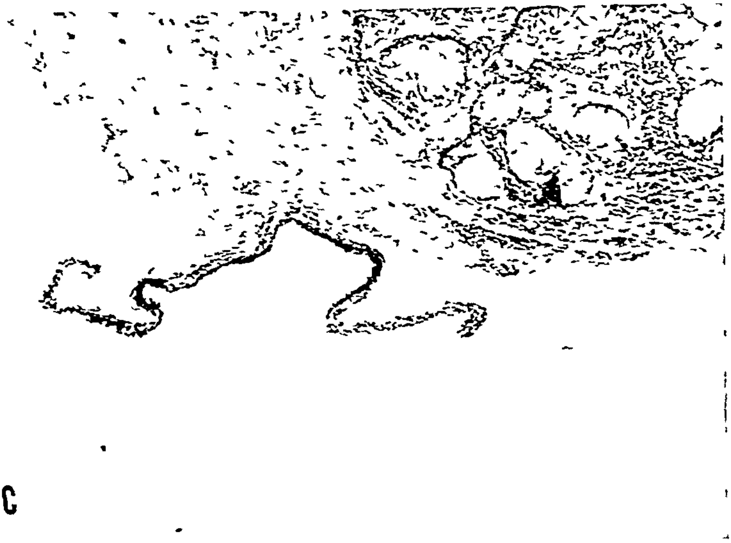


Fig. 4-c. The mucosal lining of tympanoplasty grafts is very fragile and can be lost in preparation. The absence of a mucosal lining on histological sections should not be misinterpreted to mean it was not originally present.

Skin graft failures can occur immediately, or many months after surgery. Graft failures can be decreased by the proper selection of the donor site, careful handling and approximation of the graft to the recipient site, use of cutting burrs only, and by controlling suppuration.

Tympanoplasty will achieve and maintain a position of

Fig 4-a Mucosal regeneration on tympanic surface of grafts a—Transitional-cell type epithelium on graft removed 18 months after surgery

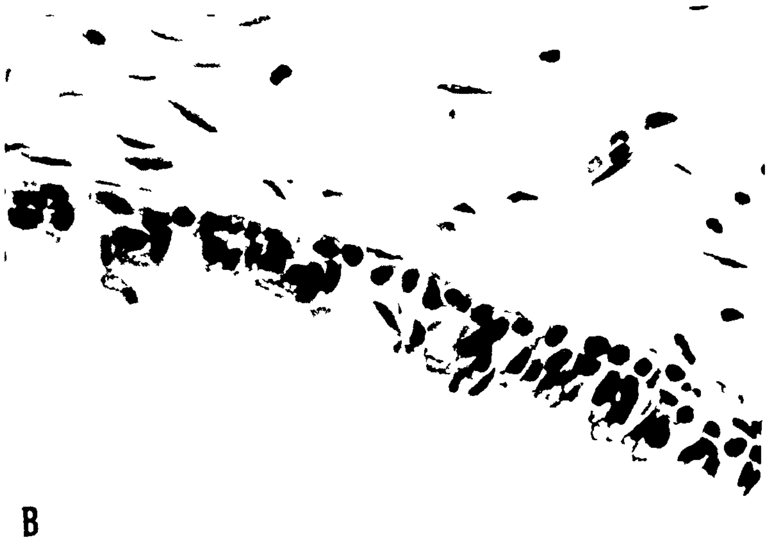


Fig 4-b Pseudostratified columnar ciliated epithelium on graft removed five months after surgery

The true physician is no soothsayer, but rather one who opens the curtains and windows of the sick room to let in the light of wisdom and the fresh air of common sense.

Authorities who have given the most attention to prognosis dwell perpetually upon decorum and dignity. We learn from their works that the establishment of a proper regime, abandonment of past errors and expectant treatment are the altars upon which "recent advances" must be laid and from which all will soon be discarded that is of no permanent value.

In surgery, the decision to operate is still comparable with an act of war, though many operations formerly attended by high mortality may now be undertaken in safety; having been trained primarily in general surgery, experience teaches how, at the onset of an operation, to identify vulnerable nerves and to ligate arteries which supply redundant tissues, thereby preserving them. The simplicity of an operation has always been the measure of its perfection. The unexpected and the uncomprehended are our main adversaries.

If, from time to time, we are—so to speak—taken into the confidence of the Almighty, in the exercise of our profession, we should be careful how we impart our knowledge to the patient. Humanity must be observed and preserved.

The attitude of otolaryngologists to our specialty should be one of gratitude that antibiotics have made safer the many new procedures in this field which are now so commonplace.

The essayist, in a paper read before the Canadian Otolaryngological Society, June, 1957, had this to say: "Many of us might consider that we are living in one of the most progressive eras of our specialty, but let us approach any medical subject with humility, for, knowledge is immense, and the spirit of man can extend infinitely to enrich itself daily with new requirements. Many of the accepted facts of today may in time become the errors of yesterday. Especially is this true of the function of hearing, and the 'resurrection' of certain so-called new procedures within the middle ear."

The hopes of a great many deafened people have been respect among otological procedures only if we, as surgeons, properly assess the underlying surgical pathology and achieve appropriate microsurgical skills

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Dr Furstenberg I now have the privilege and the honor of calling upon Dr Joseph A Sullivan

TYMPANOPLASTY *†

Joseph A Sullivan, M B, F R C S (C),

Toronto, Canada

Accurate foreknowledge elevates the art of medicine into a science and forges its diverse elements into a glowing link between the immortal achievements of the past and the infinite prospects of the future

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*Read at the Sixty-Third Annual Meeting of the American Laryngological Rhinological and Otological Society Inc Miami Beach, Fla March 15 1960

†From the Hard of Hearing Clinic and Vestibular Clinic St Michael s Hospital Toronto and the Department of Otolaryngology the University of Toronto Canada

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destructive lesions are in reality a hypersensitiveness of the tissue to bacterial proteins

One should remember that redness of the presenting tissue does not exclude allergy, and this process also explains in part the active response of tissue to an acute exacerbation in a chronic ear The initial lesion in the body may be insignificant and far removed, carried by the blood stream and producing an ID reaction in the ear Control of the nasal condition by discovery and elimination of the allergy factors may result in a cessation of the aural discharge, and the perforation may then be closed

In 1938 Lempert described the one-stage fenestration operation with the viable tympano meatal flap which he had previously used to seal a labyrinthine fistula in the performance of an endaural mastoidatticotomy for chronic epitympanic suppuration This was one of the greatest advancements in temporal bone surgery and from it numerous modifications evolved

Following this successful fenestration surgery, otologists again became aware of surgery as a means to improve hearing From that period on, numerous articles appeared in the literature on the Bondy type conservative mastoidectomy, thus the preservation of hearing in surgery for chronic ear disease was made possible by the techniques, the magnification and the illumination which were originally employed in the operative treatment of otosclerosis

With the invaluable aid of chemotherapeutic and antibiotic drugs, associated with a better understanding of skin grafting techniques and increased knowledge of the physiology and pathology of the ear, otologists now strive to eradicate the pathology and maintain or restore the function of the diseased middle ear In other words, the essence of these new techniques is the direct application of the fundamentals of adequate temporal bone surgery superimposed with the principles of plastic surgery to otology

Otologists for years have been aware of the necessity for reconstructive surgery in chronic ear disease We have a raised by the development in recent years of three operative procedures for certain types of middle ear deafness[1]

1. Fenestration

2. Stapes mobilization

3. Tympanoplasty

In another paper written over 15 years ago, the author commented as follows[2]

"The treatment of the chronically discharging middle ear depends on an understanding of the histological and physiological problems concerned It is our constant endeavor with our present knowledge and the application of the newer antibiotics in otologic treatment to prevent a radical procedure if at all possible The successul interpretation and treatment of chronic middle ear disease requires a clear understanding in each case of the etiology and of the pathology responsible for the chronicity The aims of treatment are

1 To eliminate the danger of intracranial complications

2 To obtain a dry ear

3 To preserve, and if possible, to improve the hearing"

I went on further to say "Some form of meticulous surgery with magnification in the middle ear will give the best chance of preserving hearing, but unfortunately the hearing picture in mastoid surgery for many years seems to have been of secondary importance Great care is exercised in removing granulations and diseased tissue from the area of the oval and round windows This surgery is followed by placing skin grafts in the middle ear on a mobile stapes and in the mastoid cavity If granulations can be prevented from forming in the above areas and subsequent scarring avoided, it is hoped that the round and oval windows will continue to function and the hearing be maintained or improved"

I mention one type of chronic otitis purposely, namely the allergic type[3] Allergy is a term often used to cover ignorance or give a touch of mystification to the process, yet many destructive lesions are in reality a hypersensitiveness of the tissue to bacterial proteins

One should remember that redness of the presenting tissue does not exclude allergy, and this process also explains in part the active response of tissue to an acute exacerbation in a chronic ear The initial lesion in the body may be insignificant and far removed, carried by the blood stream and producing an ID reaction in the ear Control of the nasal condition by discovery and elimination of the allergy factors may result in a cessation of the aural discharge, and the perforation may then be closed

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Otologists for years have been aware of the necessity for reconstructive surgery in chronic ear disease We have a number of patients operated by the complete radical procedure who have maintained or gained a useful level of hearing Following various forms of modified radical procedures we have had numerous excellent results in maintaining or restoring hearing, however, these results were obtained without a clear understanding of middle ear physiology We failed to apply the principles of the middle ear sound pressure transformer which was clearly described by Helmholtz in 1863 Finally it remained for Zollner[4] in 1951 and Wullstein[5] in 1952 to apply the mechanical principles of the middle ear transformer to the surgical reconstruction of the middle ear following destructive middle ear disease

Chronic otitis media causes pathological and functional defects of varying degrees, hence the method to restore the hearing must vary Stacke, writing in 1897 said, "No one will bind himself to one method in operating, rather will every thoughtful operator individualize and modify the method from case to case according to the particular pathological condition "

All these variations in technique, however, must be governed primarily by striving to eradicate the disease completely and second, to establishing a sound conducting apparatus

What do we mean by the terminology "tympanoplasty"? Well, it does not mean what it is supposed to mean, namely—a combination of surgery for eradication of chronic ear inflammation and surgical reconstruction of the sound conducting and sound protecting mechanism Rarely can it accomplish both of these requisites at one operation

We owe a great debt to Wullstein, who has classified reconstruction of the sound conducting apparatus into five types

Type I—Rebuilds a normal ear—tympanic membrane with both functions sound protection for the round window and the scala tympani, and sound pressure tranformation over the ossicular chain to the scala vestibuli

Type II—Tries to preserve, in spite of slight defects of the ossicles, a middle ear of about normal size and depth between tympanic membrane and promontory

Type III—In this type, large defects of the malleus and incus make the removal of the ossicular chain and the epitympanum necessary The tympanic membrane, in order to function as a sound pressure transformer, must be directly connected with the head of the stapes This has been called, Columella-Effect by Zollner and Myringostapediopexy by Juers This was originally done by Matte in 1901 This is a comparable problem that we are often called upon to deal with in the most difficult of all otologic surgical procedures, the repair of the congenitally atresic external ear canal and maldeveloped mastoid bone and middle ear remnants [6]

Type IV—In this type, the stapedial footplate is movable, but the crura are missing It is impossible to obtain sound pressure transformation The oval window is left open to the auditory meatus The tympanoplasty reconstructs a middle ear consisting only of the tube and hypotympanum with sound protection for the round window

Type V—In this type, the stapedial footplate is fixed, and a fenestration of the horizontal canal is required, however, it is necessary to protect the round window

The post auricular incision is preferred by some physicians, particularly when a post auricular skin graft is employed The post auricular graft can be obtained from the same incision, we prefer the skin of the forearm or the anterior canal wall as I will describe later on

I believe that the results in many cases do not justify this tedious meticulous surgery, because the hearing improvement is not predictable It is true the ears can be tested to determine the cochlea function, but you cannot test in an accurate manner how the middle ear will function after the procedure is completed In simple perforations with the ossicular chain intact wherein a paper patch restores hearing to a high degree, then of course, one could expect a similar result by tympanoplasty, but the hearing must be good to begin with to obtain any kind of satisfactory result True, once in a while you may get a spectacular result, but this type of improvement cannot be obtained constantly

I am for any kind of meticulous surgery which cleans the disease from the most remote areas heretofore neglected As one who has used the dissecting microscope, not just a loupe, from my early work in the facial nerve, beginning nearly 30 years ago until the present day, I am fully cognizant of this fundamental prerequisite Bekesy, Wever and Lawrence have clarified and established the psychoacoustic principles which are involved in obtaining an air space in the region of the round window If the stapes is not movable then, of course, creating an air space in the round window is useless Then comes the question of whether to fenestrate if the stapes is fixed Logic postulates that if an artificial membrane can be created in the middle ear with an air space in the region of the round window and a mobile membrane created over the fenestra, then there should be some improvement of hearing

My associate, Dr Kenneth McAskile,[7] in a paper read before the Canadian Otolaryngological Society in 1959, reported a method of repairing a large marginal tympanic perforation using a pedicle flap which can be rotated either from the floor or from the anterior wall of the external canal

It is well known that the pedicle flaps have been used for years to close defects in tympanic membranes and that at the present time most authors prefer free skin grafts to pedicle flaps because of the ease of accurate placement and exact adaptation to this defect, yet as with all skin grafts, the results of the operation should be assessed only after the lapse of many months

Experience has shown us, however, that the tympanomeatal flap as used in the fenestration operation will endure infection and trauma while free skin grafts used to cover raw areas in the same cavity will break down or slough out completely, sometimes several years after the original operation

In modified radical mastoidectomies with epitympanic perforations or with small perforations in the pars tensa it is relatively easy to obliterate the defect by freshening the edges of the perforation and then approximating the margins It is sometimes necessary to cut down through the tympanomeatal flap to the perforation in order to obtain good approximation In these cases, as in the fenestration, the tympanomeatal flap is usually rotated in an anti-clockwise direction, the skin from the anterior canal wall being used to obliterate the epitympanic space

In closing a large marginal defect with a tympanomeatal flap a larger portion of the skin of the anterior wall is included down to the tympanic annulus and more anteroinferiorly than is usual A bed for the pedicle flap is prepared similar to that used in a myringoplasty or tympanoplasty by denuding the epithelium from the remnants of the tympanic membrane and the adjacent canal wall The tympanomeatal flap is now rotated in a clock-wise direction, and the skin of the anterior canal wall easily closes the tympanic defect The flap is then held in position by shaped cellulose sponge impregnated with an antibiotic solution or ointment This procedure has the distinct advantage over a free skin graft in that it has an adequate blood supply of its own and also that it is tissue native to the area concerned (see Figs 1-a and 1-b)

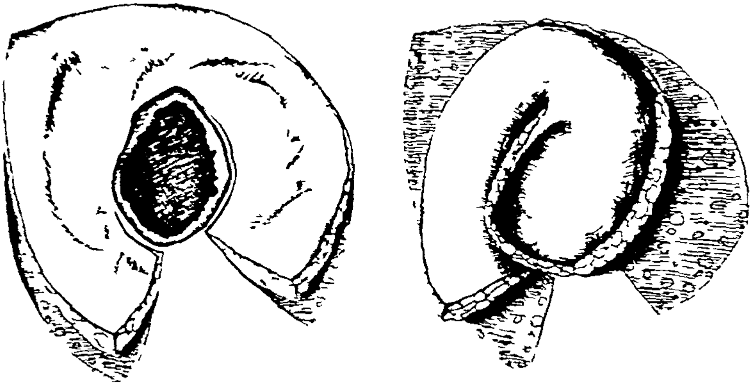

FIG a FIG b

Fig 1

We have used this procedure in eight cases of total perforation and in only one case is there a remaining perforation Unfortunately the perforation in this case is located over the round window which is not compatible with good hearing A small piece of gelfoam inserted in this defect restores the hearing to the 20 decibel level Conservative attempts to close this perforation have failed, so it will be necessary to use a free skin graft for closure

The use of artificial prostheses in the middle ear was advocated by Wullstein and Zollner to replace the crura of the stapes where a mobile footplate existed, however, tissue reaction to the acrylic and vinyl plastics forced them to abandon this material

House, Schuknecht and Shambaugh have reported the employment of either polyethylene tubing or tantalum wire to replace diseased or absent components of the conducting apparatus in the middle ear As yet they have not noticed any local reaction to these materials We have had occasion to use either tantalum wire or polyethylene tubing on a number of occasions and our observations have been similar to those stated above, not good

1 Reconstructive aural surgery as a term may suggest a certain uniformity in the operation, it is different in every case, not only in details but even in the main lines

2 We have learned that in cases of a disrupted ossicular chain, our results are much better, insofar as getting a dry ear is concerned, and also regarding final hearing, when we take away the mutilated incus and malleus and put the drum or a skin graft pushed in upon the head of the stapes, naturally, the bridge is removed In other words when the ossicular chain is not intact, we do not try to put malformed ossicles together again We prefer the apposition of the drum (with or without a graft to enlarge the effective size of the membrane) upon the head of the stapes Sometimes amniotic membrane can be placed under the drum, to prevent its adherence to the promontory

We believe that in unilateral inflammation, fenestration should be avoided

We believe in the two-stage operation because the most difficult problem is to heal the original disease

Lindsay states that operations in chronic middle ear infections present a more serious problem than fenestration Individual adaptation of the operation and diagnosis require great versatility, and the final result places heavy demands on the patience and endurance of surgeon and patient

To summarize

1 It is impossible, without exploration of the middle ear under magnification to make a prognosis as to functional improvement that may be gained by a tympanoplasty If there is gross scarring of the window regions an improvement of function is unlikely to take place

If the disease is minimal the defect in the drum is repaired with a full thickness graft, if it is necessary to remove the bridge a good functional result may occur in some cases providing that the incus is left with its covering of mucosa

2 If there is gross mastoid disease with interruption of the ossicular chain

a A most meticulous operative toilet with removal of all disease is first carried out under magnification in all cases

b Adequate toilet of the window regions is carried out

c The tympanic reconstruction is not performed until one has an ear that is fit for it In most cases it is not performed until some weeks later

d In regards to the mastoid cavity, the post auricular flap technique of Hynes and Beales is a distinct contribution

e There must be a functioning Eustachian tube

McAskile in closing his paper states—in reviewing numerous articles on this type of surgery it is interesting to note that the majority of authors put great emphasis on the functional results that have been obtained but very little time or space is devoted to reporting the presence or absence of discharge from the operated ears The main danger in tympanoplasty comes from burying infection of cholesteatoma under the skin The primary concern in chronic ear surgery is complete eradication of every bit of pathology If this principle is sacrificed in the hope of saving or restoring hearing we have failed in our duty to the patient

In conclusion, the real goal is prevention to attain this objective, we must increase our basic knowledge of hearing difficulty and be able to diagnose accurately reversible and irreversible pathology of the middle ear Meanwhile, these plastic procedures on the middle ear are reclaiming enough hearing to give real hope for the future

"There are many arts among men, the knowledge of which is acquired bit by bit by experience that causes our lives to move forward by the skill we acquire, while want of experience subjects us to the effects of chance"

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Dr Furstenberg It is my pleasure now to call upon a distinguished guest who because of his significant contributions to otology is worthy of our highest praise Dr Nils G Richtner of Stockholm

RECONSTRUCTIVE MICRO SURGERY OF THE EAR, ESPECIALLY WITH THE CAVUM MINOR TECHNIQUE *†

NILS G RICHTNER, M D ,

Stockholm, Sweden

In the last 20 years there has been great progress in surgery of the ear, resulting especially from the introduction of the microscope in ear surgery Its use was initiated by the Swedish professor Nylén and subsequently carried on by Gunnar Holmgren from 1930-1945, who was particularly interested in surgery of otosclerosis In this branch of surgery we have no infection of the ear In contrast we have the chronic otitis giving rise to impaired hearing For many years this has also presented a great problem to all ear surgeons leading to new operations in an effort to determine what will produce the best results in hearing and healing Earlier one operated primarily to obtain a dry ear and gave little thought to the patient's hearing, but since the inception of the audiometric era, starting with Fowler, Sr , the present aim of surgery is to obtain not only dry, healed ears after operation but also to give the patient better hearing

Plastic surgery of the middle ear has gained tremendous ground in otology especially through the outstanding work of Wullstein and Zollner The patient with chronic middle ear inflammation, frequently with greatly impaired hearing, can now be relieved of his infection, of the false tumors or so called cholesteatomas frequently accompanying these forms of ear inflammation, and also enjoy a certain degree of improvement in his hearing

Thanks to the advances made by these eminent workers in otologic surgery this has become possible not only through radical operation on the cellular system of the ear as per-

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*Read at the Sixty-Third Annual Meeting American Laryngological Rhinological and Otological Society Inc Miami Beach Fla March 15 1960

†From the Ear Nose and Throat Department Sabbatsberg Hospital Stockholm

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In conclusion, the real goal is prevention to attain this objective, we must increase our basic knowledge of hearing difficulty and be able to diagnose accurately reversible and irreversible pathology of the middle ear Meanwhile, these plastic procedures on the middle ear are reclaiming enough hearing to give real hope for the future

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8 BEALES, P H, and HYNES, W Rapid Healing After Mastoid Surgery by the Use of the Post Auricular Flap *Jour Laryngol and Otol*, 72 888, 1958

DR FURSTENBERG It is my pleasure now to call upon a distinguished guest who because of his significant contributions to otology is worthy of our highest praise Dr Nils G Richtner of Stockholm inflammation is very extensive and acute, in two stages Some surgeons do not undertake this operation until the ear is completely dry for many months, others report that they operate on patients with very severe infections and do the tympanoplasty in the same stage or a few months after the radical operation cavity has been cleaned

There have been many discussions during recent years concerning what should be done about the large radical operation cavity There are many views on the healing, and many surgeons contend that they do not obtain as good healing of the free full-thickness skin graft or split skin graft placed over the radical cavity They state that skin placed over the bone does not heal and grow as well as in the tympanic cavity where there is an abundance of vessels Sometimes the ear does not heal for many months after the radical operation, and it is very difficult for the ear surgeon to treat these patients, therefore some surgeons have discussed and in some cases have also tried to fill the operation cavity with a pedicle flap from muscles (Guilford and Rambo) Others prefer a pedicle skin flap taken from behind the ear and placed down in the operation cavity, which gives much better results and good healing after one or two months (Beales) Beales says that with a full-thickness skin graft not all cavities healed after six months, *i e*, not more than 30-40 per cent, while with the pedicle flap of skin behind the ear he obtains much better results These series are not yet extensive enough to permit discussion of the percentage

An effort must be made to close the operation cavity primarily, and I like to use the Guilford and Rambo technique to fill the operation cavity and to cover the horizontal semicircular canal with muscle When the ear in these cases heals and the perforation has also healed with a skin graft, the ear is wholly normal and the patient is able to swim, for instance, without any danger to the ear

Earlier I used a free full-thickness skin graft taken from behind the auricle in the same incision as the retroauricular one, but free skin grafts placed in the mastoid cavity and in the middle ear sometimes slough due to the lack of blood formed earlier but also through plastic operation on the elements of the middle ear, the mucosa, the auditory ossicles and the tympanum, resulting, in many cases, in astonishing improvement of the hearing. The operation of chronic otitis and mastoiditis with reconstruction of the ossicles and covering of the drum with a skin graft is called tympanoplastic surgery.

Sometimes we also have the so-called myringoplastic surgery in which an attempt is made at plastic closure of a greater or lesser perforation of the tympanon peraurally without disturbance of the ossicles. The indications for this approach are limited, however, and it must be done with great caution. The perforation of the drum must be centrally situated and must not involve the limbus of the tympanic membrane in any way. The ear shall have been dry for several months and no X-ray changes shall be present in the cellular system of the middle ear indicating inflammation there. The preoperative audiologic test, the transmission investigation by means of a vibrating probe and the prosthesis test shall give definite evidence of satisfactory function in the ossicle chain, and the prosthesis test must show appreciable improvement in the hearing. In cases of small perforations with a dry ear we can obtain good results, but in cases presenting large perforations the results are generally not so satisfactory.

As a transplant a meatal flap from the inner part of the external canal can be used or a free, full-thickness skin graft can be taken from behind the ear or, as has been done in some cases, mucosa from the mouth (Hall) or a vein graft as used by Shea, Jr., may be employed.

The rest of the plastic operations of the middle ear are included under the common name of tympanoplasty, and include the radical approach, either peraurally or retro-auricularly, with operation on the cellular system of the ear and creation of different types of middle ear cavities *ad modum* Wullstein and Zollner. These operations are suitable for all forms of chronic middle ear inflammation and mastoiditis, and may be performed in a single-stage operation or, if the

the incision and the skin flap and skin-muscle flap We then do the radical operation in the conventional manner, radical operation or a modified radical operation where the bridge is left *in situ* With the pedicle skin flap it is then easy to make a cavum major or a tympanostapediopexy or a small cavity as shown in Figs 2 and 3 and then to fill the radical cavity with the skin-muscle flap A part of the pedicle flap must then be excoriated as you see in Fig 3, and the posterior part of the meatus sewn to periosteum behind the skin-muscle flap

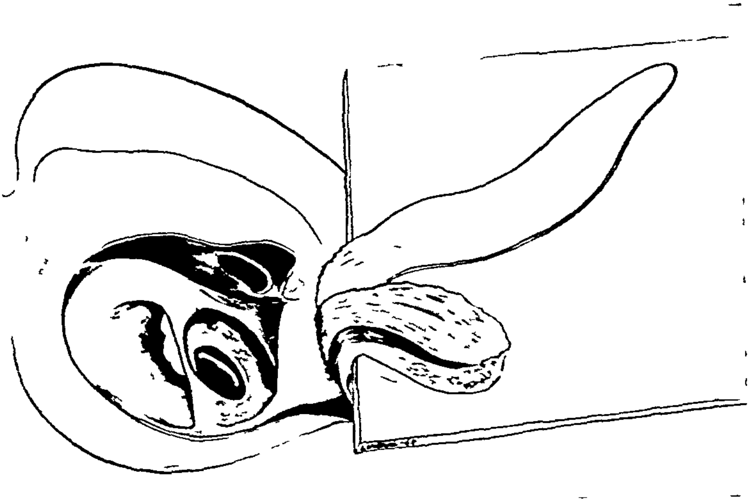


Fig 2

At Sabbatsberg Hospital, Stockholm, all these operations are performed under general narcosis (anesthesia with Pentotal and intratracheal intubation) A disadvantage of this procedure is that we can not test the patient's hearing during the operation, but it is very difficult for the patient to lie on the operating table the two or three hours or more that the operation requires

All these ear operations are preceded by a clinical study of the middle ear, frequently under microscope, for evaluation of the appearance and the type of perforation and investigation of the auditory ossicles Special measurements of the supply, therefore some time ago I started working with combined skin and muscle pedicle flaps and also with the retroauricular incision, and I shall see if this method will allow the radical cavity to heal in a shorter time and maintain the flap in good condition The pedicle flap will cover the middle ear cavity and will give a larger middle ear cavity, the combined skin-muscle flap will fill a great part of the mastoid cavity thus giving us a small meatus down to the new drum and very little cavity and in some cases no cavity

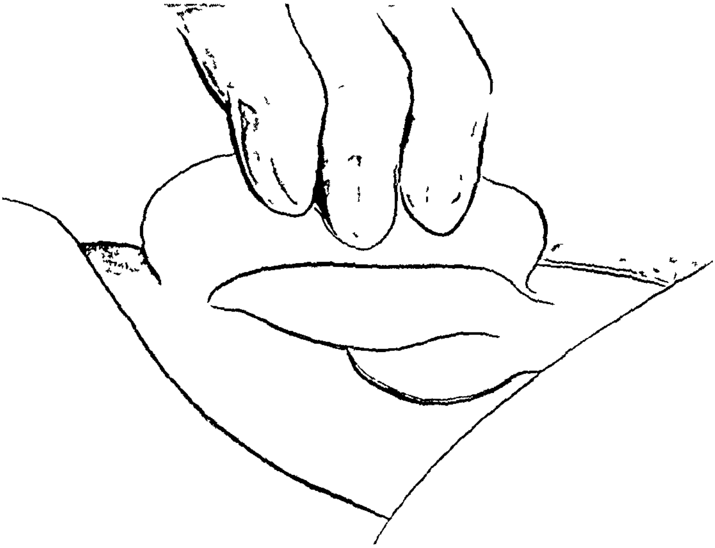


Fig 1

at all This operation is especially recommended for chronic otitis with no suppuration, chronic otitis with a cholesteatoma, etc In the presence of suppurating chronical otitis I prefer first to do the radical operation and some weeks later, when the infection is gone, to perform the plastic operation My material is still too limited to allow any definite conclusions about the healing and hearing after this new pedicle flap operation, but it seems to be a good method and in the cases I have operated the hearing is good I will show you some drawings from this form of tympanoplasty Fig 1 shows the long process comes into contact with the stapes In some cases one can fix the lenticular process of the incus to the stapes with an extremely fine steel wire or with catgut, but we have seen that merely placing the lenticular process in contact with the head of the stapes resulted after some weeks in formation of fibrous tissues giving a good contact

Transposition can also be done between the malleus, the long process of malleus, and to the stapes directly as with a columella from the long process of incus down to the stapes as has been shown by Shambaugh We can use a small polyethylene tube on the long process of the incus and down to the stapes, in fact, many variations can be used, and it is impossible before a tympanoplastic operation to know exactly what is to be done The decision must be made during the operation

Sometimes we use type III *ad modum* Wullstein, the so called tympanostapediopexy, or, according to German nomenclature, the formation of "eine flache Pauke" The primary result of this type of operation has been good, with respect to healing as well as to hearing, but the late results have been less satisfactory in some cases due frequently to the occurrence of a perforation in the flap over the stapes

In the cases presenting a pronounced defect in the ossicle chain I have employed Wullstein Type IV, the so-called "eine kleine Pauke" or *cavum minor* technique In many cases this has afforded astonishingly good results even in cases of extremely grave auditory impairment Many encouraging results were obtained with this technique even in the early days, five years ago, when I started seriously to engage in tympanoplastic surgery Stimulated by the excellent results I have, therefore, been particularly interested in undertaking treatment of the extremely grave cases of auditory impairment in chronic otitis in order to see what results could be obtained with this special technique About 200 tympanoplastic operations have been performed with not less than about 80 patients operated on according to the *cavum minor* technique, with minor variations from case to case, but with the consistent purpose of establishing a difference in pressure be-hearing are always carried out primarily with an ordinary pure tone audiogram and sometimes complemented with a speech audiogram The so-called transmission investigations are included in these studies, especially important are the vibrating probe and the prosthesis test, both the so-called complete prosthesis and the covering prosthesis tests The latter involves primarily a screening prosthesis test over the round window

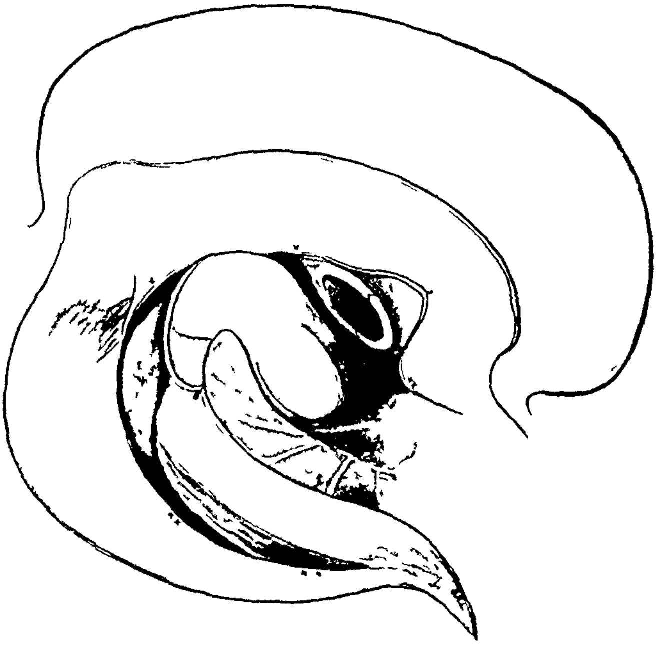


Fig 3

In chronic middle ear inflammation where we have chronic mastoiditis, osteitis or cholesteatoma of lesser extent, that can be removed without injuring the auditory ossicles or tympanic membrane, we do the *cavum major*, the large cavity

In a case presenting minor destruction of only the long process of the incus we try transposition of the incus so that

An incision is then made (see Fig 4) in the mucous membrane of the medial wall of the middle ear below the prominence of the facial nerve canal and forward over the Eustachian tube above the facial prominence so that a narrow flap is obtained there to fold up against the tube The rest of the mucous membrane is turned downward, the epithelium forming a bed for the transplant

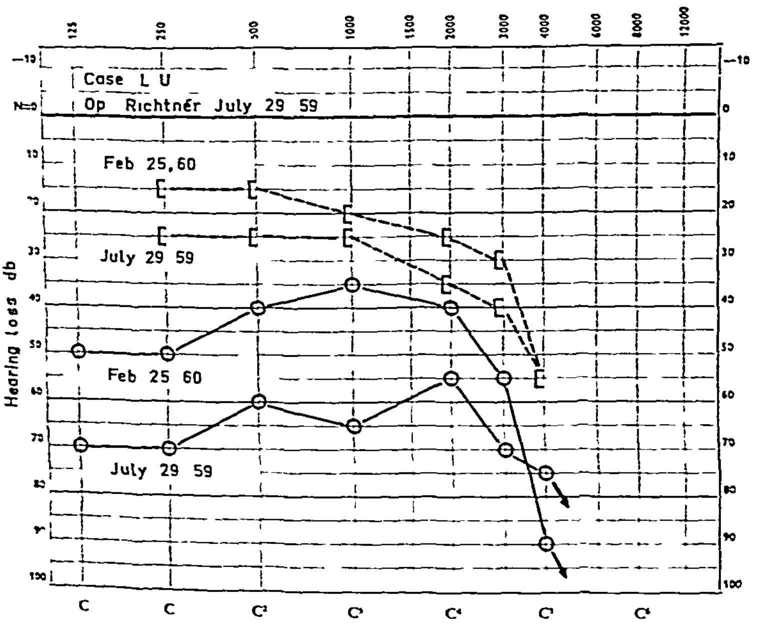


Fig 5

At operation it is generally found that a part of the limbus is still present This should be treated with the greatest care as it provides an excellent bed for the transplant which is obtained retro-auricularly in all cases in conjunction with the application of the retro-auricular incision The residues of the drum and of the inner part of the external auditory canal are meticulously denuded of epithelium The transplant is suspending on the inner part of the external auditory canal and the limbus If none of the limbus remains a little shelf is tween the round and oval windows, consequently it is this type of operation I should like to discuss in detail now, particularly with respect to technique and the auditory results, which are frequently so good that it seems difficult to explain them from physiologic point of view.

It is true in this case as in all other plastic operations on the middle ear that the Eustachian tube should be patent, permitting easy passage of insufflated air The operative procedure is largely as described by Wullstein and Zollner First the radical operation is performed with thorough removal of polyps and granulations about the residual stapes or the footplate of the stapes and the round window and the tube without damaging the epithelium more than necessary

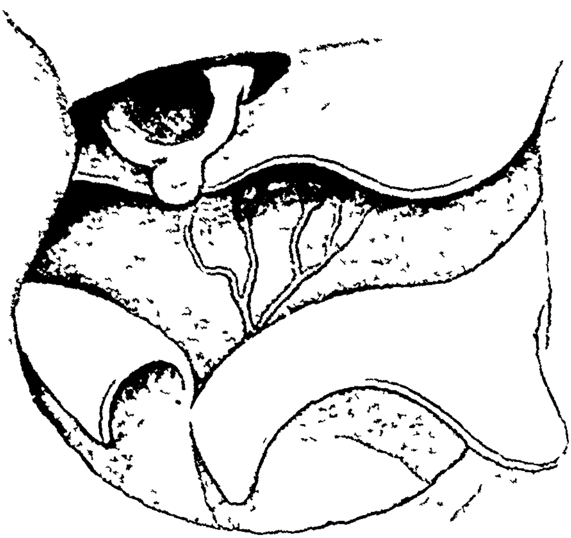
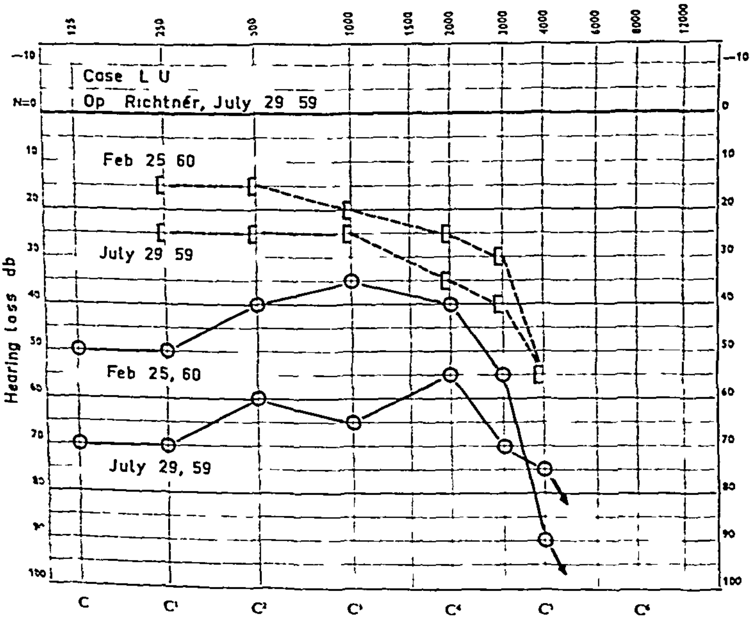


Fig 4

An incision is then made (see Fig 4) in the mucous membrane of the medial wall of the middle ear below the prominence of the facial nerve canal and forward over the Eustachian tube above the facial prominence so that a narrow flap is obtained there to fold up against the tube The rest of the mucous membrane is turned downward, the epithelium forming a bed for the transplant



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In some cases where the hypotympanum is very low it may prove very difficult to have a graft not healing directly to the mucous membrane of the middle ear, therefore, in some cases I try to place oxycel treated with chloromycetin succinate, hyaluronidase and prednisolone there Recently I have tried placing a polyethylene tube from the Eustachian orifice over hypotympanum down to the round window and covering it with a full-thickness skin graft I like to try the polyethylene tube there, because in many cases of otosclerosis treated in this manner very little reaction occurs around a polyethylene tube

The first follow-up results after operation may be seen in

Table I, which shows the percentage of improvement and impairment and the cases showing no change in the material of 37 cases of chronic otitis operated on with the *cavum minor* technique This Table shows that one to four months after the operation 49 per cent of the cases showed improvement with a mean gain of 22 db From five to nine months after the operation 53 per cent showed improvement with a mean gain of 25 db

TABLE I

Auditory Results in All Cases After Tympanoplasty *(Cavum Minor)* 39 Cases

	1 - 4 Months Post-op			5 - 9 Months Post-op			12-18 Months Post-		
	No	Per Cent	M db	No	Per Cent	M db	No	Per Cent	M
roved	18	49	22	16	48	25	9	53	
tus quo	18	49	—	17	52	—	7	41	
paired	1	2	13	0	0	—	1	6	
Total	37	100	—	33	100	—	17	100	

M db = mean decibels

The patients subjected to operation had extremely pronounced auditory impairment throughout The mean preoperative level of perception for 500, 1,000 and 2,000 cps was 55 db in the patients examined

Table II shows the auditory results in 72 cases after *cavum minor* tympanoplasty One month after the operation 38 2 per cent had improved with a mean gain of 20 8 db The mean preoperative acuity in this series of 55 cases was 60 db No change was noted in 52 7 per cent Three months after operation we have 43 2 per cent showing improvement with a mean gain of 22 db The mean perception in these 53 cases was 62 db There was no change in 46 2 per cent and 11 6 per cent showed deterioration Improvement corresponds to a gain of 10 or more than 10 db Deterioration corresponds to a loss of more than 10 db Six months after the operation 49 per cent showed improvement in the series of 53 cases, with a mean gain of 21 db, 45 4 per cent showed no change One year after the operation 51 per cent showed improvement, with gain of 25 db

In this series 53 cases were examined, with a mean per-

TABLE II

Results in Tympanoplasty Operations *(Cavum Minor)*

(Free full thickness skin graft)

Time After Op	One Month			Three Months			Six Months			One Year			Two Years			Three Years			Four Years		
	No	%	M db	No	%	M db	No	%	M db	No	%	M db	No	%	M db	No	%	M db	No	%	M db
Improved	21	38 2	20 8	23	43 2	22	26	49	21	27	51	25	19	50	26	9	39	22	6	—	24
No Change	29	52 7	—	25	46 2	—	24	45 4	—	17	32	—	18	47 4	—	13	55	—	4	—	—
Impaired	5	9 1	—	5	11 6	—	3	5 6	—	9	17	—	1	3 6	—	1	6	—	0	—	—
Total No	55			53			53			53			38			23			10		
Mean db before Op			60			62			60			60			61			53			62

Number of cases operated = 72 Mean hearing loss for 500 1 000 and 2 000 cps in the whole material before operation = 60 db ception of 60 db Two years after the operation 50 per cent were improved and 47 4 remained unchanged, 3 6 had deteriorated The mean gain was 26 db and the mean perception before operation was 61 db Three years after the operation we have 39 per cent showing improvement and a mean gain of 22 db The level before the operation was 53 db Four years after the operation I have examined only ten cases thus far This series is too small to justify discussion of the percentage, but six of these ten showed improvement with a mean gain of 24 db, none showed deterioration and four status quo

It is very interesting to note that three years after the operation the percentage showing improvement drops I do not know the significance of this decline Possibly the series is too small to justify conclusions as yet, but after one year I think it will be easier to discuss In the next series, however, four years after the operation about 60 per cent showed better This series is also too small to permit definite conclusions

I personally have treated all these cases postoperatively and I hope to follow them each year now It would be of great interest to see these patients after two, three or four years It is not ideal to obtain only 50 per cent improvement, but I hope that with the better technique and the wider experience we gain, our results will eventually become much better

Recently we have been using more and more fenestration of the oval window rather than fenestration of the horizontal semicircular canal in cases of tympanosclerosis or Paukenscleros (German nomenclature) I have used the Shea method with a vein graft and a polyethylene tube as the stapes in some cases The results of one case are shown in Fig 5 Most recently I have used gelfoam instead of a vein graft over the oval window In these cases and with this technique, there has been no impairment and no dead labyrinth, but the series includes only about ten cases and as yet I can tell you nothing definite about the results of this type of operation It seems to me to be a good solution in some cases, however, but one must be very sure no acute infection is present

TABLE II

Results in Tympanoplasty Operations *(Cavum Minor)*
(Free full thickness skin graft)

Time After Op	One Month			Three Months			Six Months			One Year			Two Years			Three Years			Four Years		
	No	%	M db	No	%	M db	No	%	M db	No	%	M db	No	%	M db	No	%	M db	No	%	M db
Improved	21	38 2	20 8	23	43 2	22	26	49	21	27	51	25	19	50	26	9	39	22	6	—	24
No Change	29	52 7	—	25	46 2	—	24	45 4	—	17	32	—	18	47 4	—	13	55	—	4	—	—
Impaired	5	9 1	—	5	11 6	—	3	5 6	—	9	17	—	1	3 6	—	1	6	—	0	—	—
Total No	55			53			53			53			38			23			10		
Mean db before Op			60			62			60			60			61			53			62

Number of cases operated = 72 Mean hearing loss for 500 1 000 and 2 000 cps in the whole material before operation = 60 db

Late graft failures may be the result of circumscribed cellulitis or epithelial inclusion cysts He believes that the majority of these cysts probably arise from epithelial appendages (hair follicles and glands) Puncturing, crushing or traumatizing the graft in a manner which will bury epidermis into its deeper layers may likewise be responsible for inclusion cysts in some instances

When Dr Schuknecht makes the statement, "one of the most common causes of tympanoplasty failure is loss of graft substance," I don't believe that he means to imply specifically that this is the precise reason why he did not obtain a good functional result in some of his ears I feel certain that the influences which were brought to bear in the destruction of the graft also had their serious effect upon other parts of the sound conducting mechanism, scar tissue occlusion of the round window, necrosis or ankylosis of the ossicular chain and hyalinized collagenous material in the mucosa of the tympanum I have seen many ears with large tympanic perforations in which the auditory function was surprisingly good On many occasions I have been amazed to discover that the patient with a large perforation in his tympanic membrane had socially adequate hearing in the affected ear I dwell upon this observation, which I am certain all of you have made many times, because I think we need a new concentration of thought on the causes of conductive deafness in patients who are considered candidates for tympanoplasty In the vast majority of cases we are dealing with multiple etiological factors and this calls for some serious questioning on our part of what we are doing and why we are doing it

I always accept the position of moderator with a feeling of embarrassment and even uncertainty as to my ability to do justice to the occasion I know that it is my responsibility not only to tell you what the panelist's presentation is about but what is more important to tell you what I think of the quality of his discussion This challenge I accept with pleasure in reviewing Dr. Sullivan's presentation

He proclaims our indebtedness to Dr Julius Lempert for the one-stage fenestration operation He emphasizes the fact

DR FURSTENBERG I am certain that every member of this audience is impressed with the wealth of information on Tympanoplasty that has been presented by our three panelists in this brief period I have had a chance to study these papers, and I doubt that any important phase of the subject has been overlooked It would be superfluous reiteration, therefore, if I took your time to dwell in detail upon the many aspects of these presentations so thoroughly and effectively described and explained by the panelists

In discharging my obligations to this audience I should like to call your attention to Dr Schuknecht's frank statement that his experience in tympanoplasty now consists of 300 operations performed during the past three years His failures have been 30 per cent, but some of these have been revised with a successful result He makes a cogent statement when he warns that, "it is increasingly apparent that it is not always possible to arrest the suppurative process and to reconstruct the middle ear at the same operation" He goes on to emphasize that, "it is also obvious that some failures occur because the nature of the tympanic pathology precluded successful reconstruction"

Dr Schuknecht lists the contraindications to tympanoplasty namely, a malfunctioning Eustachian tube which fails to ventilate the middle ear adequately Second, deficient middle ear mucosa He states that one cannot expect a good functional result "if there is any destruction of mucosa in the area between the Eustachian tube and the round window" Third, hyalinized collagen deposits (tympanosclerosis) in the mucosa of the tympanic cavity The ossicles may be immobilized by this pathological change, and he emphasizes that "these findings are particularly significant in the presence of a large bone air gap which indicates ankylosis of the ossicles"

Dr Schuknecht calls upon his vast experience to tell you why tympanic grafts fail He presents this subject in a detailed manner emphasizing that grafts may fail immediately or many months after the operation The causes are necrosis due to improper packing, trauma, inadequate blood supply and infection graphs of his paper He says, "I am for any kind of meticulous surgery which cleans the disease from the most remote areas heretofore neglected As one who has used the dissecting microscope, not just a loop, from my early work in the facial nerve beginning nearly 30 years ago until the present day, I am very cognizant of this fundamental pre-requisite" He leaves little room for argument when he again calls upon his wide experience to say, "Reconstructive aural surgery as a term may suggest a certain uniformity in the operation, it is not, it is different in every case, not only in details but even in the main lines"

We are deeply indebted to Dr Nils G Richtner, our guest from Stockholm, for his noteworthy contribution to this panel discussion He has described a type of tympanoplasty that is called cavum minor

He has also discussed in considerable detail the difference between free full thickness skin graft and the pedunculated skin and muscle graft He believes that the latter will give better healing of the graft over the middle ear and make the cavity in the mastoid bone smaller

He seeks to obliterate the mastoid cavity with a combined skin-muscle flap in order to obtain a small external auditory meatus He hastens to state, however, that if there is a suppurating otitis he prefers the radical mastoidectomy or some modification of it, and weeks later when the infection has resolved the tympanoplasty is performed

He describes some ingenious ossicular manipulations in making contact between the incus and the stapes, or malleus and the stapes when there is interruption of the ossicular chain I had a chance to read his paper hurriedly last night, but much of it pertained to the slides which have just been presented to us

In closing my comments, permit me to make two more points first, creativeness is the greatest thing in life What a thrill it is to discover some surgical procedure that will change the failures of the past into the successes of the future, but we must remember that the consequences of our creations that this magnificent contribution to otology has given not only serviceable hearing to many of the victims of otosclerosis but that it has also inspired a deeper interest in anatomy of the temporal bone and brought together some sincere and intelligent persons from the ranks of the medical sciences to study diseases of the temporal bone and find solutions for their cure From this operation many observations have been made from which theories have been derived By careful experimentation some of these theories have been reduced to facts, and more will undoubtedly come in the years ahead

Dr Sullivan points out that the aims of the treatment of the chronically discharging middle ear are *1* To eliminate the danger of intracranial complications *2* To obtain a dry ear *3* To preserve and, if possible, to improve the hearing He speaks with conviction when he states that, "chronic otitis media causes pathological and functional defects of varying degrees, hence the method to restore the hearing must vary" He quotes Stacke who in 1897 said, "No one will bind himself to one method in operating, rather will every thoughtful operator individualize and will modify the method from case to case according to the particular pathological condition" Dr Sullivan asks, "What do we mean by the terminology, Tympanoplasty?" Well, says he, "It does not mean what it is supposed to mean, namely, a combination of surgery for irradication of chronic ear inflammation and surgical reconstruction of the sound conducting and the sound protecting mechanism Rarely can it accomplish both of these requisites at one operation" he emphatically states I agree with him wholeheartedly in this assertion

Commenting further on tympanoplasty, Dr Sullivan states, "the results of many, many cases do not justify this tedious, meticulous surgery, because the hearing improvement is not predictable It is true that the ears can be tested to determine the cochlear function, but you cannot test in an accurate manner how the middle ear will function after the procedure is completed" His knowledge and judgment gained from 30 years of surgical experience in the temporal bone are, I believe, clearly revealed by two statements in the closing para-theories is taboo If you have one, let us have it, is my urgent appeal to the members of our profession Darwin formed a theory about the origin of species Some of his ideas had to be modified or discarded, but Darwin has not lost stature nor do biologists regard his work as valueless Incorrect theories about the universe have been offered in the past, but where would astronomy be now if past investigators had ever ceased to form new theories Theories led to the discovery of some comets and their orbits Ptolemy presented some astoundingly erroneous theories, but the epochs he created in the progress of astronomy have endured for centuries The sides of the road up which civilization has travelled are littered with discarded theories, but civilization would have been greatly retarded if inquisitive people had never had the courage to try or to propose anything new

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are the legacy that we leave to the men and women who follow in our footsteps. I cringe from the memories of the fads and fancies of otolaryngology during the period from 1910 to 1930 Eye complications arising from diseases of the nasal accessory sinuses, particularly iritis, choroiditis, and retro-bulbar optic neuritis were over emphasized, and the sinuses suffered radical surgical abuse The frequent occurrence of sinusitis in infants and children was stressed dogmatically, and the nasal accessory sinuses came in for another surgical assault We were led astray by the presumption that asthma was the result of a nasal reflex and that radical operations upon the nasal accessory sinuses with removal of their lining mucous membranes were curative measures Acute mastoiditis which often proved to be a phantom variety was accused of being responsible for diarrhea in infants and children and posted for mastoidectomy on the surgical schedules of many a famous otologist in the years to which I refer Convictions of the existence of a spheno-palatine ganglion syndrome were fleeting and the administration of vitamins for the restoration of auditory function in perceptive deafness has not been substantiated by scientific studies of an acceptable character These were some of the whimsical creations of our profession in the period of which I speak Most of you in this audience today are too young to appreciate the otolaryngological trends of this era, but I think you should know that you were left a legacy from which all of us are still licking our wounds, particularly in the field of medical education

Finally, I congratulate the panelists for the noteworthy contributions they have made to this important discussion They have fulfilled their obligation as our essayists by speaking out convincingly and critically of their observations and achievements in this important field of rehabilitation With freedom of thought and expression extended them as invited panelists, they have taken their responsibilities seriously and provided us with a wealth of information and practical recommendations This pooling of ideas or theories on the problems of tympanoplasty may lead to final proof or disproof or even to some other solution of our problems

I doubt that there is any science in which the offering of

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THE PRINCIPLES AND PRACTICE OF BONE CONDUCTION AUDIOMETRY

A Review of the Present Position[*]

J. D HOOD,[†]
London, England

Whereas the technique of air conduction audiometry is well established, the basic principles of bone conduction audiometry are still far from being generally understood In consequence, the tests are still too often inefficiently performed and hence fail to provide the vital diagnostic information which is their purpose

As will be shown, this disappointing state of affairs is quite unnecessary, and requires for its correction no more than a rational standardization of our technical equipment and test procedures, whereby proper use is made of the considerable body of acoustic and physiological knowledge now at our disposal

An essential requirement with all bone conduction tests is the exclusion of the untested ear by means of an efficient masking sound, so that all threshold readings can be validly related to the tested ear In order to understand how this requirement arises, we need to know first certain facts concerning the transmission of air and bone conducted sounds across the head, second, the physical characteristics of a sound which confer upon it maximum efficiency for the masking of pure tones of specified frequency

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[†] From the Otological Research Unit Medical Research Council National Hospital Queen Square London W C 1 England

The facts relating to sound transmission across the head are conveniently studied in subjects with normal hearing in one ear and complete deafness of the other

The results obtained are shown in Fig 1 The calibrated

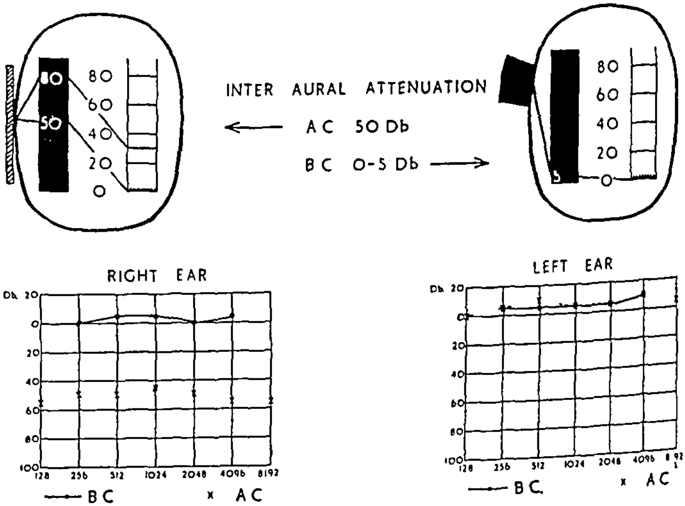

Fig 1

columns are taken to represent the available hearing capacity of each ear, the solid column at the left represents total deafness of the right ear Positive responses to the test tones of different levels above normal threshold are indicated in this and succeeding figures by serrated lines If an air-conducted sound of 50 db is applied to the right, or deaf ear, it will be heard in the left ear at threshold intensity Similarly, a sound of 80 db applied to the right ear will be heard in the left ear at a level some 30 db above threshold The reason for this is that sound from an air conduction receiver is transmitted across the skull with an interaural attentuation of the order of 50 db By contrast, the interaural attentuation for sound from a bone conduction receiver applied to the right mastoid is negligible, and the sound will be heard at the left ear with a loss of only a few decibels

In the lower half of Fig 1 are shown the pure tone audiograms of the right and left ears for air and bone conduction carried out without masking They indicate normal hearing for the left ear, and a severe conductive deafness for the right ear In agreement with this, the classical tuning fork tests applied to the right ear lead us into that well-known pitfall of clinical practice, the false negative Rinné, in fact, for the reasons given, the threshold readings, both for air and bone conduction, obtained for the right ear and shown in the audiograms, are false readings derived from the left or untested ear In the case of air conduction, owing to the comparatively high interaural attentuation of 50 db, such false readings are obtained only when the stimulus intensity exceeds this level Clearly enough, true readings would result with any degree of deafness of the right ear below this level, hence the rule that when the difference in the air-conduction thresholds of the two ears exceeds 50 db the readings for the worst ear cannot be relied upon, unless the better ear is excluded by masking

In the case of bone conduction, however, the situation is much more difficult Here the interaural attenuation is negligible, and hence, if the bone-conduction threshold of the right ear be raised by any amount above 5 db, a receiver on the right mastoid will give false normal readings derived from the opposite untested ear, hence the rule that *all* bone conduction tests require the exclusion by an air-conducted masking sound of the untested ear A variety of masking noises has been devised to serve this purpose A few, like those described by Zwislocki (1951), or Denes and Naunton (1952) are efficient, but of the majority it can only be said that they raise the threshold of the masked ear in some vague unspecified manner

In order to understand what makes a masking sound efficient, it is helpful to consider first the masking of pure tones by white noise, that is to say, noise having a flat frequency spectrum covering the whole audible frequency range with equal sound energy per cycle In Fig 2 is shown the elevation of the threshold of a pure tone of 1,000 cycles in the presence of white noise The noise intensity is expressed in decibels above normal threshold, and it will be seen that below the 20 db level it produces negligible masking of the pure tone Above this level there is an incremental equivalence in decibels of masking and of masking noise intensity At any masking level, therefore, the loudness of the white

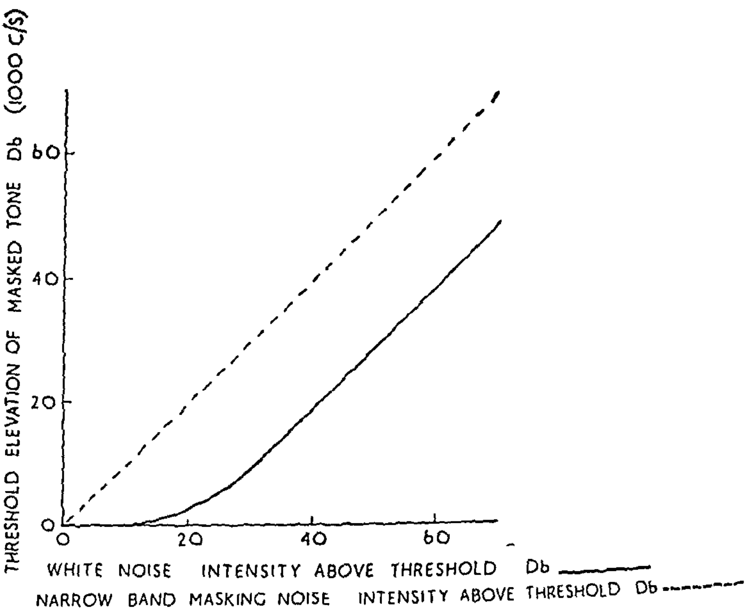

Fig 2

noise will be much in excess of the pure tone, since its intensity will at all times be some 20 db higher

This brings us to the first important requirement of a masking noise, namely, high masking efficiency which Denes and Naunton have defined as "good masking ability, but minimal loudness" Clearly, white noise does not conform to this ideal, nevertheless, it is superior to some of the masking noises incorporated in many commercial audiometers A typical example is shown in Fig 3. The masking noise is modified mains hum, its intensity level in decibels above normal threshold is shown on the horizontal scale, and on the vertical scale the resulting threshold elevation of various pure tones It will be seen that for frequencies above 1 000 cycles masking is grossly inefficient At 4,000 cycles no masking at all is produced below an intensity level of some 50 db at which point it is already becoming unpleasantly loud to the subject

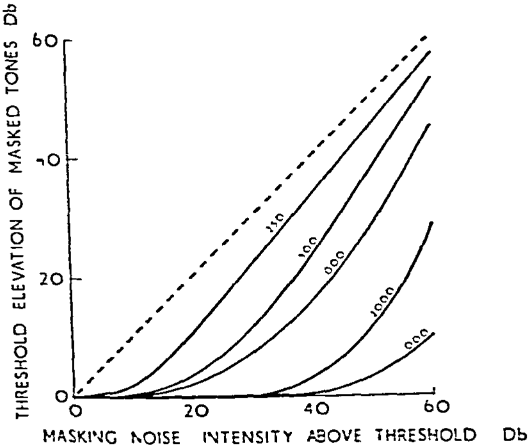

Fig 3

Having considered these examples of inefficient masking sounds, we will consider next how we set about our task of designing what we really require, that is to say, sounds which have the maximum possible efficiency for the masking of the limited range of specified pure tones, which are conventionally used for bone-conduction audiometry. Here, as usual, we turn for guidance to some of Fletcher's (1940) early work upon frequency discrimination, loudness and masking, which led him to define the existence of certain so-called critical frequency bands Their significance lies in the fact, first, that in any masking noise only those frequencies within a certain critical band centered around the pure tone contribute to its masking, and second, when the tone is just audible against the background of noise, the total acoustic power of the components within the band is the same as that of the pure tone The principles are illustrated schematically in Fig 4 This shows noise bands of varying widths centered about a single pure tone which is just audible in their presence The acoustic energy, both in the bands and in the pure tones, is represented by the enclosed area of each Band A is only three cycles wide, and since its total energy is the same as that of the pure tone, the energy per cycle of the noise is only one-third that of the pure tone Band B is five cycles wide Once again, its total energy is the same as that of the pure tone, which thus has five times as much energy as each cycle of the band The width of Band C is that of the critical band, and yet again the total energy in the band is equal to that of the pure tone

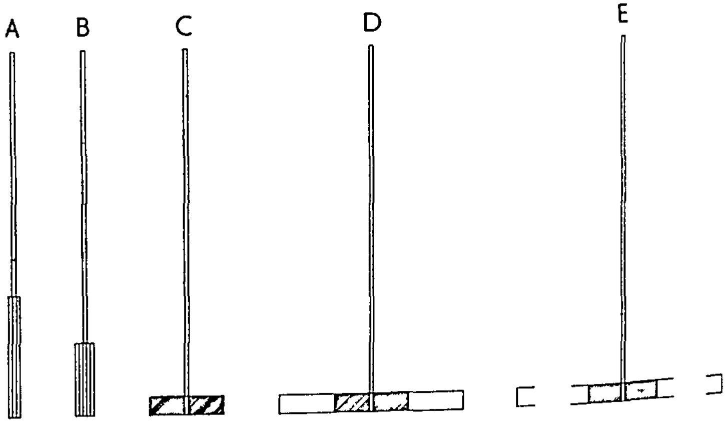

Fig 4

In bands D and C the noise spectrum extends beyond the critical band, so that the total energy of the bands is much increased Since, however, it is only the energy in the critical band which contributes to the masking, these noise bands possess no greater masking power, and the energy level of the just masked pure tone remains the same In other words, those components outside the critical band, while they may have the effect of making the noise very much louder, contribute nothing to its effective masking power This provides us with the theoretical basis for the concept of high masking efficiency, clearly our first objective must be to remove as much as possible of these unnecessary components

Are we likely to gain anything from the use of band widths which are only a fraction of the critical band, as for example in Band B? Apart from the fact that to do so would present very difficult technical problems of sound filtering, we should gain nothing in efficiency and, in addition, would be faced with the added difficulty of the tonal similarity of the pure tone and the masking noise which might result in some confusion to the patient If, however, the band width of the masking noise is extended to correspond roughly to that of the critical band, as in Band C, then we find that though masking sound and pure tone are identical in pitch, they are still readily distinguishable, and hence are not confused by the subject tested

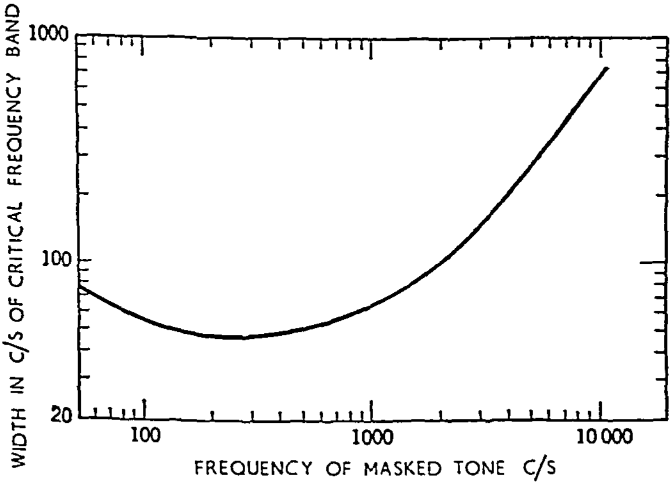

Fig 5

The critical band widths for the various frequencies are shown in Fig 5 As will be seen, they are of the order of only a hundred cycles wide It would be technically impossible to produce noise bands to these specifications, nor, as will be shown, is this necessary

In Fig 6 is shown schematically the equipment we have used for obtaining narrow bands of noise, having the masking efficiency required A white noise generator is followed in turn by a number of filters of high selectivity centered around each of the pure tones normally used in bone conduction audiometry, followed in turn by an attenuator calibrated in steps of 10 db Each of the filters can be selected by means of a switch, and passes only a very narrow band of frequencies

The selectivity curve for the band centered at 1,000 c/s, which is typical of the other curves, is shown in Fig 7 At the center, the two vertical lines give the measure of the width of the critical band The curves extend somewhat beyond the critical band at the base, nevertheless, the maximum energy in the band is centered within the two parallel lines

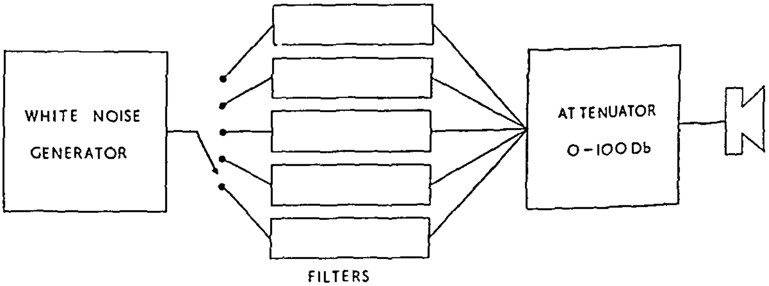

Fig 6

The dotted line in Fig 2 illustrates the high masking efficiency obtained with this band

In contrast to the masking effect of white noise, there is 100 per cent masking efficiency The curve begins at zero, and there is an exact one-to-one relationship between noise level and masked threshold Thus, if the noise is raised 20 db, the pure tone threshold is raised by an identical amount, and so on All the noise bands are similar in this respect, and we regard it as one of their most important features Thus, if it is desired to raise the threshold of a pure tone by, say 40 db, it becomes a simple matter of raising the intensity level of the noise band by exactly the same amount The practical importance of this is best illustrated by considering the simple application of these noise bands to the technique of bone conduction tests

Fig 8 is a schematic illustration of the rationale of the procedure employed at the Otological Research Unit, and referred to for convenience as the "shadowing" technique Let there be a pure nerve deafness of 40 db of the subject's right

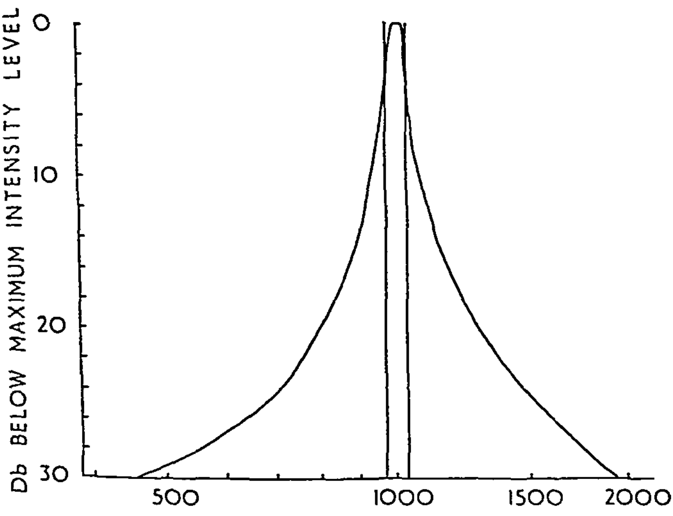

Fig 7

ear to be tested, and no loss of hearing of the left ear, as before, the columns of each side are taken to represent the available hearing, and are calibrated in decibels The solid portion of the left column represents the nerve deafness of the right ear A bone conducted sound at normal threshold intensity applied to the right mastoid is heard in the subject's left ear This provides us with the first point on the graph shown below, in which the masking intensity at the left ear is plotted on the horizontal scale and the bone-conduction threshold responses on the vertical scale

Now, as shown in Fig 8, the masking noise at a level of 10 db applied to the left ear raises its threshold for the bone conducted test tone by 10 db In the same way, increases in masking noise intensity of 20 or 30 db will bring about increases of identical magnitude in the threshold values for the test tone Reference to the graph shows, up to this point, this step by step correlation, the "shadowing" effect between the intensities of masking noise and test tone thresholds When, however, the masking noise reaches the 40 db level it

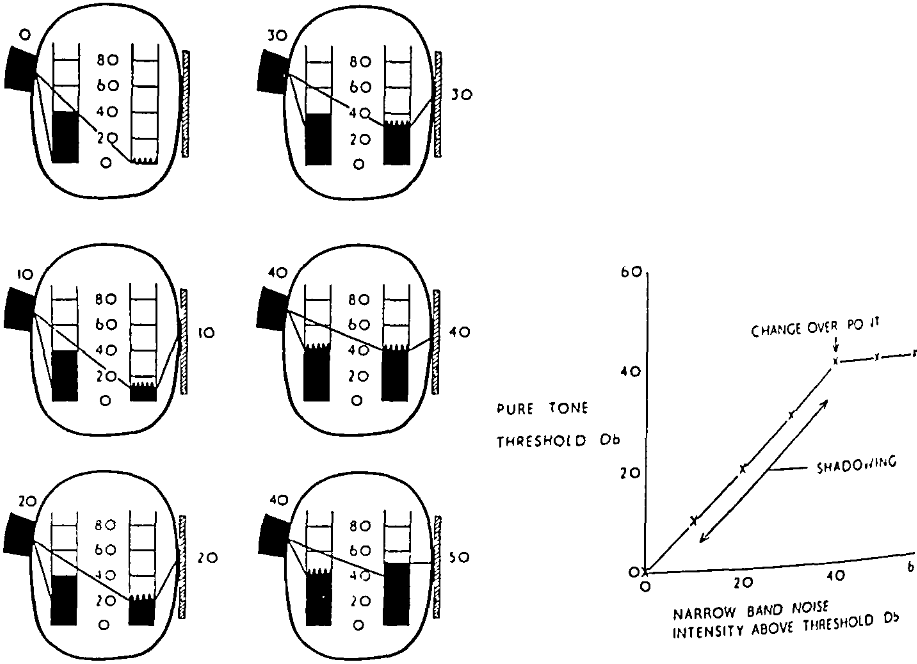

Fig 8

will be seen that the bone conducted test tone will be heard *at threshold by the right as well as the left ear* A further intensity increase of the masking noise to 50 or 60 db will further raise the threshold of the left ear alone, leaving the test tone to be heard by the right ear alone without further elevation above its true threshold value, 40 db The true *threshold for the right ear* will thus be shown at a point on the graph, referred to as the "change-over" point, at which the slope of the curve changes from 45 degrees and becomes horizontal In the case being considered, the change-over point occurs at 40 db If, extending the argument, the deafness of the tested ear had been 50, 60 or 80 db identical values of masking noise intensity would have been required to reach the "change-over" points In all cases, true threshold readings would be obtained

The essential steps of the test procedure are thus

1 The demonstration of the "shadowing" effect This establishes that both masking noise and test tone are confined to the untested ear

2 The identification of the "change-over" point This establishes the true threshold of the tested ear

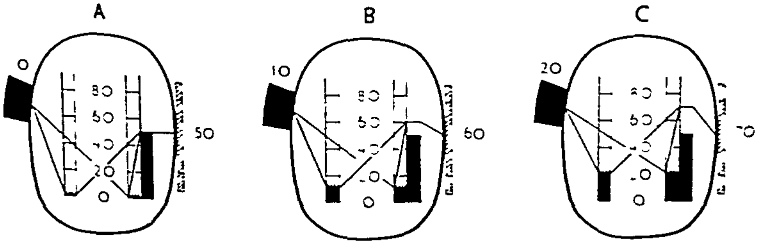

Fig 9

The case analyzed in Fig 8 is one of comparative simplicity in that the unilateral deafness was of the "nerve" type In unilateral deafness of the conductive type, however, we are confronted with a situation of considerably greater difficulty This arises, as will be shown, from the fact that the interaural attentuation of an air-conducted masking sound does not exceed some 50 db

The situation is illustrated in Fig 9

Here, the subject has normal hearing of the right, or tested ear, and a pure conductive deafness of 50 db of the left ear This is indicated by the solid area adjoining the calibrated column which represents the inner ear hearing capacity of the left ear, reduced in B and C by masking conducted test tone by 10 db In the same way, increases in masking noise intensity of 20 or 30 db will bring about increases of identical magnitude in the threshold values for the test tone Reference to the graph shows, up to this point, this step by step correlation, the "shadowing" effect between the intensities of masking noise and test tone thresholds When, however, the masking noise reaches the 40 db level it

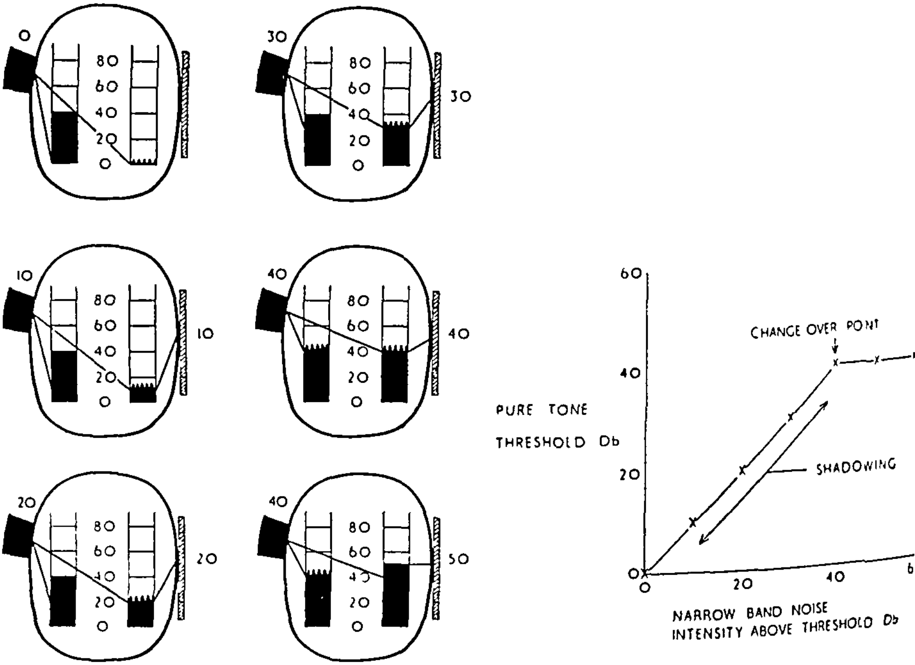

Fig 8

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As Littler, et al (1952) so ably pointed out, the practical importance of this finding is considerable, making possible as it does the accurate determination of bone-conduction thresholds in the presence of conductive deafness well beyond the limit of 50 db This is illustrated in Fig 10 Masking noises of intensities up to 90 db are applied to the left ear by means of a rubber-tipped-insert receiver without fear of cross conduction to the right ear

Thus, at any intensity between 50 db, the air-conduction threshold of the left ear, and 90 db, the masking noise will be effective in the left ear alone If we now apply the "shadowing" procedure, and make step by step changes of masking intensity within this range, they will be found to leave unaltered the bone-conduction threshold readings In other words, the "shadowing" effect is absent, and the threshold reading, 0 db, can be accepted as valid for the right ear

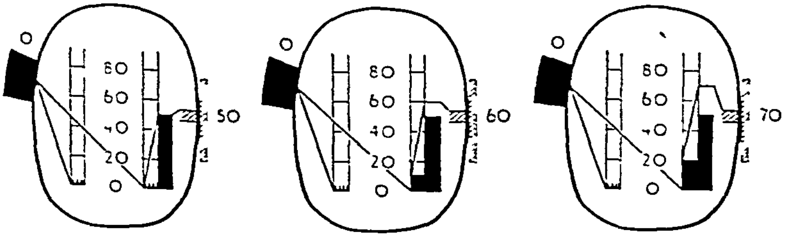

Fig 10

When the deafness is bilateral, and of the mixed conductive and nerve type, bone-conduction tests may be greatly complicated, and indeed it is only within certain limits that accurate results are then obtainable.

An audiometrician needs to know what these limits are, and it is fortunately possible to define them with reasonable accuracy The situation is analyzed in the cases shown in Fig 11 (A, B and C) In 11-A the subject has a nerve deafness of 60 db of the right ear and conductive deafness of 30 db of the left In order to establish the bone-conduction threshold of the right ear, the usual masking noise must be

In ascertaining the bone-conduction threshold of the right ear we must, as usual, apply an air-conducted masking noise to the left ear Here a serious difficulty arises, since before this noise can become effective its intensity must reach 50 db, the amount of the conductive deafness At this intensity it will just overcome the interaural attenuation for air-conducted sound, and begin to exert a masking effect upon the right ear as well as the left If the intensity of the masking noise be further increased in steps of 10 db, a "shadowing" effect will occur, and the bone-conduction thresholds, derived equally from the two ears, will rise *pari passu* In this instance, however, the "shadowing" effect will continue indefinitely, and no "change-over" point will be reached

The underlying difficulty is clearly to be found in the fact that the interaural attenuation of the air-conducted masking noise has a limit of some 50 db What is needed, therefore, is some means of extending this limit, and providing a zone of masking noise intensities which will be above the air-conducted threshold of, and at the same time confined to, the left ear Within this zone, the "shadowing" procedure could be applied, and the absence of a "shadowing" effect would then prove that bone-conduction threshold readings were derived from the right ear, and could be accepted as valid

The manner in which such an increase of the interaural attenuation can be accomplished stems from a brief report on the use of insert receivers which appeared in the Report of the Electro-Acoustics Committee "Hearing Aids and Audiometer," 1947 In this it was suggested that the acoustic linkage between the two ears which occurs when any air conduction receiver is used arises from the vibrations imparted to the skull at the air-bone interface bounding the receiver Both Littler, et al (1952) and Zwislocki (1953) have carried out further studies, and it has been shown that the vital factor affecting the attenuation is the area of the head exposed to the air conducted sound waves Thus, the smaller the area the greater the attenuation, and in the limiting case when an insert receiver is used, thus presenting the smallest possible surface to the skull, the interaural attenuation increases to a figure as high as 90 db the maximum practicable level, will raise the threshold of the left ear by a further 10 db while leaving unaffected at 60 db that of the right ear This, therefore, will be its true threshold

Two factors determine the results thus obtained First, 100 db is accepted as the maximum intensity of masking noise which it is practicable to use for clinical tests With this the critical change-over point occurs at a level 10 db lower, 90 db, and to this it is convenient to refer as the maximum critical masking intensity Second, the threshold of the tested ear can be determined only if it lies at or below the level of masking induced by the maximum critical masking intensity in the untested ear

Now, in Case 11-A this level is reduced by 30 db, the amount of the conduction deafness and hence, 60 db is the maximum measurable elevation of the bone-conduction threshold of the right ear From this we may derive the limiting formula

Maximum measurable bone-conduction threshold=90 - conductive deafness (db) of opposite ear

The application of the test procedure to the three cases A, B and C, represented in Fig 11, is shown in the graph

In all three the amount of the nerve deafness of the tested ear is the maximum measurable according to the limiting formula Thus

Case A	60 db	(90-30)
Case B	40 db	(90-50)
Case C	30 db	(90-60)

In each case the true threshold is given by the change-over point which occurs at the maximum critical intensity, 90 db of masking noise

Although for simplicity of explanation the cases shown in Fig 11 have been selected to exhibit conductive deafness alone of the untested ear and nerve deafness alone of the tested ear, accurate bone-conduction tests are still possible within the limits of the formula when the deafness, of either

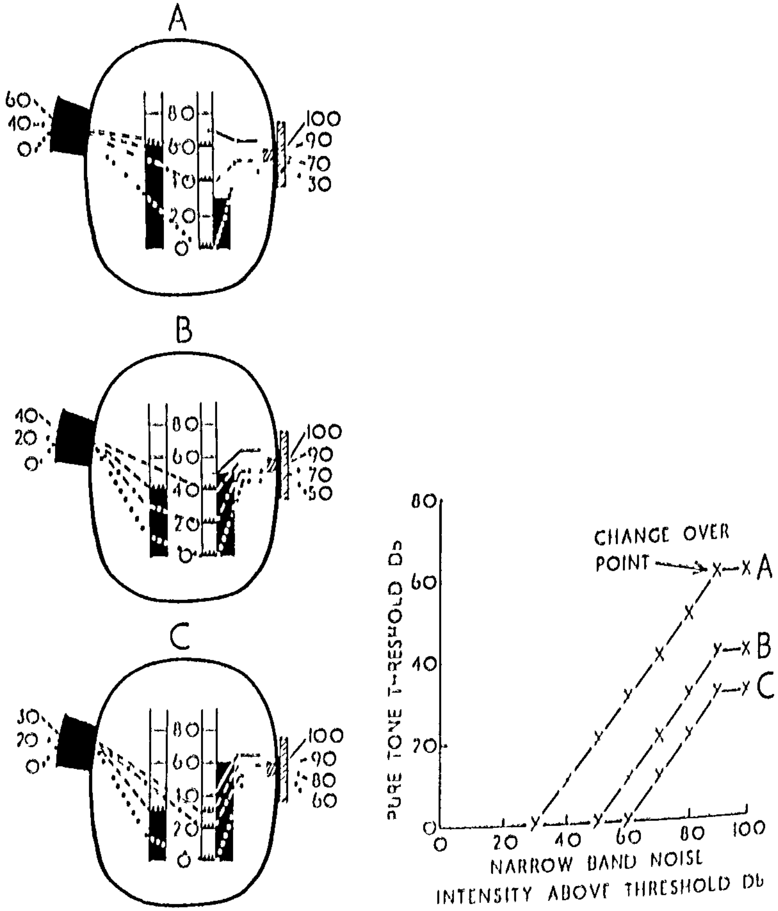

Fig. 11

applied to the left, and owing to the conductive deafness its minimum effective intensity will be 30 db. Above this point shadowing will occur, as shown in the graph (A); that is to say, both masking noise and test tone will be confined to the left or untested ear. When the masking intensity reaches the 90 db level, its true masking effect upon the left ear will be restricted to 60 db by the conductive deafness and the test tone will now be heard at threshold in the right ear as well as in the left. This is the critical change-over point, and beyond it a further increase of masking intensity to 100 db,

Right ear 100 – 60=40 db
Left ear 80 – 50=30 db

In each case, the amount of the nerve deafness is the maximum which can be measured according to this formula Thus for the right ear

60 db (amount of nerve deafness) =90 – 30 (amount of conductive deafness of left ear)

For the left ear

50 db (amount of nerve deafness) =90 – 40 (amount of conductive deafness of right ear) As will be seen from the foregoing analyses, the use of the maximum efficiency masking technique described in this communication brings within the resolving power of air and bone-conduction audiometry all but the few exceptional cases which lie beyond the bounds of the limiting formula

In all cases however, careful attention should be given to the fundamental features of the test procedure which is outlined as follows

1 Establish the air-conduction audiogram of both ears in the normal way with masking, if necessary, of the untested ear, *i e*, when the difference in the hearing loss between the two ears exceeds 50 db

2 Find the bone-conduction threshold with the bone-conductor applied to the mastoid of the tested ear without masking of the untested ear

3 Apply the masking noise of the appropriate band to the untested ear by means of an insert receiver and find a bone-conduction threshold reading

4 Apply the "shadowing" procedure thus Increase the level of the masking noise by 10 db above threshold and retest the bone-conduction threshold If the bone-conduction threshold is raised by 10 db increase the masking intensity by another 10 db and repeat Continue this procedure until the point is reached at which the bone conduction remains constant with further additional incremental steps of 10 db one or both ears, is of the mixed type This is illustrated in Fig 12 In the right ear there is a conductive deafness of 40 db with a nerve deafness of 60 db The air-conduction audiogram thus shows a loss of 100 db In the left ear there is a conductive deafness of 30 db with a nerve deafness of 50 db. The air-conduction audiogram thus shows a loss of 80 db With no masking noise applied to the left ear the bone-conduction threshold reading is 50 db, derived, in fact, from the left ear, though this cannot be specified at this stage of the test

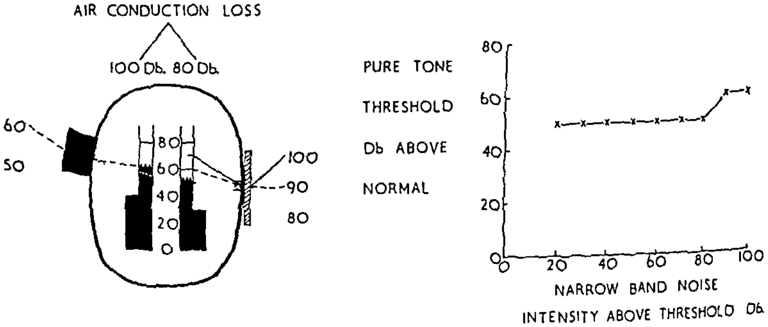

Fig 12

This reading remains constant with increase of masking noise intensity up to 80 db With the masking intensity increased to 90 db, however, and still confined at this level to the left ear, the threshold reading is increased to 60 db and remains unchanged when the masking intensity is further increased to 100db As shown in the graph, "shadowing" occurs between the 80 and 90 db levels of masking noise intensity with the "change-over" point at 90 db It follows that up to the 80 db level of masking noise intensity, the threshold reading, 50 db, must be derived from the left ear, further, that at and above the 90 db level, the threshold reading, 60 db, is derived from the right ear Subtraction of these bone-conduction losses from the air-conduction losses gives the amounts of the conductive deafness, thus

FRONTAL SINUS SURGERY

The Use of Mucoperiosteal Flap for Reconstruction of a Nasofrontal Duct[*]

JOSEPH H OGURA, M D ,
RONALD K WATSON, M D ,[†]
and
A A JUREMA, M D ,[‡]
St Louis, Mo

INTRODUCTION

Surgery of the frontal sinus has been fraught with failure for one main reason closure of the nasofrontal duct[8,12] This paper presents a method of maintaining patency of the nasofrontal duct The original description was published in the South American literature by Tato[9] in 1948 The method consists of rotation of mucoperiosteal mucous membrane flaps from the nose upward into the frontal sinus to create a new nasofrontal duct common to both sinuses We present a small series with an advanced form of frontal sinus disease, *i e*, unilateral orbital complications, fistulae, mucoceles, and/or bony wall destruction, where the problem of the nasofrontal duct is obvious

REVIEW OF LITERATURE

In reviewing the literature on surgery of the frontal sinus it is apparent that closure of the nasofrontal duct is stated to be the greatest cause of recurrence[12] In 1942 Goodale[5] reviewed 182 cases of frontal sinus disease at Massachusetts Eye and Ear Infirmary and found a recurrence rate of 30 8

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of the masking noise This is the "change-over" point, and gives the true bone-conduction threshold of the tested ear

Suggestions for the improvement of standard equipment tend to encounter two kinds of objections first, that they involve excessive technical difficulties, second, that the solution they offer is far from complete, and hence further development should be awaited

Neither of these objections appears to be applicable in the present situation No serious difficulties attend the generation or application of the masking sounds described, and as for further developments, these cannot be expected since in many respects the efficiency obtained is 100 per cent, at which level an ample reserve is provided for the attainment of practically all the clinical results which are required It is, therefore, to be hoped that the methods described will find useful application in improving the design of standard audiometric equipment

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THE JAMES E NEWCOMB AWARD

Dr Joel J Pressman, of Los Angeles, was chosen by the members of the American Laryngological Association at its annual meeting recently, to be the recipient of the James E Newcomb Award for his outstanding contribution to research in otolaryngology of the frontal sinus cavity with scar tissue depends upon the total obliteration of the sinus and of the nasofrontal duct with removal of all mucous membranes

The Lynch type external frontal is satisfactory if the region of the nasofrontal duct is not involved by disease When it is, a new nasofrontal duct is required This is maintained by the use of artificial tubes for a variable period of time and is not ideal because of the lack of cilia and normal glands, a mucosal-lined duct is preferred If a large opening is made into the ethmoids this will frequently close in time regardless of initial size

The De Lima approach to the frontal via the antrum and ethmoids is the procedure of choice for primary disease in antrum and ethmoids with secondary frontal involvement, but this method should not be used to provide permanent drainage when the nasofrontal duct is irreversibly obliterated by the disease A combination of the Bergara osteoplastic or Lynch type methods with the De Lima approach still does not always assure a permanent drainage from the frontal sinus, in view of the marked tendency to scar formation in the region of the frontal floor which may obliterate the surgically created opening into the ethmoids

The trephine method is an emergency procedure and is done to relieve the acute purulent infection and provide temporary drainage

The use of mucosal flaps from the nose is not new They may be unilateral or bilateral Actually this method is a means for reconstruction of the new nasofrontal duct combined with an approach to the frontal sinus The bilateral flap technique is preferred because the new opening is lined on two sides with an absence of a midline partition and is more likely to stay open than one lined only on one side By turning both flaps the new duct is made wider, and tendency to stenosis is reduced by loss of the midline wall, which in essence is the inferior extension of the intersinus septum

INDICATION FOR FRONTAL SINUS SURGERY

A generation ago a great deal of radical sinus surgery was per cent for infection and 22 2 per cent for mucoceles. Prior to his paper and subsequent to this, various methods have been suggested to maintain patency of the duct. These include the use of reamers, dilators, skin grafts, mucosal grafts and the permanent placement of plastic, gold and tantalum tubes Walsh's[11] suggestion that the nasofrontal duct be left alone, if not damaged by disease, is obviously the best treatment; however, there are many instances where the duct is obliterated or destroyed by disease, but where there is no osteomyelitis It is for these cases that the mucosal flap method is advocated to reestablish adequate drainage

At the present time several surgical procedures are available for treating disease of the frontal sinus and the principle underlying each is different These are

1 The classic obliterative procedure (Riedel and Lyman) [7]

2a The osteoplastic flap approach for exposure (Gibson and Walker) [4,6]

2b The osteoplastic flap procedure with obliteration (Bergara) [1,10]

3 The Lynch type external frontal and its variations

4 The Ermiro de Lima frontotomy through a trans-antral ethmoidectomy

5 Simple trephining of the frontal floor

6 Mucosal flaps turned up from the nose either unilateral (Sewell and Boyden[2,3]) or bilateral (Tato[9])

Each method has its advantages and disadvantages The classic obliterative procedure is advocated only for cases of osteomyelitis of the frontal bone, it is usually deforming and is not necessary if alternative methods are available

The osteoplastic flap gives very wide exposure in dealing with tumors or osteomas of the frontal sinus, but this method does not in itself provide drainage and presumes a functioning nasofrontal duct

The success of the osteoplastic approach with obliteration of the frontal sinus cavity with scar tissue depends upon the total obliteration of the sinus and of the nasofrontal duct with removal of all mucous membranes

The Lynch type external frontal is satisfactory if the region of the nasofrontal duct is not involved by disease When it is, a new nasofrontal duct is required This is maintained by the use of artificial tubes for a variable period of time and is not ideal because of the lack of cilia and normal glands, a mucosal-lined duct is preferred If a large opening is made into the ethmoids this will frequently close in time regardless of initial size

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INDICATION FOR FRONTAL SINUS SURGERY

A generation ago a great deal of radical sinus surgery was performed, and irreversible harm was done Exenteration of the interior of the nose markedly alters the normal physiology and left many people "nasal cripples" Since then there has come a better understanding of the normal function of the nose and a more conservative approach to sinus problems Whenever possible normal structures are left undisturbed and mucosa which has not suffered irreversible changes due to infection is left intact The main objective has been to achieve good drainage When this is established much of the pathology will clear up without radical surgery Hence surgical attack on the frontal sinus is indicated primarily for *1* orbital complications such as cellulitis, abscess or displacement, *2* mucocele, *3* fistula, *4* chronic severe recurrent frontal sinusitis with severe local signs and symptoms due to infection or associated with osteoma We assume that the nasofrontal duct must be closed to produce this localized pathology Some cases require surgery to the antra and ethmoids as well as to the frontals This is usually done as a separate procedure either before or after the frontal surgery, but it may be combined

SURGICAL TECHNIQUE

A curved incision as described by Lynch is made down to and through the periosteum It begins in the eyebrow and extends medially to a point midway between the bridge of the nose and the inner canthus of the eye The incision is continued inferiorly along the lateral edge of the nasal bone to its distal edge and then curves laterally two or three millimeters The periosteum is elevated from the supra-orbital area, the medial wall of the orbit, the nasal bone and the ascending process of the maxilla The anterior and posterior ethmoidal arteries are divided and tied or cauterized The supra-orbital nerve and vessels are sacrificed The trochlea and the lacrimal sac are detached and displaced laterally with the globe The frontal sinus can then be entered either through the floor or via the anterior ethmoid cells

Frequently the disease process has ruptured through the frontal floor and the sinus is entered while making the incision In many cases the entire roof and medial wall of the orbit have been eroded, and the frontal and ethmoid sinuses are one large cavity full of fibrous and granulation tissue The frontal sinus is entered and the opening enlarged by removing the floor and overhanging supra-orbital bone with rongeurs

If the ethmoids are not involved, they are not disturbed any more than is necessary for the formation of mucoperiosteal flaps A complete ethmoidectomy is done at this stage if indicated When this exenteration of frontal and ethmoid regions is complete the formation of the mucoperiosteal flap is begun

Using a cutting burr as used in mastoid surgery and curettes, the nasal bone, lacrimal bone and a portion of the ascending process of the maxilla on the involved side are removed (see Fig 1) The intersinus septum is taken down, and the nasal spine of the frontal bone is thinned There is a triangular wedge of bone lying between the medial portion of the two frontal sinus cavities and the bridge of the nose This must be removed and made smooth

There should now still remain a mass of bone lying anterior to the cribriform plate and just superior to the vault of the nose This is the root of the perpendicular plate of the ethmoid and the nasal septum (see Fig 1) This bone is carefully removed until the mucous membrane of the nasal vault is exposed Bone is removed anteriorly as far as possible without lowering the projection of the dorsum in order to give as much antero-posterior dimension as possible to the mucosal flap When one side has been exposed further bone is removed to expose the vault on the opposite side The width of the flap is limited anteriorly by the midline projection of the nose and posteriorly by the anterior end of the cribriform plate (see Fig 1) The flaps are then pushed down into the nose, and the bony septum is removed with burr and rongeurs Great care must be taken not to tear the flaps When the mucosal domes have been well exposed and their future bed fashioned the nearest side is cut first, the two layers are cut with curved scissors as far anteriorly as pos-

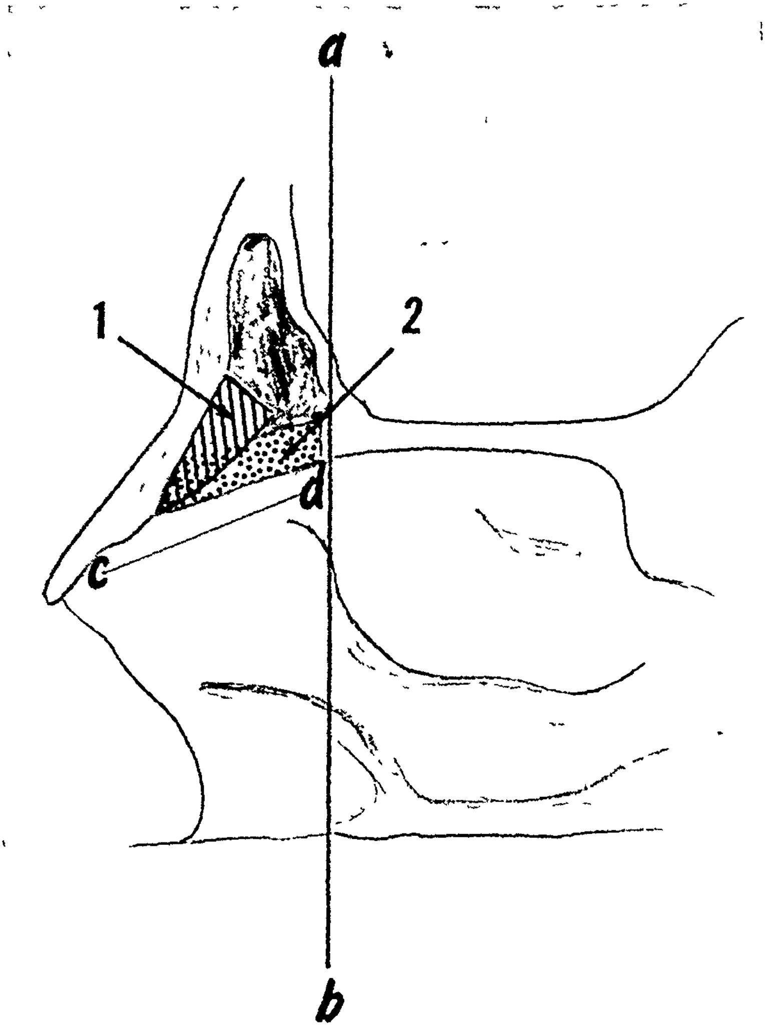


Fig 1 Sagittal section of frontal sinus View of right medial wall of frontal sinus ab—An arbitrary line marked posteriorly by the posterior wall of the frontal sinus When projected inferiorly this represents the anterior attachment of the middle turbinate The cribiform plate is posterior to this line cd—antero-posterior dimension of dome of mucoperiostial flap 1—heavy wedge of bone deep to glabella 2—root of perpendicular plate of ethmoid

sible The medial layer is cut as far inferiorly as can be reached A similar incision is made posteriorly, usually sloping downward and forward (see Figs 1 and 2)

Pressing the double layered flap laterally the mucosal attachment to the septum is cut and the flap turned upward and laterally Usually a further small portion of the ascending

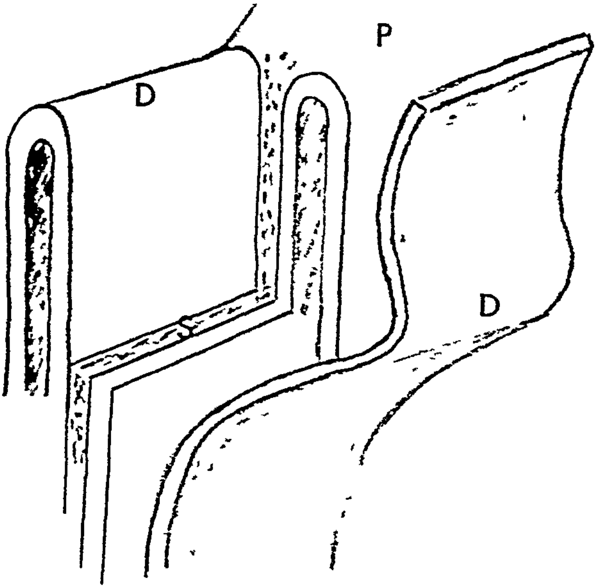


Fig 2 Preparation of septo-mucoperiostial flaps (modified after Tato) D—dome S—perpendicular plate of ethmoid and septum partially removed P—posterior wall of frontal sinus

process of the maxilla must be removed so that the flap will lie flat on the peri-orbita (see Fig 3) This flap is carefully tucked out of the way and the opposite flap cut in a similar manner The area of bone lying between the open field and the opposite nasal bone, trochlea and lacrimal sac, must be smooth and flat so that the mucosal flap can be turned upward and laterally into the opposite frontal sinus (see Fig 3) We must try to achieve a symmetrically funnel-shaped opening into both frontals draining into the anterior nasal

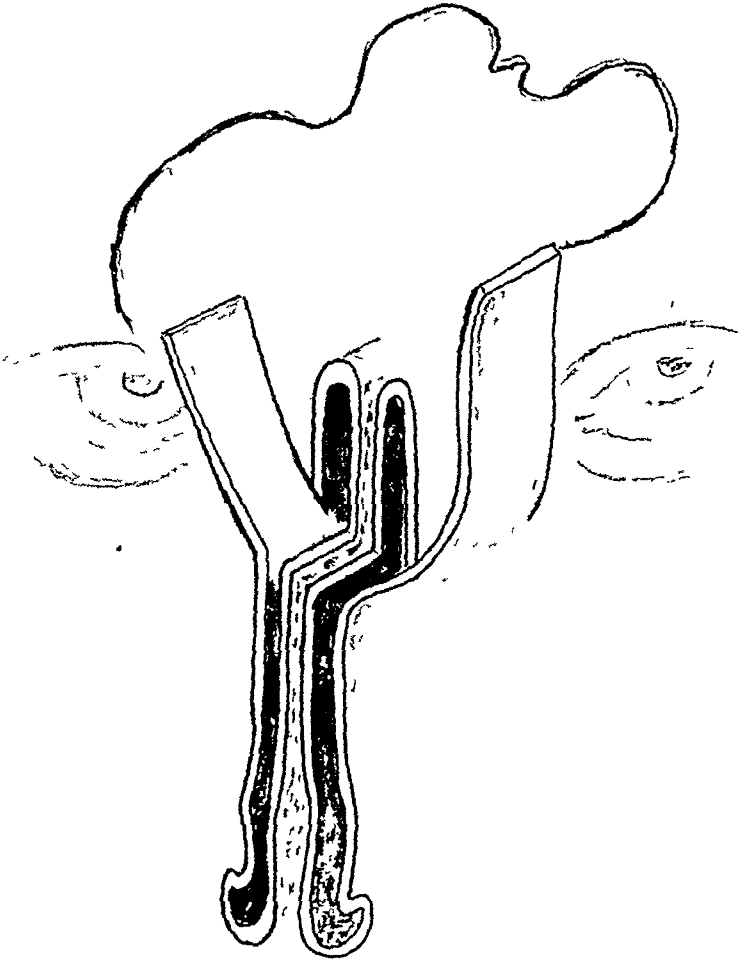


Fig 3 Final position of bilateral septo-mucoperiostial flaps (modified after Tito)

vault with intact mucous membrane flaps lining both the lateral walls of the funnel On the involved side the flap is sutured once or twice to the peri-orbita On the opposite side the flap is just laid into position

Two large rubber catheters are inserted through the nose, up through the new funnel opening into both frontal sinuses These help hold the flaps in position They are also held by nasal packing and by a heavy silk suture passing through the catheters and over a button on the forehead This is left in for at least three weeks The nasal packing can be removed in two to four days

VARIATION OF TECHNIQUE

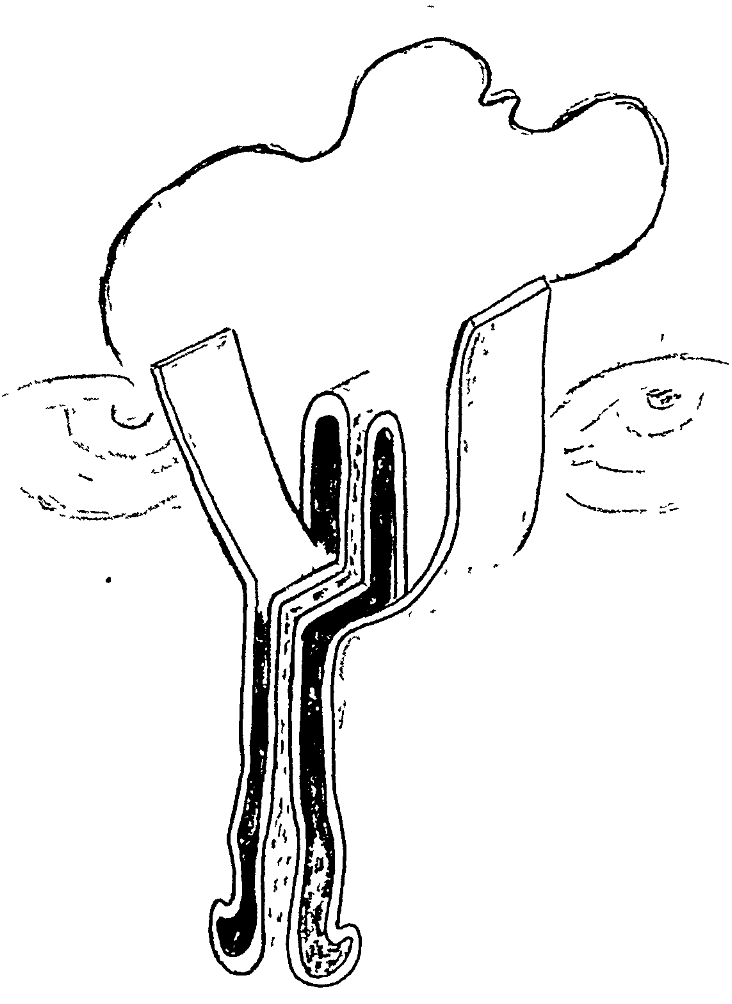
Once the frontal floor has been opened the sinus is examined and the area of the nasofrontal duct inspected This area is usually markedly involved with granulation tissue and scarring, thus requiring the creation of a new duct Such cases are frequently bilateral It is best to remove all the frontal floor to prevent pocket formation laterally

Van Alyea and others have advised leaving mucous membrane in the sinuses when irreversible changes have not occurred, providing adequate drainage is possible The mucosa is not completely removed routinely, only that portion showing what is considered irreversible changes, such as intra-mucosal abscesses and large amount of granulation tissue, etc

We have left inactive diseased mucous membrane, such as mucocele lining over posterior wall erosion, on the assumption that, with a new functioning nasofrontal duct which provides permanent drainage, the mucosa would return to normal, and the follow-up has borne out this assumption

If the supra-orbital margin has been eroded anteriorly the edges are smoothed with rongeurs When the posterior wall has been destroyed and the dura is exposed this area is examined for extra-dural abscesses

The anterior end of the middle turbinate usually marks the posterior extent of the frontal floor in the midline Hence in preparation of the mucosal flaps dissection anterior to this point clears the cribriform plate (see Fig 1) The anterior ethmoids only are entered in fashioning the bilateral flaps A complete ethmoidectomy is not routinely done nor is a sphenoidectomy, however, the anterior ethmoid cells deep or lateral to the flap must be well exenterated and the anterior part of the middle turbinate freed and laterally displaced to form a smooth bed for the flaps Obvious disease is of course,



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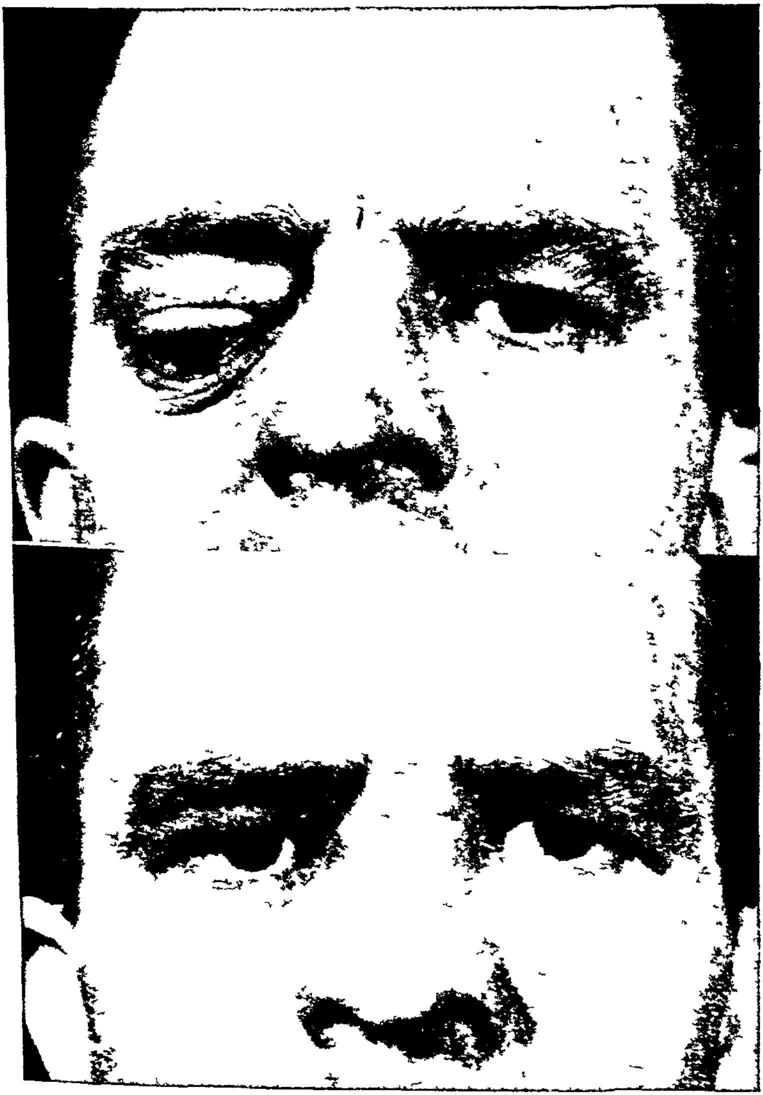

Fig 4 Pre- and postoperative photograph of patient

removed When disease is present in the ethmoids and sphenoids *on the* operated side, these are thoroughly cleaned from above When these are present on the opposite side also a *De Lima* transantral ethmoidectomy without frontotomy is done, thus not disturbing the mucosal flap When disease is present in the antrum, on the operated side a Caldwell-Luc procedure is performed These procedures can be done either before or after the frontal operation or as a combined procedure

CLINICAL FINDINGS

In a series of 21 patients, 20 presented the usual subjective and objective findings of chronic obstructive frontal sinusitis such as *1* bone destruction, *2* fistulae, *3* adjacent orbital signs

Seventeen of the cases had bone destruction of the floor of the frontal sinus by disease Three more had a floor defect due to a previous Lynch procedure One of the original 17 had had a previous Lynch operation but had had floor destruction by disease at that time

There were three cases with a cutaneous fistula

Only one of the 21 did not have a bone defect at the time of the mucoperiosteal flap operation

Fifteen patients had globe displacement Six of these presented a soft, slightly tender, cystic swelling just above the inner canthus of the eye as in the classic description of mucocele (see Fig 4) The remaining nine had a previous orbital cellulitis and presented themselves with an edematous, indurated upper lid in various stages of resolution One of these had left frontal sinusitis which resolved on treatment elsewhere with antibiotics Two months later he developed blindness in the left eye associated with local pain and swelling of the forehead and upper lid A diagnosis of optic atrophy secondary to left frontal sinusitis was made when he was first seen in our clinics three weeks later

There was one incidence of orbital displacement with ophthalmoplegia and pallialedema but normal vision

RESULTS

In this series of 21 patients careful follow-up examination was possible in 20 from one to ten years One has been lost to follow-up All but one have been free of any complications One of the twenty had an episode of mild swelling of one eyelid in the postoperative period She had had a previous Lynch type external frontal operation which had been followed by recurring orbital swelling soon afterwards, requiring surgery She had no complications in the later followup On examining her recently the nasal passage to the right frontal is open but narrow instead of being wide and funnel-shaped at the root of the nose We think this is the reason for the less than optimal result

Two have had occasional mild headaches on the side of original pathology In all cases the new nasofrontal duct has remained open and functioning during upper respiratory infections During colds their complaints are no more severe than those of other normal people with the same temporary infection

We feel that in our series we were successfully able to create a new functioning nasofrontal duct in all cases Boyden[2] has published a much larger series of unilateral flap cases with a longer follow-up, and his results prove that the mucosal flap technique is very satisfactory We feel that the bilateral flap method permits a much wider opening to be made with less chance of narrowing during upper respiratory infections

Inactive mucosa lining a frontal sinus, as with mucocele, need not be entirely removed There are many who would wonder at the advisability of leaving mucocele lining which appears thicker than normal Actually this lining was left since we have felt a patent new nasofrontal duct is more important than the removal of mucosa that appears thicker than normal Obviously, we do not advocate leaving intramucosal abscesses or infected lining, however, meticulous excision of all mucosa while theoretically advisable seems to be not necessary at least in our mucocele cases Return to a "normal" inactive lining apparently occurs

In another, a large osteoma involving the floor of both frontals, the lacrimal bone, ethmoid labyrinth and front face of the sphenoid on the right was the cause of nasofrontal duct obstruction and orbital cellulitis

Of the four patients who had a previous Lynch type operation, one developed a cutaneous fistula, one developed orbital abscess with some globe displacement, and two had recurring swelling of the upper lid which subsided on antibiotics

Only one of the total number had persistent local discomfort resisting conservative management as the sole indication for surgery

Bony destruction of the posterior wall of the sinus or the medial wall of the orbit occurred only in the presence of a destroyed frontal floor in this series In every case, at surgery the nasofrontal duct was found to be obliterated by disease or markedly narrowed

SUMMARY OF CLINICAL FINDINGS

Previous Surgery—	
Frontal floor trephine	2
I & D Periorbital abscess	4
Lynch type external frontal	4
Intranasal ethmoidectomy	1
Associated Surgery—	
Caldwell Luc	3
Transantral ethmoidectomy	3
Bilateral Disease	8
Unilateral Disease	13
Etiologic and Pathologic Findings—	
Infection with orbital displacement	9
Mucocele with orbital displacement	6
Allergy	2
Osteoma	1
F B Shrapnel	1
Fracture with infection	2
Bone Destruction	
Floor of frontal—by disease	17
Floor missing previous Lynch	4
Floor missing at first Lynch	1
Posterior wall	6
Orbital Displacement	18
Functional Ocular Symptoms	7
Cutaneous Fistula	3
Total Cases	21

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UNIVERSITY OF TORONTO—POSTGRADUATE COURSE IN OTOLARYNGOLOGY

On May 11, 12 and 13, 1961, there will be presented a graduate course in this specialty by the staff of the Department of Otolaryngology, assisted by two distinguished guests Dr Philip E Meltzer, Professor of Otolaryngology, Harvard Medical School, Chief of Otolaryngology, Massachusetts Eye and Ear Infirmary, and Dr W G Hemenway, Department of Otolaryngology, University of Chicago

The first session will begin in the afternoon of May 11th, in the Royal York Hotel, Toronto, in conjunction with the Section of Otolaryngology of the Ontario Medical Association The remainder of the sessions will be held in the clinical areas of the University of Toronto

An attempt will be made to assess, discuss and demonstrate the newer procedures employed in the surgery of deafness The present surgical treatment of head and neck problems will be presented with special consideration of the new conceptions of the responsibilities of our specialty in their management

The fee for the course will be $40 00 and will include a complimentary dinner

Please address all inquiries to the Director, Division of Postgraduate Medical Education, University of Toronto

SUMMARY

A method of reconstructing the nasofrontal duct is presented It is based on the work of Boyden and Tato and consists of the rotation upwards and laterally of mucosal flaps fashioned from the lateral wall and septum of the nose A series of 21 cases is discussed There were 17 cases with destruction of the bony walls of the frontal sinus, 15 with displacement of the globe and three cases of cutaneous fistula Mucocele lining has been left without any follow-up complication The mucosal flap technique was used in all cases and was bilateral in 17 and unilateral in four In all cases, the method successfully reconstructed a functioning nasofrontal duct

CONCLUSION

The common cause of failure in surgery of the frontal sinus is obstruction of the nasofrontal duct The use of bilateral septoperiosteal mucous membrane flaps from the nose is a very satisfactory method of reconstruction of the nasofrontal duct Admittedly our series is small, but short term results, from one to ten years, appear satisfactory

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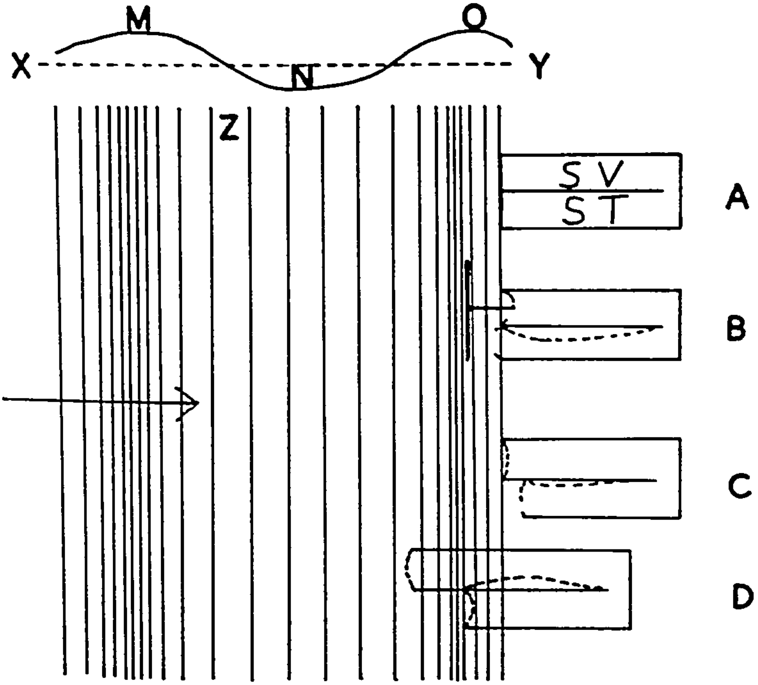

Fig. 1. A—This is a box representation of the cochlea (v. Bekesy) as applied to the approaching pressure peak. SV—scala vestibuli. ST—scala tympani. The oval and round windows are at the left and the helicotrema at the right. The basilar membrane with the organ of Corti is indicated by the line between SV and ST. B—A conduction mechanism placed in contact with the oval window creates pressure differential at the two windows. The pressure is greater at the oval window and the structures move toward the round window as indicated by the dotted lines. C—The usual anatomical relationship is for the oval window to be closer to the pressure peak than is the round window. Limited movement of the cochlear structures is indicated by the dotted lines. D—A fraction of a second after C the pressure peak is at the round window and a reversal of displacement of the structures is indicated.

which can be propagated through any deformable medium. Sound spreads with a circular front in all directions from its source. Sound energy is gradually consumed and dissipated by the inertia of the medium through which it passes, plus the fact that it spreads over an overwhelming area. While sound is usually represented as a curve (see M-O, Fig. 1), it actually is a succession of fronts of positive and negative pressure changes (compression—M, rarefaction—N) with relation to a point of equilibrium. This point of equilibrium is represented in Fig. 1 by the dotted line XY on the curve and at

CLINICAL ASPECTS OF TYMPANIC SOUND CONDUCTION*

ARTHUR L JUERS, M D ,

Louisville, Ky

Recent advances in the surgical restoration of effective tympanic sound conduction make it necessary that the otologist has a full understanding of the normal, the pathological and the surgically reconstructed sound conduction mechanism The advent of fenestration surgery provided the initial impetus to understand better the mechanisms of sound transmission to the inner ear No attempt will be made here to review the complete evolution of this knowledge to date Particular credit for experimental and theoretical contributions should go to v Bekesy,[1] Wever and Lawrence[2] Lempert,[3] Wullstein[4] and Zollner[5] have made contributions on the clinical aspects of the subject The author has recorded some clinical observations and theoretical considerations, and these previously published ideas will be used together with some additions[6,7] Many details concerning the mechanics of tympanic sound conduction were presented by Lawrence and Wullstein at the Workshop for Functionally Reconstructive Ear Surgery presented at Northwestern University by Shambaugh in March, 1959[8]

Four diagrams will be used to show the fundamentals of effective sound transmission to the inner ear under normal, pathological and surgically reconstructed conditions No attempt is made to conform to normal anatomical relationships Actually, the wave lengths of audible sound frequencies are much longer in relation to the cochlea than is indicated in these diagrams

PHYSICS OF SOUND

Sound can be defined as an audible vibratory disturbance

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*Presented at the meeting of the Indianapolis Ophthalmological and Otolaryngological Society Nov 12 1959

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with the oval window This accomplishes two things the wide surface (top of the T) "collects" sound pressure from a wide area and transmits it to the oval window, this collected sound has entered a solid medium, the conduction mechanism, which conducts sound at a much greater speed than is true of the sound which will reach the round window by air As a result of these two factors, the pressure differential in favor of the oval window moves the cochlear fluids and basilar membrane structures toward the round window as indicated by the dotted lines

V Bekesy[10] has shown that the oval window is usually slightly lateral to the round window in relation to the axis of the external auditory meatus This is indicated in C, Fig 1 Consequently, even in the complete absence of a tympanic membrane and ossicles, there is some pressure differential at the two windows and some hearing by air conduction The relative laterality of the two windows is somewhat variable D, in Fig 1, shows the reversal of cochlear fluid movement when the pressure peak has passed the oval window and has reached the round window

NORMAL CONDUCTION MECHANISM

The following functions can be listed for the normal conduction mechanism

1 Areal ratio

2 Round window sound protection

3 Lever action

4 Some limitation of excessive excursion in response to loud sounds

The chief function of the normal conduction mechanism is to provide what is referred to as the areal ratio The larger surface of the tympanic membrane "collects" sound pressure which is then transmitted to the smaller stapes footplate through the ossicular chain (see Fig 2). The areal ratio is the ratio of the surface area of tympanic membrane to that of the stapes footplate The effective, mobile area of the tymthe point Z in the vertical lines M-N represents a single vibration of a tuning fork and M-O a double vibration (d v)

A full cycle of sound is indicated from M to O, or 360 degrees The term phase is used to designate a particular point in the cycle Phase difference is the difference in degrees between two points of the cycle From M to N would constitute a 180-degree phase difference

The physical qualities of a medium determine the speed with which sound is propagated through it and also influence the rapidity of absorption of sound energy (decrease in intensity) Sound traverses air at approximately 1100 feet per second, water at 4700 and aluminum at 16,700 This speed differential has some significance in normal tympanic sound conduction as well as in pathological changes

Alterations in elasticity and/or mass of the conduction mechanism are of particular importance in clinical otology[9] Any increase in elasticity tends to absorb more energy from the lower than from the higher frequencies Lesions which fall in this category are early stapes fixation and adhesive limitations of ossicular motion Since low frequencies of a given intensity normally produce greater amplitude of ossicular motion than do the higher frequencies, it is apparent that a slight limitation of the degree of ossicular excursion will impede sound conduction for low frequencies more than that for higher frequencies

Fluid in the middle ear with an otherwise normal conduction mechanism tends to cause greater loss for the higher frequencies The fluid adds mass to the conduction mechanism As each cycle of sound passes through, the inertia of the total mass—conduction mechanism and fluid—must be overcome twice, consequently, the energy required to overcome this inertia in a given period of time, is greater for the higher than for the lower frequencies

In A, of Fig 1, the pressure at the two windows is equal, and there is no movement of the basilar membrane Bone-conducted sound is not considered here

In B, of Fig 1, a conduction mechanism is placed in contact with the oval window This accomplishes two things the wide surface (top of the T) "collects" sound pressure from a wide area and transmits it to the oval window, this collected sound has entered a solid medium, the conduction mechanism, which conducts sound at a much greater speed than is true of the sound which will reach the round window by air As a result of these two factors, the pressure differential in favor of the oval window moves the cochlear fluids and basilar membrane structures toward the round window as indicated by the dotted lines

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As the sound pressure peak moves the tympanic membrane inward and transmits this movement to the stapes as indicated by the confluence of lines and arrows (see Fig 2), there is at the same time some sound passing into the air of the tympanic cavity This is dissipated in all directions, going in part to the stapes via air as well as to the round window In view of this, as well as the fact that the speed of the ossicular conducted sound greatly exceeds that reaching the round window membrane via the air of the tympanic cavity, the round window protection factor of the tympanic membrane is relatively unimportant in a normal ear This perhaps accounts for the fact that a small tympanic perforation in a normal ear will cause relatively little loss of air conduction acuity

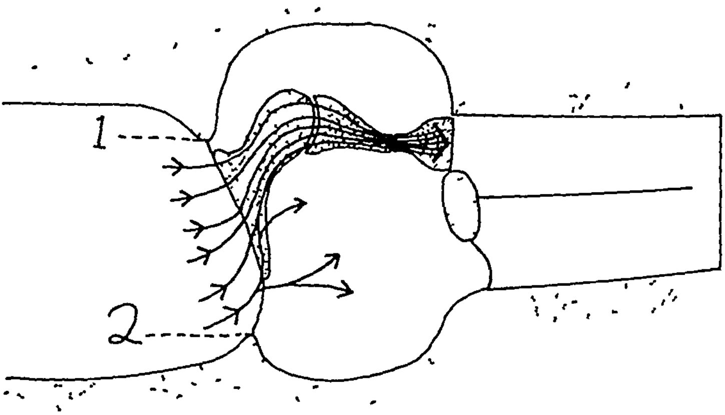


Fig 2 The mobile portion of the tympanic membrane is between 1 and 2 Sound pressure against this portion of the membrane is transmitted through the ossicular chain as indicated by the lines and arrows A small amount of sound pressure passes into the air in the tympanic cavity It is obvious that the pressure transmitted through the stapes greatly exceeds that which passes through air in the tympanic cavity to the round window

The lever action of the ossicular chain adds only about 2 5 db to the hearing acuity The ligaments and muscles attached to the ossicular chain serve chiefly to limit to some extent excessive excursions of the conduction mechanism This undoubtedly lessens inner ear damage which might otherwise result from some types of loud sound

PATHOLOGICAL CONDUCTION MECHANISM

The four chief mechanical changes found in conduction lesions are as follows. *1* Increase in elasticity, *i e*, limitation of excursion *2* Increase in mass *3* Loss of a portion or all of the tympanic membrane *4* Interruption of ossicular chain continuity These lesions may be present individually or in combination Certain features of pure tone air conduction audiometric curve configurations are characteristic of some individual types of lesions Combinations of lesions produce a summation of these characteristics Variations in the extent of the air-bone gap when comparing low tones and higher tones are sometimes apparent and suggest a certain type of lesion

Increase in Elasticity

The most common pathology producing this type of conduction change is either otosclerotic fixation of the stapes or ossicular fixation by adhesive otitis The earliest shift in the pure tone curve usually shows more loss for the low frequencies than for the high, if there is no impairment of cochlear function In earlier lesions the air-bone gap tends to be greater for the lower than for the higher frequencies This could be designated as a converging air-bone gap (going from low to high frequencies) As the fixation becomes complete, the air conduction curve usually becomes more flat The type of air conduction curve associated with adhesive middle ear changes tends to be somewhat variable but in general shows greater low tone loss in less severe lesions

Increase in Mass

Middle ear effusion is the most common cause of this type of conduction loss The audiometric curve here shows greater loss for the higher frequencies The air-bone gap tends to diverge when comparing the lower and higher frequencies

If the effusion is extremely tenacious and completely fills the tympanic cavity and attic area, the curve may be more of a flat type because the nature and extent of the effusion has added an elasticity factor to that of mass This is the "glue-ear" described by Jordan A middle ear effusion superimposed on a pre-existing partial ossicular fixation may summate into a flat curve

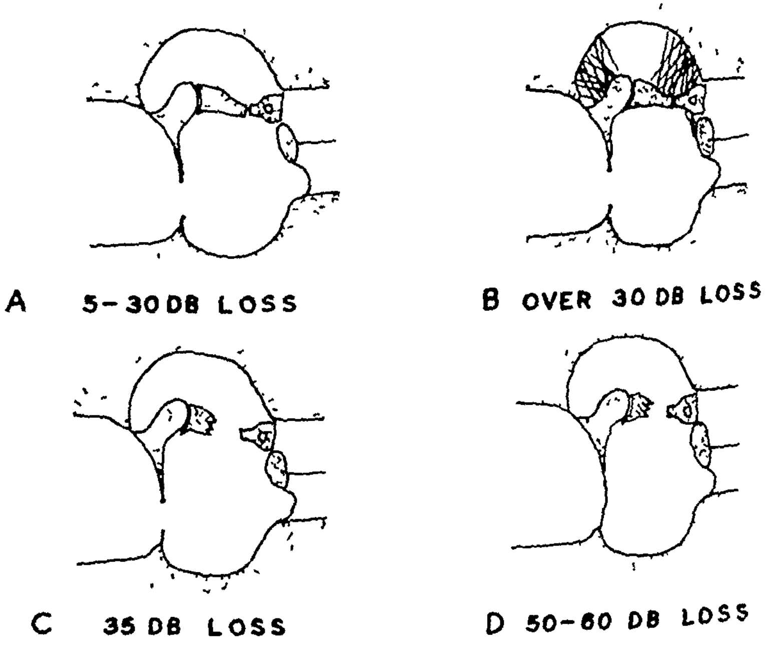


Fig 3 A—A central perforation with normal ossicular function and windows B—A central perforation with some adhesive impedance of ossicular motion C—A central perforation with interruption of ossicular chain continuity D—Intact tympanic membrane with interruption of ossicular chain continuity

The db loss indicated for each of the lesions in these diagrams is the usual average There are occasional deviations which may be due to anatomical and physical variations in the window areas

Pars Tensa Perforation

Fig 3-A shows a perforation in an ear with a normal ossicular chain and round window The air conduction loss will vary from no significant shift for small perforations to a maximum of 30 db loss for larger ones Variable anatomical factors such as relative laterality of the oval and round windows, overhang of posterior-superior bony meatus over stapes area, axis of external meatus to cochlea could conceivably alter the air conduction threshold

Fig 3-B shows some adhesive changes associated with a tympanic perforation Whenever the air-bone gap exceeds 30-35 db in a perforated ear, the possibility of ossicular impedance or window pathology is likely

Interruption of Ossicular Continuity

When there is an interruption of ossicular continuity associated with a sizeable central perforation, the air-bone gap is in the vicinity of 35 db Variations in window relationships previously mentioned would influence the threshold If the oval window recess is extremely narrow, *i e*, only the capitulum of the stapes is visible between the promontory and the facial nerve, then the sound pressure differential between the two windows would undoubtedly be less favorable

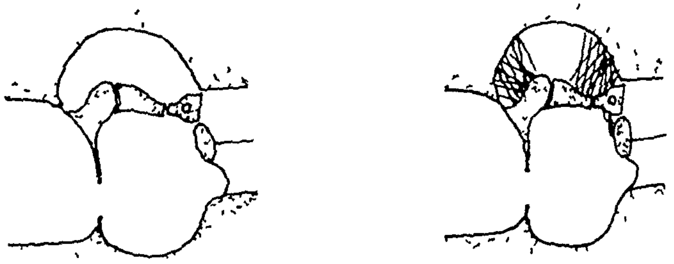
Interruption of Ossicular Continuity and Intact Tympanic Membrane

(See Fig 3-D) The tympanic membrane here excludes about 15-20 db of sound pressure and the air-bone gap is 50-60 db This points out the advisability of determining by means of a paper patch placed on the perforation whether closure will improve or further decrease hearing While interruption of the ossicular chain in the presence of a perforation is uncommon, closure of such a perforation could be embarrassing if such a situation has not been anticipated Interruption of ossicular continuity in an ear with an intact tympanic membrane is most commonly the result of previous surgical trauma or head trauma This should be particularly suspected if the air-bone gap is 50-55 db and there is a history of previous mastoid surgery or head trauma[11]

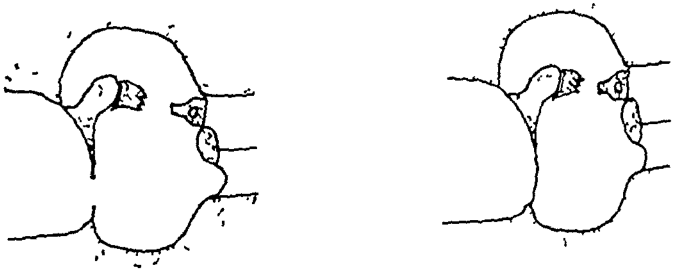
SURGICALLY RECONSTRUCTED CONDUCTION MECHANISM

The conduction physiology of a fenestration of the lateral semicircular canal is shown in Fig 4-A The tympanic membrane here plays a different primary function as compared to that in a normal ear The absence of a favorable areal

If the effusion is extremely tenacious and completely fills the tympanic cavity and attic area, the curve may be more of a flat type because the nature and extent of the effusion has added an elasticity factor to that of mass This is the "glue-ear" described by Jordan A middle ear effusion superimposed on a pre-existing partial ossicular fixation may summate into a flat curve



A 5-30 DB LOSS B OVER 30 DB LOSS



C 35 DB LOSS D 50-60 DB LOSS

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ing air conduction loss would not be due to a mass effect added to the conduction mechanism The tympanic effusion in such an ear would rather serve to conduct sound to the round window with greater speed and hence decrease pressure and phase difference between the two windows

In myringostapediopexy (see Fig 4-B) the tympanic membrane plays a variable role as far as providing an effective areal ratio is concerned It is usually technically difficult to arrange the structures to permit establishing a 14-1 areal ratio This is particularly true in ears which have extensive pathology due to cholesteatoma or chronic infection necessitating a modified radical mastoidectomy In such cases the sound protection of the round window becomes a more important function of the tympanic membrane than is true in the normal middle ear

If the conduction lesion is due to an absence of the incus and there is no middle ear or attic disease, then removal of some of the posterior superior bony meatus and the creation of a myringostapediopexy may provide a good areal ratio and closure of the air-bone gap Shambaugh has described such cases[11]

An effusion in an ear with a myringostapediopexy would decrease hearing by two possible methods If a good areal ratio is present, the fluid would exert more of a mass effect If there was not a good areal ratio, then the fluid would decrease hearing more through the phase difference decrease effect

SUMMARY

1 The major function of the normal tympanic conduction mechanism is to provide a favorable areal ratio between the tympanic membrane and the stapes Lesser functions are to provide sound protection to the round window and a lever action

2 Early elasticity and mass lesions produce different air conduction curves with converging and diverging air-bone gaps respectively

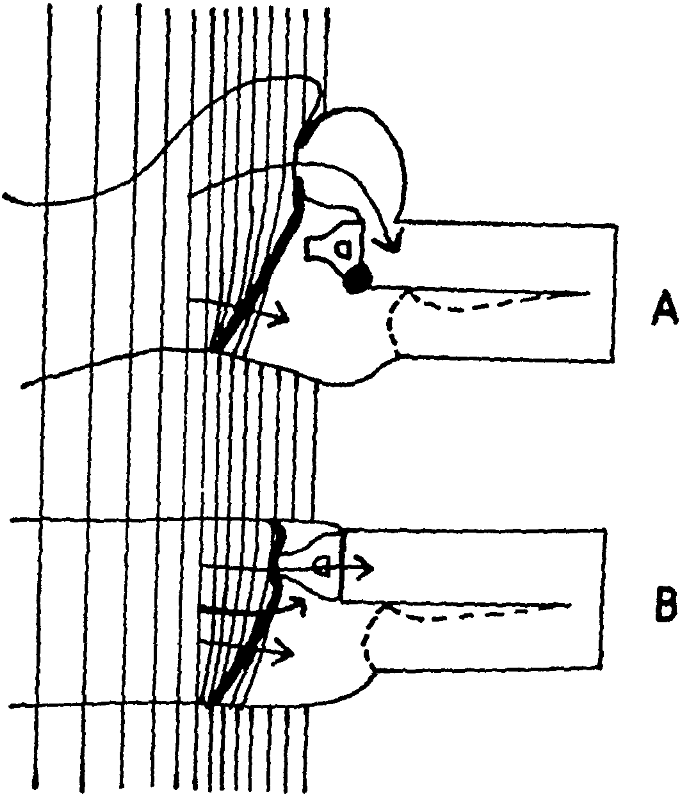

Fig 4 A—In a fenestrated ear the pressure peak of a sound wave enters directly through the fenestra in the lateral semicircular canal as indicated by the top arrow The tympanic membrane serves primarily to shield the round window This increases the pressure differential at the two windows The stapes is fixed and does not participate in sound conduction B—In a myringostapediopexy the tympanic membrane (or skin graft) is in contact with a mobile stapes The sound pressure peak is conducted through the stapes A stapes which is recessed between the promontory and facial nerve would be less favorable to the reconstruction of a good areal ratio than would a stapes which is more exposed

ratio accounts for the residual "conduction deficit" or air-bone gap of 20-25 db When the pressure peak approaches the ear, it enters the fenestra first The tympanic membrane serves to shield or protect the round window somewhat from sound pressure This increases the pressure differential between the fenestra and the round window The sound which enters the fenestra travels more rapidly in the fluid of the labyrinth to the basilar membrane than does that which passes through the air of the tympanic cavity toward the round window If there is fluid in the tympanic cavity, the result-

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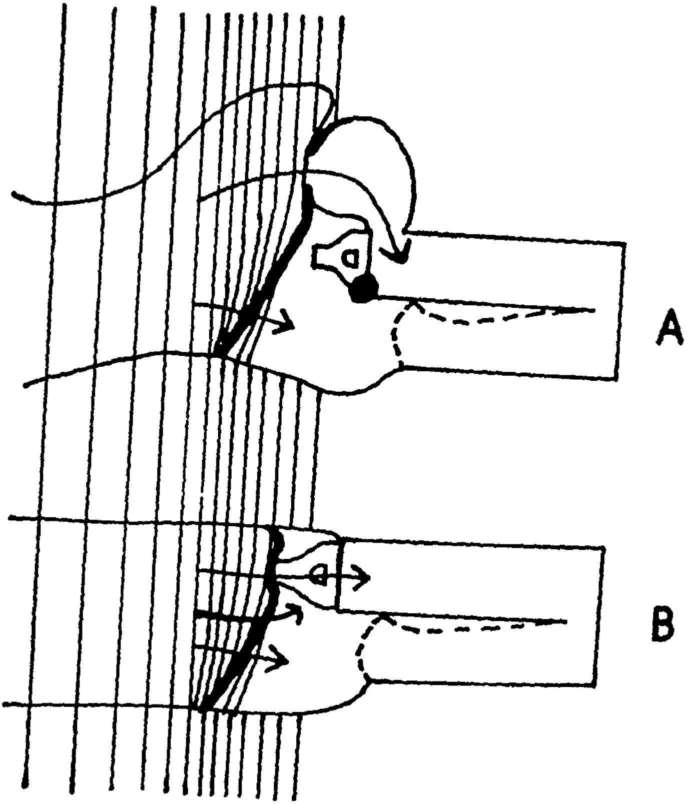

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2 Early elasticity and mass lesions produce different air conduction curves with converging and diverging air-bone gaps respectively

3 In a fenestrated ear and in a myringostapediopexy with a low areal ratio, the round window sound protection function of the tympanic membrane assumes greater importance

4 In an ear with a good areal ratio, thin middle ear effusion decreases hearing chiefly by its mass effect In an ear with a low areal ratio, middle ear effusion decreases hearing chiefly by its phase difference decreasing effect

5 In an otherwise normal conduction mechanism, tenacious middle ear effusion filling the entire middle ear will decrease hearing because of its mass effect, elasticity increase, as well as phase difference decreasing effect

6 Tympanic perforation in an otherwise normal conduction mechanism causes a hearing loss primarily because of a decrease in areal ratio Loss of round window protection is of secondary importance in this situation

7 An unusually large air-bone gap in the presence of an intact tympanic membrane is suggestive of ossicular chain interruption, particularly if there is a history of previous mastoid surgery or head trauma

8 The total displacement of the cochlear structures is dependent on the summation of several factors which influence maximum pressure differences possible at the inner surface of the two windows These factors include the elasticity and mass of the conduction structures, areal ratio and phase difference increase or decrease effect of structures and media through which sound passes en route to the two windows

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CRICOTHYROID DISLOCATION*

JOHN S KNIGHT, M D,

Kansas City, Mo

This paper was prepared to report the results obtained from the study of nine patients with cricothyroid dislocation

Review of previous work and literature revealed that dislocations of the cricothyroid joint were first considered to be a common condition but not recognized according to Jackson and Jackson[1] They state that dislocations of the cricothyroid joint can be traumatic or habitual The laxity of the ligaments and the habitual manipulation of the patient have something to do with this phenomenon The first dislocation may occur during yawning or wide opening of the jaws or from external trauma of any form The displacement is of the cricoid backward relative to the thyroid

Appearance of the larynx shows nothing abnormal except possibly some feebleness of tension The symptoms of stiffness, local tenderness and slight hoarseness are present Thereafter the only symptom complained of was the snapping in and out of place, or the frequent necessity for replacement with the finger

The treatment does not call for urgent handling If the patient is constantly annoyed and demands relief, the joints may be invaded surgically to produce ankylosis Dr Chevalier Jackson has operated on one case with a successful result The prognosis indicates that it is not a condition endangering life, and the voice is impaired only slightly The usual sequel is a habitual dislocation The patient can dislocate the joint at will, and frequently it dislocates itself to the annoyance of the patient

The above comments are from foremost authorities, and it

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*Submitted as Candidate s Thesis to the American Laryngological Rhino logical and Otological Society Inc 1960

Editor s Note This manuscript received in The Laryngoscope Office and accepted for publication Jan 22 1960

is not intended to belittle or change their statements, however, experience has shown that the condition as stated above can be classified as subluxation of the cricothyroid joint In the cases to be presented, a situation has been found that is far more difficult to handle than the mere manipulation of the voice box by the patient Accordingly, one might presume that a complete dislocation was encountered in a small series of cases, and in two cases an ankylosis also was noted The dislocation of the inferior cornu has been posterior to the cricoid

Further study of the literature reveals that dislocations of the cricothyroid articulation have been exceedingly rare, according to Jackson and Coates[2] in 1929 Since that time several articles have appeared in the literature Kernan and Cracovaner[3] in 1942 described dislocation of cricothyroid joints as rare conditions Jackson and Jackson,[4] in 1943, described the case of a man who, in an attempt to hang himself, dislocated both arytenoid cartilages and the cricothyroid articulation Elsbach[5] reported a case of dislocation of the thyroid cartilage in 1948 Gisselsson[6] commented that, judging from the literature, dislocations of the larynx were very rare, indeed He reported a case in 1950 In 1951, Loeb[7] presented eight patients with cricothyroid subluxation and described such symptoms as pain of the larynx upon swallowing, referred ear pain, and a feeling of a lump in the throat He noted that there was displacement of the thyroid cartilage either to the right or left By employing manipulation of the subluxed joint, he was able to effect dramatic relief of pain Surgical correction was not necessary to obtain a clinical improvement

The articulation of the lesser horn of the thyroid cartilage with the cricoid is a synovial or true joint This is essentially necessary for proper and complete approximation of the vocal cords The thyroid cartilage is movable anteriorly and posteriorly to a mild degree

Binding the thyroid cartilage to the cricoid anteriorly is the middle cricoid ligament Surrounding the inferior cornu of the thyroid cartilage with the junction of the cricoid cartilage is a thin lax ligament, the cricothyroid capsule This capsule is assisted or reinforced by the lateral and posterior ceratocricoid ligaments, these acting as a partial check to the movement

Disturbance of the normal anatomical and physiological relationships of this joint result in certain changes of voice, neck comfort, and ability to swallow

In the 1945 edition of Jackson and Jackson's[8] "Diseases of the Nose, Throat and Ear" the subject of trauma of the larynx is discussed quite thoroughly "Laryngeal injury may be direct when inflicted by an external agent, or indirect when due to action of the laryngeal muscles In no part of the literature of traumatology is there such a dearth of data as in wounds of the larynx Most of the cases are buried in the classification of injury of the neck"

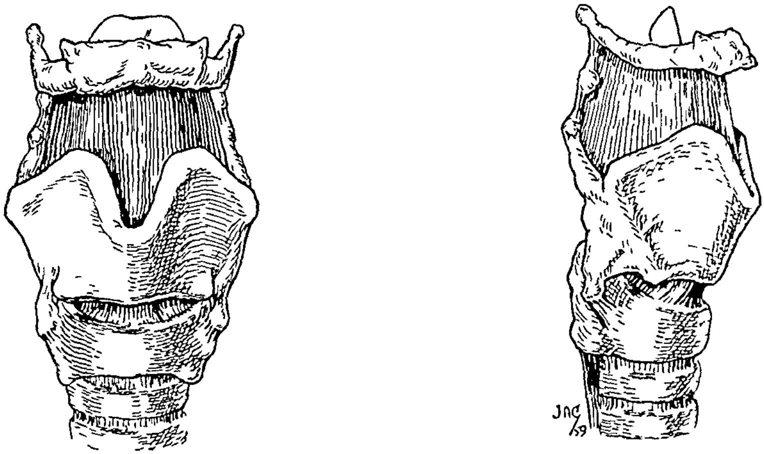


Fig 1 Normal laryngeal structures

Holinger[9] and Proud[10] state that they have not had a recognized case of cricothyroid dislocation in their practice Lejeune[11] has reported that his experience with cricothyroid dislocations is limited to three or four cases

Much has been written about the dislocation of one or both arytenoid cartilages, and it is not the purpose of our paper to discuss this dislocation but to confine our comments to cricothyroid dislocation

Twenty-nine years ago the author had the privilege of seeing six cases of cricothyroid dislocation of the larynx in a period of one year Since that time he has not seen or known of any

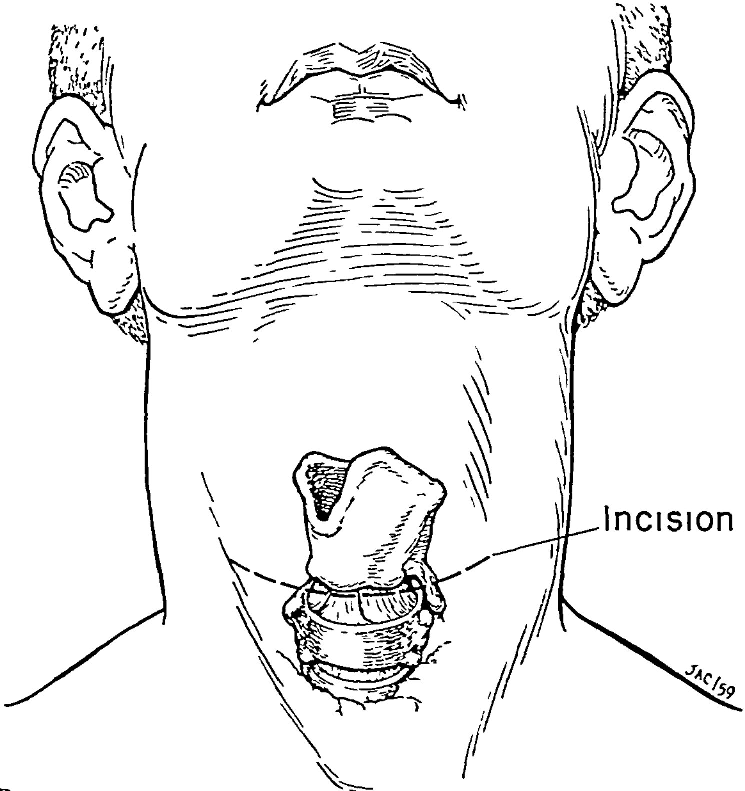


Fig 2 Cricothyroid dislocation showing displacement of larynx and thyroid notch laterally

similar cases until the past three years In this period, two case reports are presented involving cricothyroid dislocation In each case the dislocation was associated with severe automobile injuries affecting other parts of the body The dislocations were not diagnosed until four months later in the first case and one month later in the second case A third case of dislocation was associated with multiple fractures of the larynx resulting from a blow by a fist

Case 1 Mr H D, a lawyer, age 38, developed his first dislocation in 1929 while leaning back in his chair, turning his head to the side and talking at the same time He suddenly developed pain and discomfort in the neck and the left side of the face He came under observation almost immediately The voice box was rotated to the left, and the pain was referred to the left ear Efforts at swallowing resulted in a great deal of pain, and there was a huskiness to his voice The thyroid notch was displaced at least 1 cm to the left of the midline of his neck The right side of his voice box was flattened Indirect laryngoscopy revealed a change of approximately 30 degrees from the midline to the left of the anteroposterior dimensions of his glottis (see Fig 3)

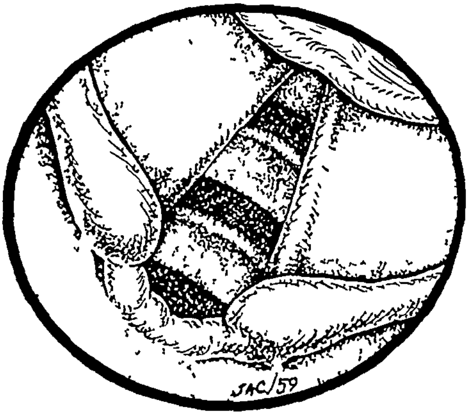


Fig 3 Indirect laryngoscopy appearance

Efforts to reduce the dislocation by manipulation were not successful, and the patient was given an ether anesthetic It was then very simple to relocate the voice box by manipulation When the patient awakened he was comfortable This patient has been seen at various times since his original disability He says that he is always quite conscious of the act of swallowing, and that he dares not put his head back and talk at the same time In the course of the past years he has had approximately 20 dislocations of his larynx which he was able to correct himself by inserting his finger into his mouth and pulling upward and forward on the base of the tongue

Case 2 Mr J J G, age 55, was seen in the same year with the possibility of having swallowed a foreign body in chicken pie He seemed quite frightened and very uncomfortable He did not remember pain or discomfort that might have occurred as the result of ingestion of a foreign body Pain and discomfort particularly on swallowing and with reference to the ear were present The thyroid notch was displaced to the left side, and indirect laryngoscopy revealed no foreign body but an angulation of the glottis By manipulation, which included anterior elevation of the base of the tongue and epiglottis with a finger, it was possible to reduce the dislocation with a sudden snap, and the patient immediately became comfortable, stating that something had slipped back and he felt better Indirect laryngoscopy showed normal laryngeal find ings. There has been no follow up in this man's case, and we assume that he did not have any further trouble.

Case 3. Mr. F. S., age 60, was an extremely nervous and hypertensive type of individual when first seen in consultation with the complaints of acute discomfort of the throat and referral of pain to his left ear. While he was eating he had turned his head quickly to the left on hearing his name called. The thyroid notch was displaced to the left, and indirect laryngoscopy showed the glottic chink to be angulated 25 to 30 degrees off the midline. He was given a general anesthetic and the dislocation was reduced rather easily. His case was followed for a number of years, and there was no evidence of any recurrence.

Case 4. Dr. T. E., a 25-year-old intern, developed a dislocation with similar findings and complaints that have been described. He was a tall thin individual. He stated that his first dislocation occurred three years previously after a visit to the dentist. Spontaneous recovery developed in about five minutes. Three or four times a year since then his larynx would dislocate. He learned to relocate his larynx by manipulation. The complaints of sudden onset of pain referred to his ear with difficult, painful swallowing and a lump in the throat were associated with the findings of a displaced thyroid notch and angulation of the glottic chink. By manipulation of the larynx, with particular attention to the maneuver of lifting anteriorly on the inferior cornu, a satisfactory anatomical reduction was accomplished with subsequent remission of symptoms. This patient was last seen in 1931, at which time there was no history of recurrence.

Case 5. R. A., age 13, was choked from behind by a classmate on a school playground and immediately afterward was noted to be quite short of breath, with difficulty in speaking. His mother, a registered nurse, recognized that a potentially dangerous injury to the voice box had been sustained and sought medical attention without delay. On examination the boy revealed symptoms of referred pain to the right ear aggravated by swallowing, difficulty in swallowing, moderate shortness of breath, and a change in voice with signs of a displaced larynx and the displacement of the thyroid notch to the right. Direct laryngoscopy revealed a displaced glottic space with angulation to the right, poor mobility of the vocal cords, and submucosal hemorrhage involving the right cord and arytenoid. The glottic space was sufficiently narrowed to warrant an elective tracheotomy under local anesthetic prior to manipulation and realignment of the larynx under general anesthetic. It was considered possible that this patient had suffered a fracture of the thyroid cartilage in addition to the dislocation, however he made an uneventful recovery and was decannulated within a period of ten days. For approximately six months following surgery some difficulty in developing normal speech was noted, but after this time his voice became normal and there was no discomfort on swallowing.

Case 6. F. D., a 26-year-old robust full-necked athletic type of man, was seen in 1930 with complaints of neck pain and difficulty in swallowing following a sudden turn of the head while sitting at his office desk. He stated, "I felt as though someone had put a knife through my throat, but now the pain is behind my Adam's Apple." He did not complain of referred pain to the ear. The voice was slightly husky, there was a feeling of a lump in the throat and an almost complete inability to swallow. There was sufficient shortness of breath to require resting three times during the four block walk to the physician's office. Examination disclosed a dislocation of the larynx that was successfully reduced by manipulation under general anesthetic, again utilizing the technique of lifting the inferior cornu anteriorly with one hand and elevating the

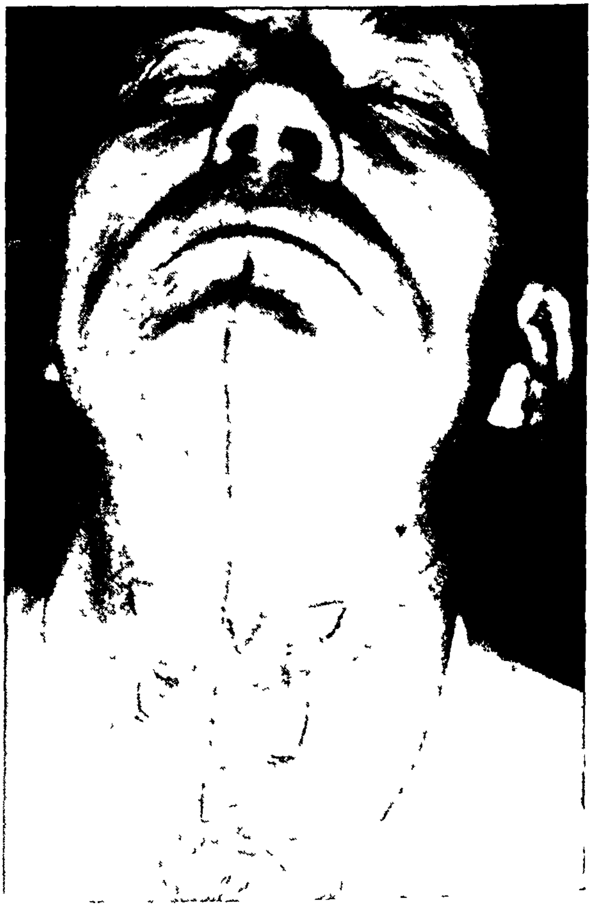


Fig 4 Case 7 R B, age 47 Preoperative photograph showing left lateral displacement of the larynx with flattening of the left side of the voice box This condition had been present for over four months

base of the tongue with fingers of the other hand Other than complaints of a sore throat for two days postoperatively his recovery was uneventful

Case 7 R B, age 47, was seen on April 16, 1956, on referral because of the complaints of hoarseness, a sensation of a lump in the throat, and discomfort on swallowing which he stated had been present since an automobile accident on January 26, 1956 In the accident his head had been forced through the windshield He stated that the intensity of the discomfort had been gradually less severe since the accident Palpation of the neck revealed a displacement of the thyroid notch to the left, and laryngoscopic examination demonstrated an angulation of the glottic space with bilaterally edematous vocal cords and decreased motility of the left vocal cord X ray studies showed no abnormalities (see Fig 4) As attempts to correct the laryngeal deformity by manipulation were unsuccessful it was deemed advisable to utilize an external surgical approach supplemented by simultaneous direct laryngoscopic observation

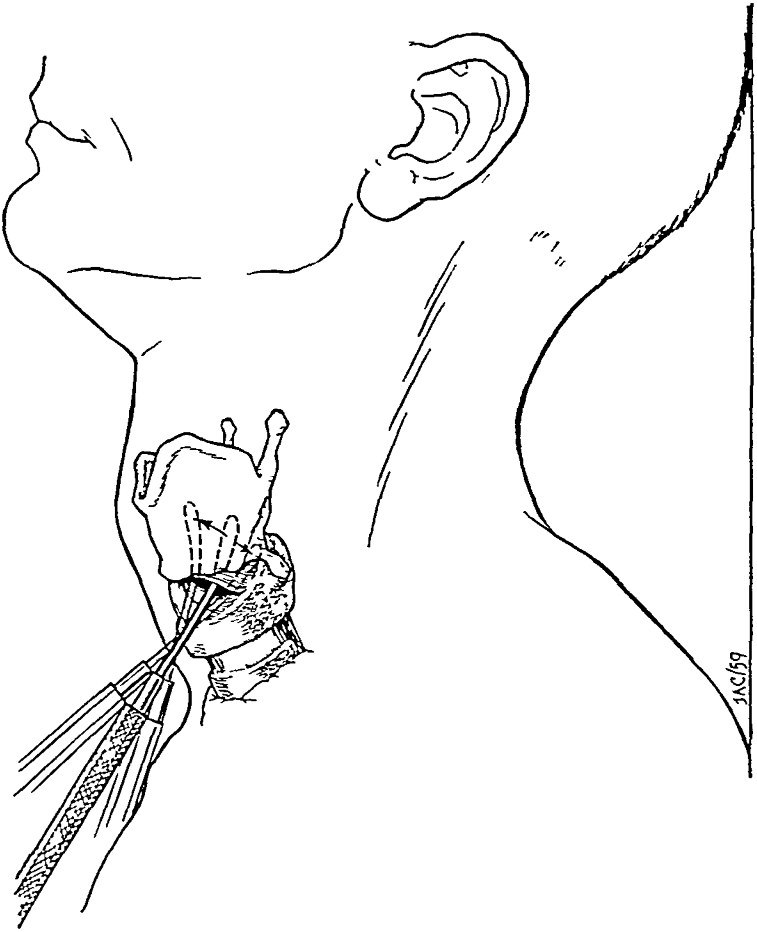


Fig 5 Surgical approach for relocation with simultaneous elevation of the voice box with the laryngoscope

Under combined local and general anesthetic an eight-centimeter collar incision was made over the mid portion of the larynx and the ribbon muscles separated and retracted without disturbing the underlying laryngeal perichondrium The cricothyroid membrane was cut on each side of the midline being careful not to enter the larynx A Freer septal elevator was then placed medially to the thyroid cartilage and was used to free the thyroid ala bluntly bilaterally and to reduce the displaced and ankylosed inferior cornu (see Fig 5) At the time of external manipulation an associate used a Jackson anterior commissurescope to displace the larynx anteriorly a definite reduction of the deformity was accomplished The wound was closed and the dressing fortified by additional packing on the right side was designed to maintain proper laryngeal alignment The patient s recovery was uneventful with a marked improvement When last seen his thyroid notch was in the mid line and he swallowed without discomfort

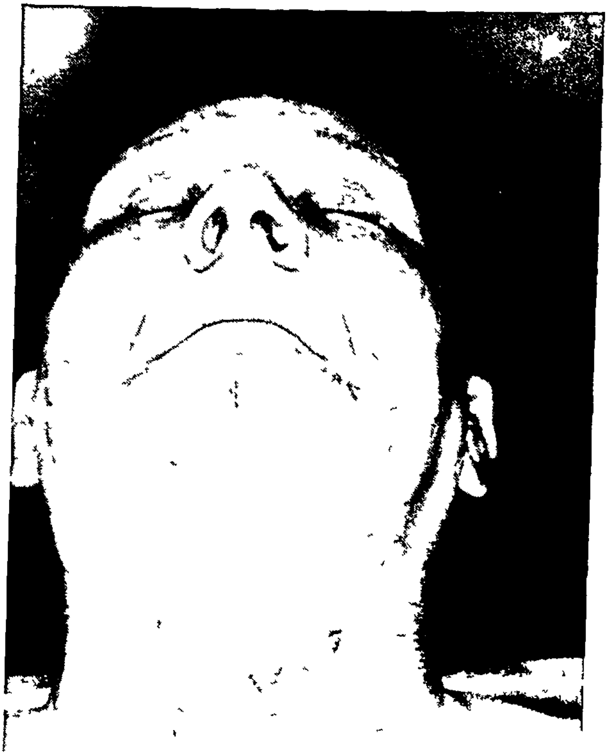


Fig 6 Case 8 H B Preoperative photograph showing displacement This condition had been present for a month following an automobile injury caused by striking his neck on the steering wheel

Case 8 H B, age 47, was first seen on June 12, 1956, a month after sustaining an injury to the larynx and fractures of the legs in an automobile accident He believed that he had struck his neck on the steering wheel and had experienced marked shortness of breath for approximately one hour after the accident in addition to severe discomfort in the throat and a husky voice Difficult and painful swallowing with pain referred to the left ear had persisted, and he had found it necessary to remain on a liquid to soft diet Upon examination it was noted that the thyroid notch was displaced approximately 1 cm to the left and that there was fullness and tenderness on the right side of the larynx and a flattening on the left side (see Fig 6) The glottic chink was displaced about 20 degrees to the left in its anteroposterior dimensions X ray studies showed no abnormalities Efforts at reduction of his laryngeal deformity by manipulation were unsuccessful and a laryngoplasty similar to the procedure outlined in the above case was performed His postoperative

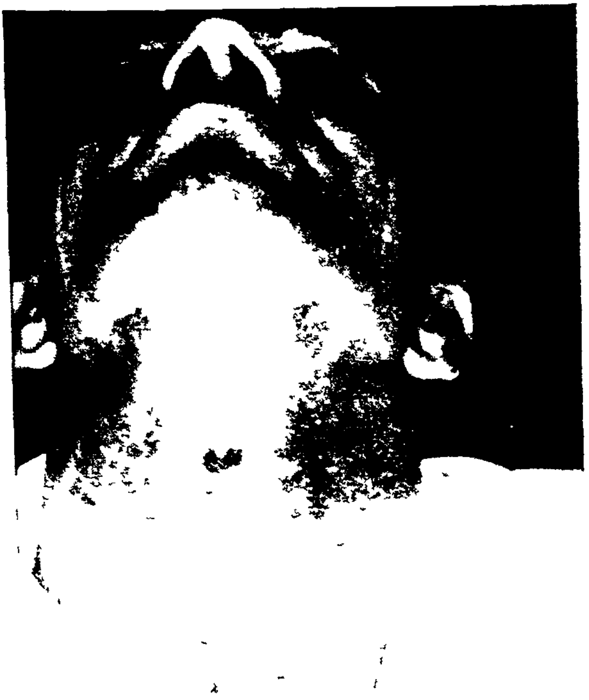
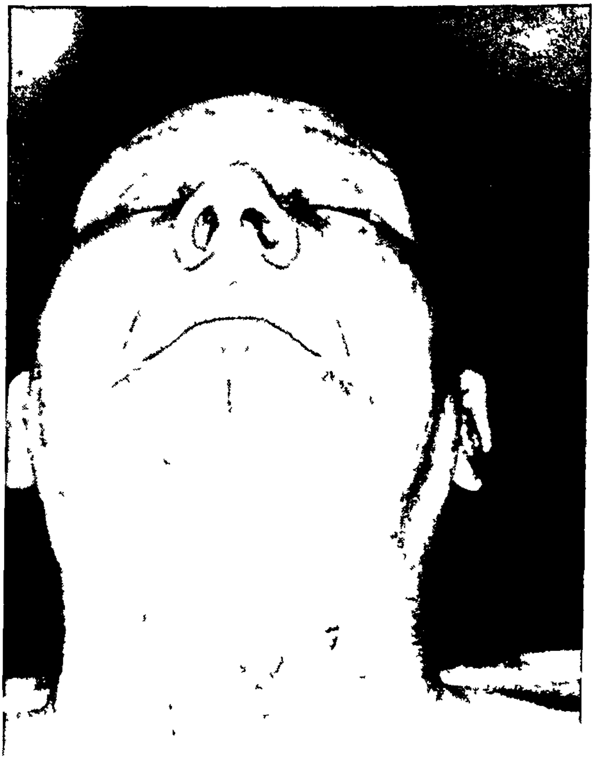


Fig 7 Case 9 H C Postoperative findings of normal external laryngeal landmarks Laryngoplasty for fracture and dislocation of the larynx two years 9 months previously

course was uncomplicated and there was definite improvement in swallowing function and the location of his voice box When he was last seen his voice had returned almost completely to normal

Case 9 M C age 49 was first seen in the hospital on July 15 1956 the day after he had been struck in the neck by one of three assailants Immediately after the injury he was unable to speak above a whisper and had expectorated a small amount of blood External examination revealed a swollen tender larynx with flattening of the left side laryngoscopy demonstrated angulation of the glottic space to the left with edematous cords and a fractured and partially dislocated arytenoid cartilage (see Fig 7) Under combined local and general anesthetic a tracheostomy was performed and an 8 cm collar incision was made over the mid portion of the larynx After vertical separation and retraction



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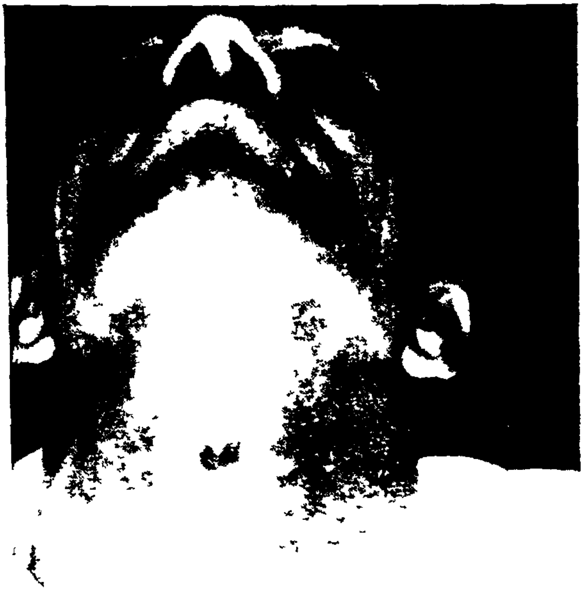

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CONCLUSIONS

1 Cricothyroid dislocation does occur

2 Acute dislocations should be reduced under general anesthesia for two reasons it is more comfortable for the patient, and as in shoulder dislocations, it makes for better relaxation and reductions

3 Long standing ankylosed dislocations require reduction by open surgery

4 Subluxation of the cricothyroid joint may be common, but the dislocation is rare

5 Vague complaints and discomfort in the neck may be the result of subluxation tendency

6 Early diagnosis simplifies management, and it is probable that the condition fails to be recognized on occasion

SUMMARY

Information gained from the study of nine patients with cricothyroid dislocation is presented, and a surgical technique for reducing long-standing dislocations is introduced

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THE MOUNT SINAI HOSPITAL POSTGRADUATE COURSE

In order not to conflict with the International Congress of Otolaryngology to take place in Paris in July, 1961, the annual intensive postgraduate course in Rhinoplasty, Reconstructive Surgery of the Nasal Septum and Otoplasty will be given January 7-20, 1961, by Dr Irving B Goldman and staff at the Mount Sinai Hospital, New York, in affiliation with Columbia University

Candidates for the course should apply to Registrar for Postgraduate Medical Instruction, The Mount Sinai Hospital, Fifth Avenue and 100th Street, New York 29, N Y

OBSERVATIONS WITH CERTAIN EXPERIMENTS ON THE PARALYZED VOCAL CORDS IN DOGS*

MATTHEW H EVOY, M D ,

Seattle, Wash

Injury to one or both recurrent nerves in thyroid surgery is occasionally inevitable, and the consequent cord paralysis is often permanent The inevitability of nerve damage is acknowledged by anatomists as well as surgeons, as post mortem dissections repeatedly demonstrate the vagaries of the human recurrent nerves My own findings in cadaveric explorations have shown an absence of a single nerve trunk at mid-thyroid level in fully one-fourth of the cases These studies, untroubled by haste, lack of exposure, or bleeding, support the findings of Rustad[1] who reported that 20 per cent of human nerves in his series had divided at or below the level of the lower pole of the thyroid

With a full realization of the inevitability of bilateral abductor cord paralysis on rare occasions, experiments were conducted to simulate as far as possible the actual nerve interruption that may occur in thyroid surgery With full knowledge that either adductor or abductor components of the nerve can be exclusively injured- and thus affect the behavior of the ipsilateral cord, we have attempted to divide all of the recurrent nerve fibers at the thyroid level With the cord thereby paralyzed, we have studied the possibility of voluntary muscle control of the immobilized arytenoid cartilage by use of the sternothyroid muscle, a muscle that has a voluntary as well as involuntary action

The movements and actions of the arytenoid cartilages were studied in eight fresh human cadavers that were kindly made available to us by our coroner In these observations, the

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sliding action at the cricoarytenoid joint was demonstrated A suture was fixed to the arytenoid cartilage, and, through a window in the thyroid cartilage, the movement of the vocal cord was observed while traction was applied in various directions With the suture secured just at the insertion of the posterior cricoarytenoid muscle, it was found that the greatest amount of abduction, and hence widening of the glottic chink, was produced when the pull was applied downward, medialward and slightly backward Such an axis, of course, is exactly that of the acting posterior cricoarytenoid muscle It was interesting to find, also, that when the traction was released, the cartilage invariably coiled back to bring the vocal cord into the paramediam position It was our hope that the turgor of the muscles and ligaments about the arytenoid cartilage in the living tissues would also bring about this spontaneous adduction recoil

METHODS

Twelve large adult dogs were anesthetized with sodium nembutal intravenously The vocal cords were examined and then the animals were intubated A vertical incision was made to extend well above and below the thyroid tubercle The pretracheal muscles were retracted laterally and the lobe of the thyroid gland identified The recurrent nerve was almost always visible as a large trunk passing in the tracheal-esophageal groove The thin lobe of the thyroid was divided between hemostats and all of the tissue in the space between the carotid sheath and trachea and esophagus was sectioned to insure, as far as possible, the total interruption of the recurrent nerve fibers In every instance except one, subsequent visualization of the vocal cords established proof of the immobility of the cord

In each case the immobile arytenoid cartilage was exposed In four dogs the contralateral sternothyroid was detached from its insertion, brought through a tunnel fashioned between the posterior larynx and the upper esophagus, and the muscle was secured to the immobile arytenoid cartilage In two animals, fascia was taken from the foreleg and inserted from the contralateral sternothyroid, through a tunnel, and

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COMMENT

It appears that for a few reasons the results of the transposition of muscle in the larynx of the laboratory animal may not be projected to humans In the first place, voluntary contraction of a somatic muscle cannot, of course, be obtained in animals Then again, the anatomy of the canine larynx is significantly different from the human, and exposure of the arytenoid cartilage in the dog is quite difficult because of the marked posterior and medial projection of the wings of the thyroid cartilage The formation of scar tissue, although inevitable, from even the most gentle handling of the tissues, probably was largely a result of mild sepsis in these animals

Although our observations suggested the futility of successful muscle transplantation about the larynx in the dog, we were encouraged to note that in none of our animals was there evidence that the muscle or fascia or wire had pulled away from its attachment We had feared that the repeated involuntary elevation of the larynx that occurs with swallowing would place undue tension on the suture lines

The scar tissue fixation of the arytenoid cartilage to the wing of the thyroid cartilage even where only a Surgaloy strut was employed led us to believe that handling and possibly sepsis were the engendering agents rather than any reaction occasioned by the transplanted tissue In the two cases in which the fascia was placed in an artificial sheath of autogenous vein, we found no more gliding tendency than in those without a covering The intima of the vein still glistened somewhat, but was slightly adherent to the fascia throughout the entire length

CONCLUSIONS

Experiments with the transplantation of a voluntary muscle to the paralyzed arytenoid cartilage in dogs have been carried secured to the articular head of the arytenoid cartilage In two others, Surgaloy wire was interposed in the same fashion Segments of autogenous (external jugular) vein were used as a jacket outside of the two fascial struts

In the last four dogs in this series, the ipsilateral sternothyroid muscle was used In two instances the muscle was brought directly to the arytenoid cartilage, while in one animal, fascia, and in the other, Surgaloy wire, were interposed

The animals were reoperated upon in from six weeks to three months following the original procedure, and all were sacrificed after the second operation The larynx was removed for a more detailed study in three of the dogs

RESULTS

At the completion of the initial operation, the cord action was visualized through a laryngoscope while the sternothyroid muscle was activated by electrical stimulation Almost without exception, some widening of the glottic chink occurred, although the powerful muscle contraction depressed the entire larynx and made observation less than satisfactory

With the exception of one dog, all animals lost the ability to bark There was one superficial wound separation, but otherwise the wounds healed promptly without evidence of infection No signs of inspiratory distress, either resting or exertional, ever developed

At re-operation, a moderate amount of scar tissue was found in the subcutaneous regions and about the strap muscles on the operated side The transposed sternothyroid muscle was viable in every instance, and in no case had the muscle or fascia or Surgaloy pulled away from the cartilage Electrical activation of the sternothyroid depressed the ipsilateral larynx, but in no case was there abduction of the cord as viewed through a window in the trachea This disturbing and disappointing finding was not surprising in view of the great amount of scar tissue that was found in every animal about the tract occupied by the transplanted muscle, or fascia, or Surgaloy The articular prominence of the arytenoid cartilage was frozen to the adjacent wing of the thyroid cartilage in every case The cricoarytenoid joint was found to be smooth and free in all instances except one in which a metallic suture was inadvertently passed into the joint cavity

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REVERSIBLE SENSORI-NEURAL DEAFNESS

FRANK KODMAN, JR , Ph D ,
O A CULL, M D ,
Owenton, Ky ,
and
THOMAS O LAWSON, M A ,
Lexington, Ky

Frequently the clinician will encounter a patient who shows a perceptive or sensori-neural hearing loss which on later examination is completely or partially reversed As Juers[6] points out, the assumption that all perceptive deafness is irreversible is no longer valid Clinical evidence is accumulating which shows that much remains to be learned about reversible, perceptive deafness

The terminology used in relation to irreversible deafness deserves a rhetorical comment The ability to send the patient on his way with the explanation that his "ear nerves are dead" or that he has "nerve deafness" is most often a feat of skill How many diagnostic tests do we have today which pinpoint hearing impairment as specifically related to that short bundle of nerve fibers sometimes referred to as the "hearing nerve?" While the explanation that the nerve is "dead" may serve to satisfy and even impress the patient, the statement is often unsubstantiated and should be relegated to the linguistic junk heap

Recently the term *sensori-neural hearing loss* has been suggested to replace the historical and much maligned rubric "nerve deafness" The newer term implies that the locus of the loss is cochlear or retro-cochlear without limiting the etiology to the acoustic nerve A discussion of the published clinical and experimental literature on hearing impairment attributable to the cochlea, central auditory pathway or auditory cortex is beyond the scope of the present paper

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out It appears that in the laboratory animal, scar tissue fixation of the transplanted muscle, and an invariable fibrous union between the arytenoid cartilage and the ala of the thyroid cartilage, prevent the desired action of the transposed sternothyroid muscle

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Juers believes that a broad physiological approach should be applied to the range of etiological factors causing perceptive (sensori-neural) deafness Certainly a change in our philosophy toward permanent hearing loss is worth considering and should bear much fruit

Early audiologic evaluation, through otologic and pediatric appraisal and increased research efforts are in order, particularly regarding pre-school children about whom we have the least information

Improved hearing testing methods beyond those simply determining the degree of loss—objective puretone and speech audiometry, the tonal decay test, the aural overload test, more valid tests for recruitment, diplacusis, tinnitus and the clinical use of delayed speech feedback—offer new insights into the complexity of the auditory mechanism and auditory pathology

Stapes mobilization and exploratory excursions into the middle ear have added a new chapter to reconstructive ear surgery and to reversible *conductive* deafness These advances and many others recommend that we adopt a more optimistic outlook toward certain types of perceptive deafness

Carhart[3] has demonstrated that a portion of the perceptive loss is reversible in successful cases of stapes mobilization An average increment of approximately 9 db may be expected in the bone conduction thresholds from 500 to 4000 cycles after ossicular function has been maximally improved

Widespread use of bone conduction audiometry, speech audiometry and tests of cochlear function by audiologists and physicians using standard quantitative audiometric procedures rather than qualitative techniques will permit a more accurate diagnosis of the type of loss and enhance the accuracy of treatment and prognosis

Miles and Echols[8] reported a case of reversible deafness following surgical extirpation of a pearly tumor of the cerebello-pontine angle Simonton[13] has presented an excellent discussion of the course of variable deafness in progressive Meniere's disease There are instances wherein the hearing returns to normal and then regresses as the nerve cells in the spiral ganglion degenerate In contrast to the preceding cases, Bordley and Haskins,[1] and Walsh and Goodman[15] call our attention to those patients who manifest an inability to comprehend speech despite a mild-to-moderate puretone air conduction hearing loss They may have retrocochlear rather than cochlear lesions Loch[7] cites evidence that a certain percentage of tonal dips at 4,000 cps and 10,000 cps is reversible with proper diagnosis and treatment The literature is replete with illustrated cases of reversible traumatic deafness

Mizukoshi, Konishi and Nofamura[9] demonstrated that certain cytochemical changes found in the hair cell nuclei of the organ of Corti are generally reversible They studied the effects of acoustic stimulation and certain toxic drugs on the metabolism of the cochlea in guinea pigs

A small percentage of patients show perceptive deafness due to toxic drugs or poisons Taylor[14] has written an excellent chapter on the effects of drugs and poisons on auditory function The chapter appears in the classic text, *Medicine of the Ear*, edited by E P Fowler, Jr Sayers and Davenport[11] have presented a thorough review of the physiological effects of CO The consensus of opinion is that perceptive loss resulting from drugs and poisons is usually reversible, at least in part Unfortunately, adequate measurements rarely accompany the clinical description

The authors of this paper encountered a patient who incurred a severe, bilateral perceptive loss as a probable result of salicylates and similar medication administered for rheumatoid arthritis plus an accidental exposure to carbon monoxide Periodic audiological measurements were made over a 13-month period during the recovery phase of his perceptive deafness The results are presented in detail and related to several theoretical assumptions

CASE HISTORY

Our patient a white male, aged 42, had been receiving treatment for rheumatoid arthritis His medication consisted of Pabalate H C a salicylate hydrocortisone compound and chloroquin (Aralen) Complications arose when he was exposed to an unknown dose of carbon

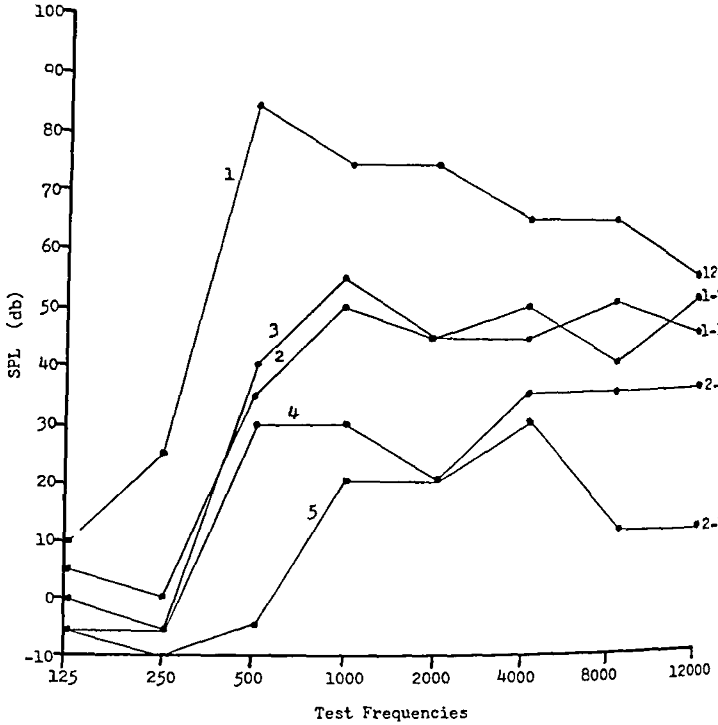

Fig 1 Right ear recovery (air conduction thresholds)

monoxide from a recently installed, defective damper on his furnace He was taken to a local hospital for diagnosis and treatment after he was found at his home in a comatose condition The question of deafness arose due to carbon monoxide poisoning A week later, December 30, 1957, he was given an audiological evaluation at the University of Kentucky Audiology Clinic

The results of the audiological evaluation revealed a speech threshold of 80 db in the right ear and a speech threshold of 90 db in the left ear Speech discrimination was 36 per cent in the right ear and 28 per cent in the left ear His audiometric loss was verified by Psychogalvanic Skin Response Audiometry, the Lombard Test and Delayed Speech Feedback Patient complained of tinnitus aurium, bilaterally He described the tinnitus as "ringing" "chimes" and "hen cackles"

Due to the etiology of his loss we requested the patient to return a week later for re evaluation His retest on January 7, 1958, showed partial recovery of his hearing loss There was no evidence of diplacusis He received further evaluations on January 13, 1958, January 23 1958, February 3, 1958, February 17, 1958, March 4, 1958, March 17, 1958, April 21, 1958, and February 18, 1959

AIR CONDUCTION THRESHOLDS

In Fig 1, the air conduction thresholds for the right ear are plotted for eight frequencies obtained on five of the examinations The results show gradual recovery at all frequencies with a residual loss remaining from 1,000 to 12,000 cycles The thresholds at 125, 250 and 500 cycles are slightly better than normal

Fig 2 shows the corresponding thresholds (air conduction)

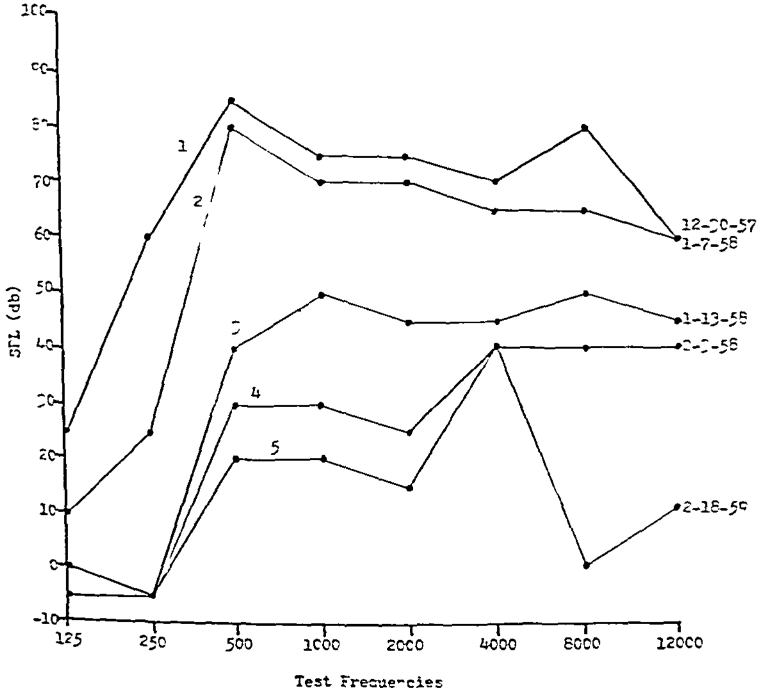

Fig 2 Left ear recovery (air conduction thresholds)

for the left ear The recovery is similar to that obtained for the right ear, except that the residual loss extends from 500 to 4,000 cycles with slightly better sensitivity at 8,000 and 12,000 cycles

BONE CONDUCTION THRESHOLDS

The bone conduction thresholds displayed in Figs 3 and 4 parallel the air conduction curves shown in Figs 1 and 2 On

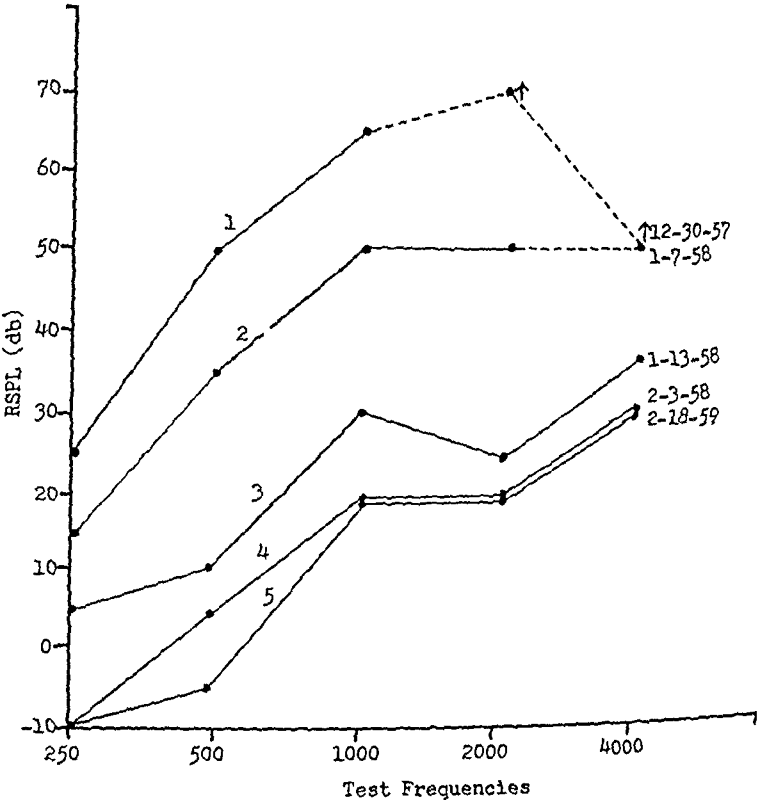

Fig 3 Bone conduction thresholds during recovery period Right ear

this basis, the loss bilaterally would be classified as predominantly perceptive or sensori-neural The initial examination ruled out the possibility of a psychogenic (non-organic) loss or malingering

SPEECH RECEPTION TESTS

The speech thresholds represented by the bar graph in Fig 5 correlate highly with the average puretone thresholds from 125 to 1,000 cycles The residual loss for the left ear is reflected in the speech threshold as well as the puretone thresholds The ten examinations (t_1 - t_{10}) permits a longitudinal look at the threshold recovery in the form of the speech thresholds The right ear recovered gradually from

t_1 - t_5 The left ear dropped from 90 db to 20 db from t_1 - t_6 The right ear dropped from 80 db to normal from t_1 - t_5

In Fig 6, the speech discrimination scores for each ear show gradual improvement from t_1 - t_5 From t_5 - t_{10}, speech discrimination returned to a near-normal level The reversible nature of this patient's hearing loss is supported by the pure-tone, speech and bone conduction thresholds and the speech discrimination scores The patient's subjective evaluation of his hearing always coincided with the gradual improvement demonstrated by the auditory measurements

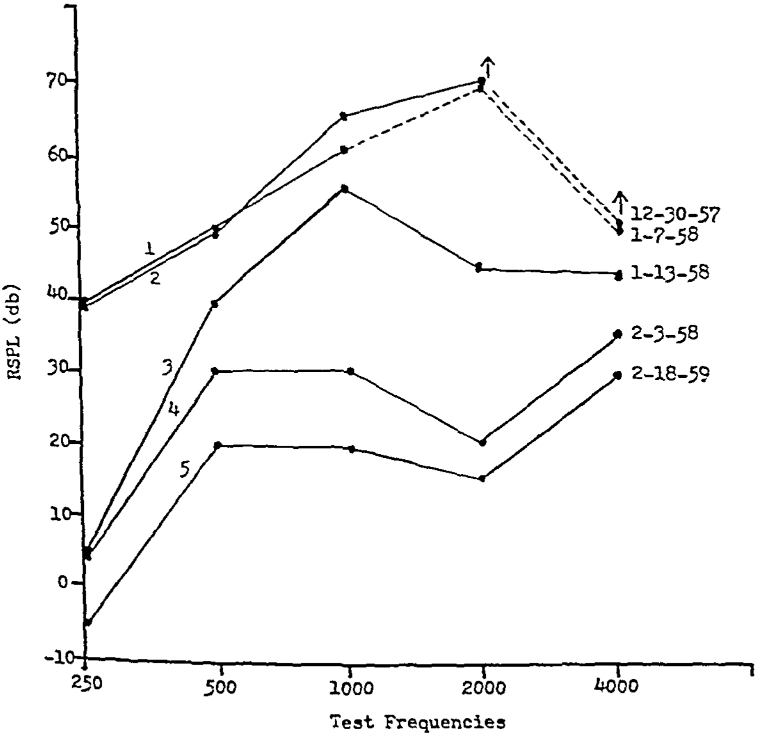

Fig 4 Bone conduction thresholds during recovery period Left ear

On his final examination, there was a diminution in the degree of recruitment and in the loudness of his tinnitus At present, he functions adequately in communicative situations There is a residual puretone loss in the higher frequencies in each ear Speech discrimination remains near-normal

Response reliability was consistent during all the psychophysical measures Threshold fluctuation was not a clinical symptom in this case No vestibular impairment was noted

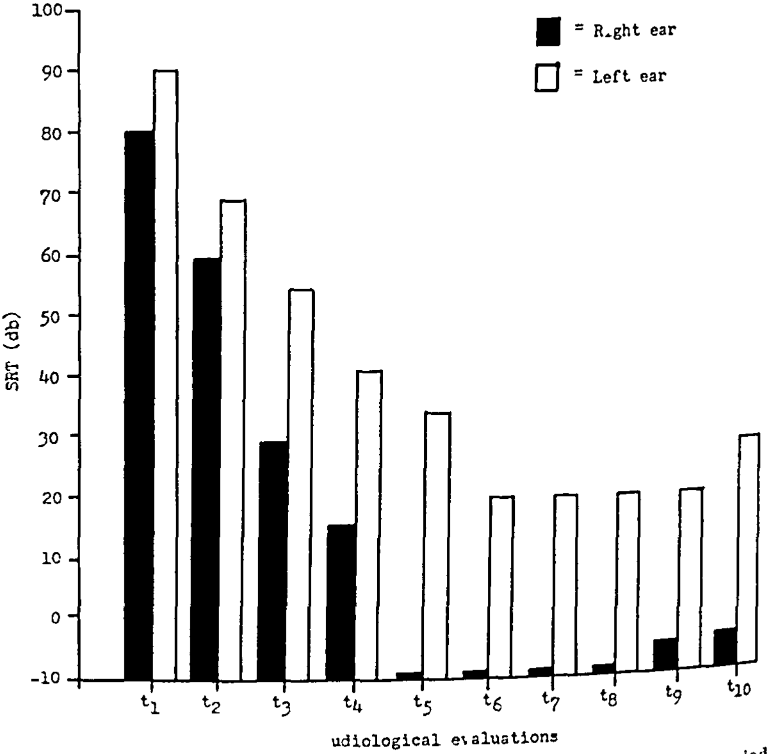

Fig 5 Speech thresholds measured periodically during recovery period

There is no evidence of an emotional disorder, and the patient has returned to work He was also returned to his former medication and dosage schedule

IMPLICATIONS OF THE DATA

We are unable to ascertain the separate effects of the salicylate-type medications and the exposure to carbon mo-

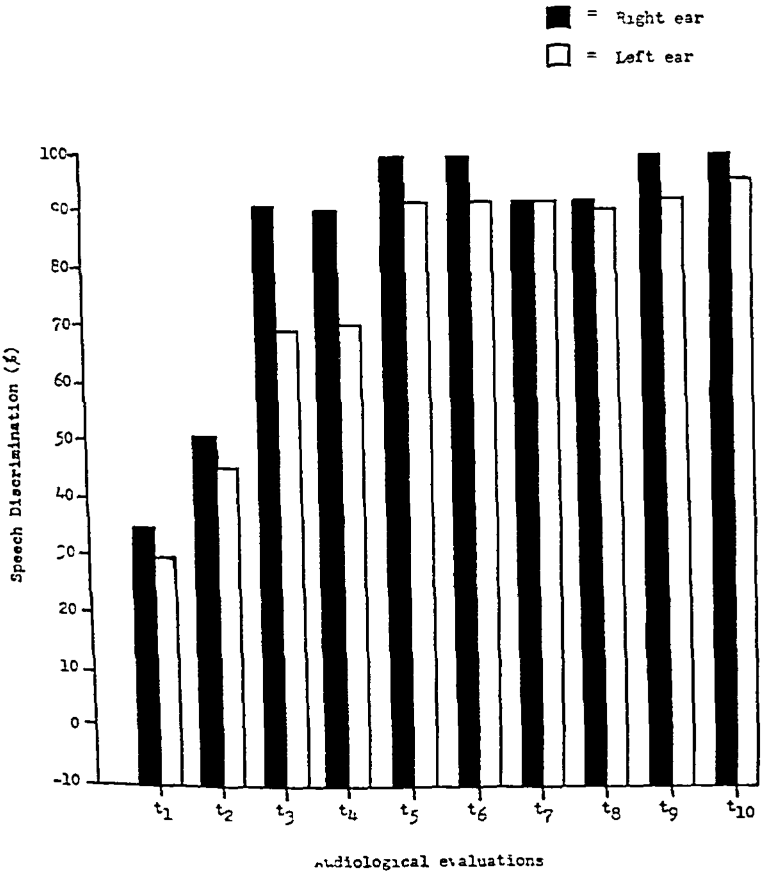

Fig 6 PB score measured periodically during recovery period

noxide It seems worthwhile to relate the aural pathology to certain physiological mechanisms in the auditory system

The clinical evidence points strongly to a cochlear lesion and a retrocochlear lesion which were at least partially reversible Ruttin[10] feels that bone conduction impairment and tinnitus are indicative of a cochlear lesion Recruitment was present bilaterally and is generally considered to have its locus in the cochlea Diplacusis was not present, even though it is commonly associated with cochlear lesions Speech discrimination did not decrease with an increase in the sensation level of the stimulus words In this respect, our case present, he functions adequately in communicative situations. There is a residual puretone loss in the higher frequencies in each ear. Speech discrimination remains near-normal.

Response reliability was consistent during all the psychophysical measures. Threshold fluctuation was not a clinical symptom in this case. No vestibular impairment was noted.

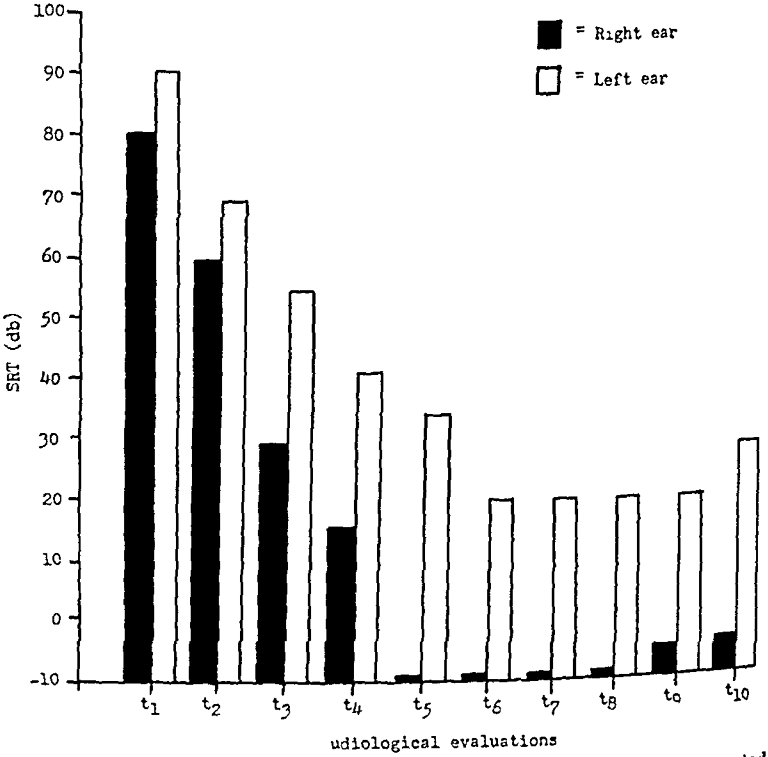

Fig. 5 Speech thresholds measured periodically during recovery period

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did not differ from five of the six cases of nerve fiber degeneration reported by Eby and Williams[5] They also reported that 15 out of 17 cases with end-organ lesions showed a decrease in speech discrimination as sensation level was increased Their findings appear to support nerve fiber pathology in our patient, since speech discrimination did not decrease when speech was presented at 110 db above the normal threshold

Dandy,[4] and Schuknecht and Woellner[12] have performed partial resections of the auditory nerve which produced various degrees of hearing loss The latter experimenters associate diminished speech discrimination with degenerative changes in the spiral ganglion Crowe, Guild and Polvogt[3] correlated cochlear lesions with high-tone loss in 79 cases Our patient had a high-tone loss and near-normal thresholds in the low frequencies even after his recovery was stabilized.

The complete clinical picture suggests *both* cochlear and nerve fiber pathology in our illustrated case We cannot, however, rule out a lesion of the central auditory pathway A portion of the pathology was reversible since hearing function was largely regained CO and salicylates may produce changes in the cochlear structures with resultant impairment of the hearing function The durability and versatility of the auditory mechanism to sustain injury is to be noted A great deal of pathology may be present with the hearing function showing only a slight loss This fact may often be overlooked by non-medical specialists The reversible nature of certain types of perceptive deafness is worthy of critical examination

The outstanding advances in understanding auditory function and aural pathology offer new hope for the large percentage of the population who suffer from hearing disorders These advances call for a re-organization of our theoretical considerations of aural rehabilitation Here is a virgin field for the theoretician, the progressive clinician and the laboratory scientist Biochemistry and neurophysiology offer audition a major hope for breaching the impaired sound barrier as a sequence Acute reticulo-endotheliosis (Letterer-Siwe) is the earliest, most acute, fatal form, it is followed by a polyphasic phase (Hand-Schueller-Christian), and by the granulomatous and xanthomatous phases Emerson (1958) formulated the cycle of the manifestations in noncholesteremic conditions as follows There is an early proliferation of masses of reticuloendothelial cells, including many eosinophils; these masses may be solitary and slow-growing, as in the eosinophilic granuloma of bone, or widespread and rapidly growing throughout the reticulo-endothelial cell system, as in Letterer-Siwe disease, showing a tendency to accumulate intracellular cholesterol and to be transformed into foam cells, typical in Hand-Schueller-Christian disease, and may revert to granulomatous cells with ultimate fibrosis and spontaneous disappearance

Participation of the hearing organ is mentioned in the very first reports In Schueller's case (1915), a 16-year-old, otitis media was among the first manifestations of the disease, with defects in the X-ray picture of the temporal squama and pus in the external canal, assumed to be present behind the stenotic canal entrance Christian's (1920) patient, a five-year-old girl, suffered impairment of hearing among the first symptoms She heard loud tones, but not the watch, the test was not considered to be accurate as later there was no evidence of deafness

Reports on manifestations in the ear and in the upper airways were slow to accumulate, but Lederer, Ponchet and Fabricant, in 1935, emphasized the relative frequency of ear-region involvement

Around the 1950's growing interest and advanced diagnostic methods resulted in rapidly multiplying publications Some of these showed manifestations in the upper airways to be more frequent than those in the ear Greifenstein's 26 cases yielded a proportion of 18 to 12, the seven cases of Schuknecht and Perlman (1948) a proportion of four to three On the other hand, among the 18 cases of Goodhill (1950) the ear participated in 11

HISTOCYTOSIS INVOLVING THE TEMPORAL BONE[*]

(Letterer-Siwe, Hand-Schueller-Christian)

GEORGE KELEMEN, M D ,

Boston, Mass

Thannhauser's textbook (1958) gave the following description of the Hand-Schueller-Christian syndrome Osseous lesions of eosinophilic xanthomatous granuloma, defects in the membranous bones of the skull, exophthalmos and diabetes insipidus Greifenstein (1932) treated participation of the hearing organ in this condition in a monograph After this beginning a well-rounded literature tried to integrate the findings in the ear and in the upper airways into the framework of general knowledge regarding histocytoses

Fraser (1934) called the clinical entity, in terms of the underlying pathology, lipoid granulomatosis of bones He felt that endothelial cells from the vessel walls, containing lipoid, pass into the surrounding tissue and provoke lymphocytic infiltration, phagocytic xanthoma or foam cells may contain degenerated leukocytes and even minute bone particles Arising from the lipoid-containing endothelial cells, their growth results in the multinuclear giant cell

The monistic theory of Farber (1941) found ever widening acceptance until it reached its present leading role He considered eosinophilic granuloma not to be a distinct entity but a variant of the basic process of which the clinical pictures known as Hand-Schueller-Christian, and Letterer-Siwe syndromes are other examples, the disease may be suspected on the basis of clinical and roentgenological data, but those have to be correlated with the investigation of biopsy material before a positive diagnosis is established

Thannhauser explained the concept of the different phases

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[*] From the Department of Otolaryngology Harvard Medical School and the Massachusetts Eye and Ear Infirmary Boston Aided by a grant from the Godfrey M Hyams Fund

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drainage from the right ear was noted since 17 months of age but worsened at 21 months and a large swelling appeared behind the ear At this time, she was first examined at the Children's Medical Center, where it was shown that she had swellings behind the right ear and in the right mandible, accompanied by destructive lesions in the temporal bone and mandible, diabetes insipidus, mild pulmonary infiltration, a scaly scalp eruption, necrotic and exudative stomatitis with loosening of teeth, red papular eruption on chest and back, and mild anemia Radio therapy was given to the right side of the skull (including the pituitary area), but the diabetes insipidus did not improve She was started on repository ACTH (30 units ti wk.), which was continued for four months and chlorambucil (2-4 mgms q d), which was maintained, with occasional interruptions for the next seven months Her response to chemotherapy was disappointing, with the skin eruptions continuing in varying degrees and involvement noted in the perineum and perianal area Replacement therapy with pitressin tannate in oil was only partially effective, and at 23 months of age another course of radiation was given to the pituitary area In subsequent months, the impression was obtained that her disease was under some control, in that her pulmonary lesions cleared, new bone lesions did not appear, and the liver and spleen did not enlarge, however she remained very fussy, and scalp and skin rashes were most troubling Drainage from the ears especially the right was nearly continuous, but it was never possible to establish that this exudate was of any origin other than the moist eruption in the external canals *per se* The second radiotherapy course had not diminished the severity of her diabetes insipidus, and pitressin injections remained irregularly effective Prednisolone (up to 50 mgms q d) was substituted for the ACTH (at 25 months of age), but it produced no difference in response

The final episode began at a time (28 months of age) when her general condition had been relatively good She developed fever, increased irritability, vomiting and diarrhea two weeks after she had had a known exposure to measles (and one week after she had been given a protective dose of gamma globulin) These signs of infection increased, and she required admission to the hospital to aid in the control of fluid balance There she had her usual skin findings, dehydration a "toxic" appearance, pharyngitis and a clear chest to general examination On intravenous fluid therapy and antibiotics her vomiting was adequately controlled, but there was a gradual increase in fever, unresponsiveness pulse rate, and respiratory rate Finally she became cyanotic and comatose, and died 36 hours after admission in spite of extensive supportive measures Her terminal complications and the virologic studies on her post-mortem tissues are described (as "Case 3") in a recent article by Enders, McCarthy Mitus and Cheatham (1959)

Autopsy (Pathol Department of Dr Sidney Farber) confirmed a Hand-Schueller Christian process involving the skull the infundibulum, pituitary stalk scalp, epicardium, lungs and lymph nodes In the skull and the infundibular region the process appeared to be in a fairly advanced state of lipidization Histochemical analysis showed a high proportion of cholesterol inside the lipid-containing cells From the lungs affected by a diffuse giant cell pneumonia measles virus of a high titre was isolated One of the lymph nodes near the stomach contained Warthin-Finkeldy cells (multinucleated giant cells), while involvement of the lymph nodes by Letterer Siwe disease was minimal

Analysis of the tissue lipids (Dr Allen C Crocker) yielded the following

Spontaneous healing or the beneficial effects of X-ray therapy (since 1930) was probably the reason why only biopsy material was offered for histopathologic investigation Nager stated, in 1933, that in cases of general xanthomatosis a number of clinical reports on the state of the hearing organ were given, but no histological examination of the petrous bone was known up to that date It took another three years before studies of the latter kind could be added

Druss in 1936 recorded the ear manifestation of lipoid granulomatosis, Hand-Schueller-Christian disease in a 48-year-old male whose morbid history reached back through nine years with bilateral aural discharge and considerable loss of hearing, in the last seven years he was treated for diabetes insipidus In numerous bones and soft organs, changes were found corresponding to a universal lipoid granulomatosis, lipoid granulations were found in various parts of the temporal bone but not in the middle ear

Goodhill registered 18 pertinent cases, 11 had temporal bone lesions, ten with chronic otorrhea One pair of temporal bones of a three-year-old girl (as reported by Dr Dorothy Wolff) revealed areas of infiltration into all the pneumatized portions, including mastoid and petrous apex The infiltrations contained, in various regions, representations of every phase of histocytic granuloma reticulum cells, foam cells, and eosinophils in clumps The bony labyrinthine capsule was not invaded The sensory end organs were intact, the infiltration was restricted to the cancellous bone and the cellular trabeculae of the temporals

CASE REPORT

K T, born September 11, 1955 C M C No 460061 Referred by Drs E F Carniglia and E W Heublein

This little girl's first symptoms were a seborrhea like scalp eruption at 11 months of age, increasing irritability by 14 months, and soon there after a papular rash on trunk and groin An exudative eruption was noted on her gingivae, and at 15 months a swelling in the right side of the mandible was shown by biopsy to be an eosinophilic granuloma There was a temporary shrinkage of this mass after radiotherapy Severe polydipsia and polyuria began at 20 months of age An intermittent thin drainage from the right ear was noted since 17 months of age, but worsened at 21 months and a large swelling appeared behind the ear At this time, she was first examined at the Children's Medical Center, where it was shown that she had swellings behind the right ear and in the right mandible, accompanied by destructive lesions in the temporal bone and mandible, diabetes insipidus, mild pulmonary infiltration, a scaly scalp eruption, necrotic and exudative stomatitis with loosening of teeth, red papular eruption on chest and back, and mild anemia. Radio therapy was given to the right side of the skull (including the pituitary area), but the diabetes insipidus did not improve She was started on repository ACTH (30 units t.i wk), which was continued for four months, and chlorambucil (2-4 mgms q d), which was maintained, with occasional interruptions for the next seven months Her response to chemotherapy was disappointing, with the skin eruptions continuing in varying degrees and involvement noted in the perineum and perianal area Replacement therapy with pitressin tannate in oil was only partially effective, and at 23 months of age another course of radiation was given to the pituitary area In subsequent months, the impression was obtained that her disease was under some control, in that her pulmonary lesions cleared new bone lesions did not appear, and the liver and spleen did not enlarge, however she remained very fussy, and scalp and skin rashes were most troubling Drainage from the ears especially the right was nearly continuous, but it was never possible to establish that this exudate was of any origin other than the moist eruption in the external canals *per se* The second radiotherapy course had not diminished the severity of her diabetes insipidus, and pitressin injections remained irregularly effective Prednisolone (up to 50 mgms q d) was substituted for the ACTH (at 25 months of age), but it produced no difference in response

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Analysis of the tissue lipids (Dr Allen C Crocker) yielded the following

Tissue	Liver	Spleen	Lung	Temporal Bone Tumor
Total lipids—per cent of wet weight	3 91	2 25	2 25	14 4
Phospholipids—				
per cent of wet weight	1 81	1 00	1 13	0 94
(per cent of total lipids)	(46)	(44)	(50)	(7)
Cholesterol—per cent of wet weight	–	–	–	4 39
(per cent of total lipids)				(31)

Analysis of the soft orange mass which had destroyed and occupied much of the right temporal bone showed it to be an extremely fatty lesion, most striking for its cholesterol content—with a phospholipid normal for what would be found in most connective or stromal tissues

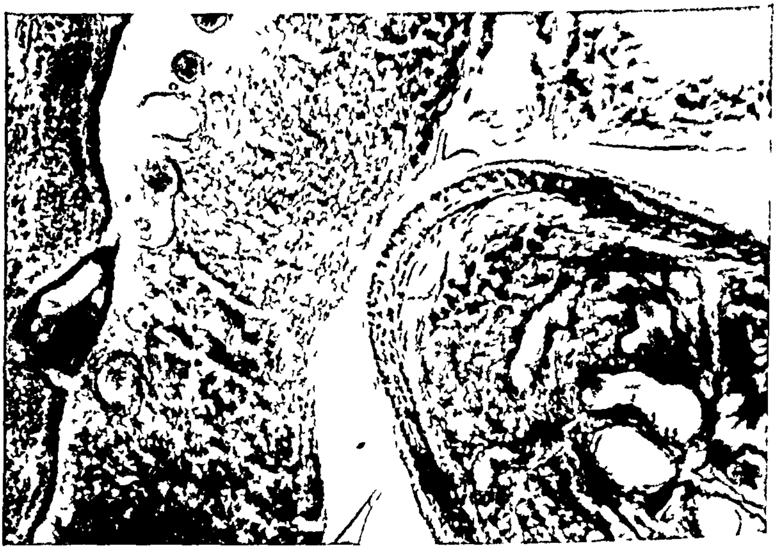

Fig 1 Gomori X50 Promontory with Jacobson's nerve high promontorial mucous membrane, granulomatous blocks suspended in exudate (upper right) tympanic membrane retracted ad maximum without adherence in external canal, granulomatous mass

The *right temporal bone* was obtained for detailed examination Decalcification needed about the usual time, embedding in celloidin, sectioning in the horizontal plane, staining with hematoxylin eosin Heidenhain Mallory, Sudan Scarlet-red, Gomori impregnation, with unstained sections mounted for polarized light followed

In the *external meatus* (see Fig 1) a thick mass completely filled the lumen Strands of desquamated epithelium were packed together by the granulomatous block which, *in vivo*, probably protruded in form of a polyp The tympanic perforation, if it was present at all, healed Retraction of the tympanic membrane brought it into immediate vicinity of the promontory but no adhesion developed

In the *middle ear* (see Figs 1 and 2) the osseous walls except the medial one, were attacked, resulting in severe destruction The lamellae of the tympanic tegmen still contained well stained osteocytes, its layers were pried apart by the advancing granulomatous mass the latter replaced one portion of the bony wall of the hypotympanum The mucoperiosteum was extremely high (see Fig 1) even for the young age, some of its very high papillae were branching, and exudate penetrated the interpapillary spaces High cushions were connected by slender stalks with their bases In the depth of the mucosa cysts in rosary formation bore evidence of the chronicity of the process while engorgement and cellular infiltration marked it to be of inflammatory origin Cysts were lacking in the niche of the oval window In the niche of the round window, invasion and organization were present side by side, the

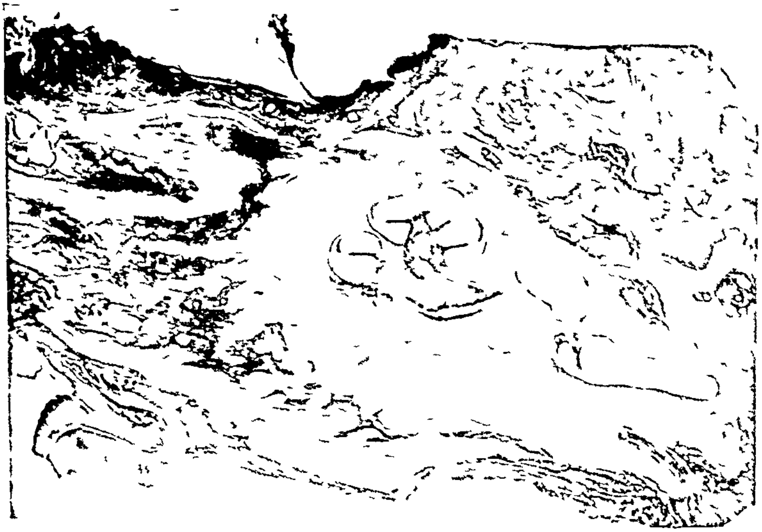

Fig 2 Gomori lupe Survey center and right lower intact inner ear right upper invaded middle ear with large ossicles filled by granulomatous mass left petrous apex filled by fibrous mass

granulomatous mass penetrated from the aperture, while a hyaline but still richly vascularized cushion in contact with the secondary tympanic membrane filled the upper medial corner of the niche The annular ligament was intact and so was the secondary tympanic membrane

The *ossicles* were completely surrounded by the invading mass (see Fig 2) The lateral circumference of the capsule of the incudo malleolar articulation was destroyed and the articular space was penetrated (see Fig 2) One corner of the incus was separated as a sequester from the main body The anterior crus of the stapes was bent in a right angle, near fractured possibly by pressure of the invading mass The latter broke at the stapes head through the cortex and filled a considerable portion of the head

All spaces of the middle ear were filled with an exudate of inflammatory character as shown by clusters of infiltrating cells especially dense in the tubal corner The granulomatous mass was surrounded by the exudate

Tissue	Liver	Spleen	Lung	Temporal Bone Tumor
Total lipids—per cent of wet weight	3 91	2 25	2 25	14 4
Phospholipids—				
per cent of wet weight	1 81	1 00	1 13	0 94
(per cent of total lipids)	(46)	(44)	(50)	(7)
Cholesterol—per cent of wet weight	–	–	–	4 39
(per cent of total lipids)				(31)

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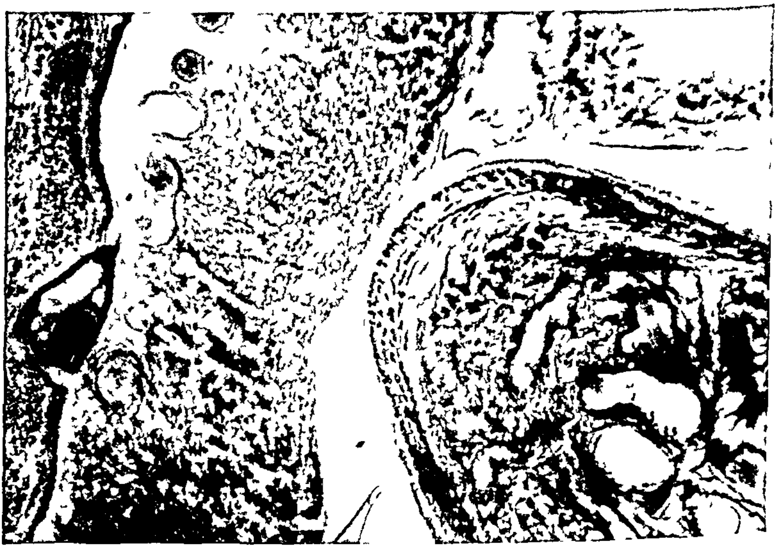

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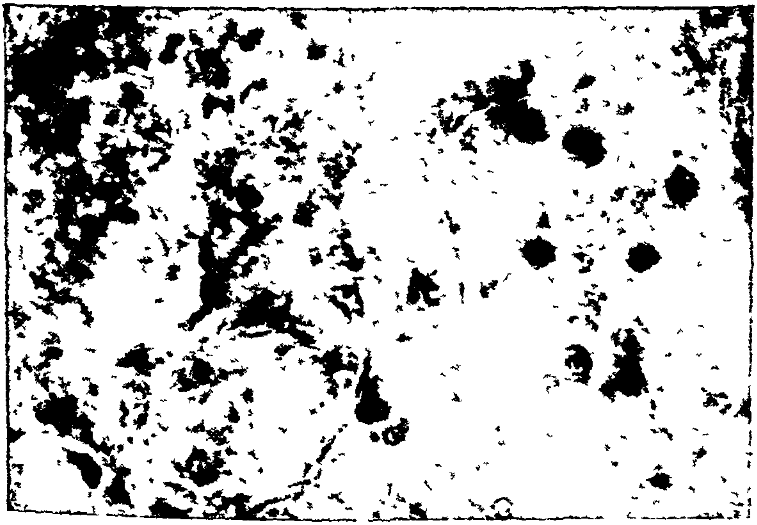

Fig 4 Hematoxylin-eosin X900 Same granulomatous mass as Fig 3

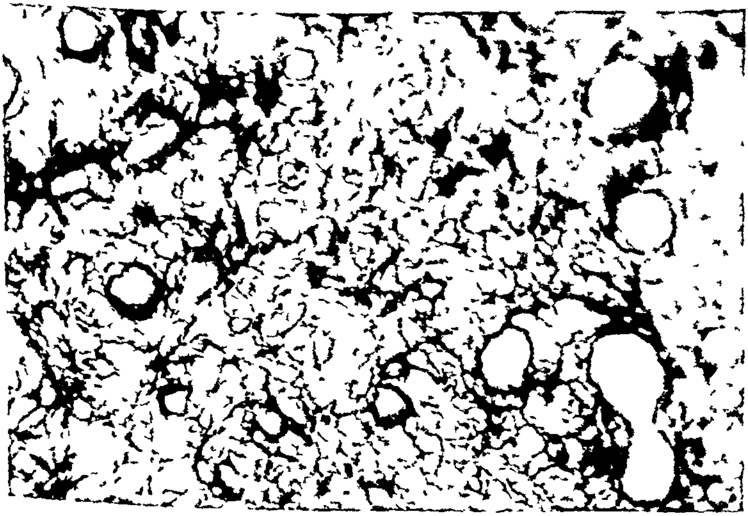

Fig 5 Gomori X250 Reticular mesh in the granulomatous mass the fibers arising from the vessel walls

and suspended in it (see Figs 1 and 2) This mass formed a solid wall around the ossicles, filled the niches, surrounded the facial canal The peritympanic pneumatic cells, like those in the tegmen, were opened by the granuloma, which broke down their septa and filled the lumina

The invading mass can be described as a granulomatous block with the cells arranged in long rows or sheets, suspended in a dense reticular meshwork and harboring clusters of lymphocytes (see Figs 3 and 4) Increase of the vascular bed transformed some sections almost into an angioma, the reticular fibers could be shown, by impregnation (see Fig 5) as arising directly from the vessel walls Palisades were formed almost exclusively by eosinophils among which, in isolated groups, giant cells were found, and—not too numerous—foam cells Double refracting elements, sparsely scattered, possibly represented very small bone particles on their way to resorption as a result of destruction

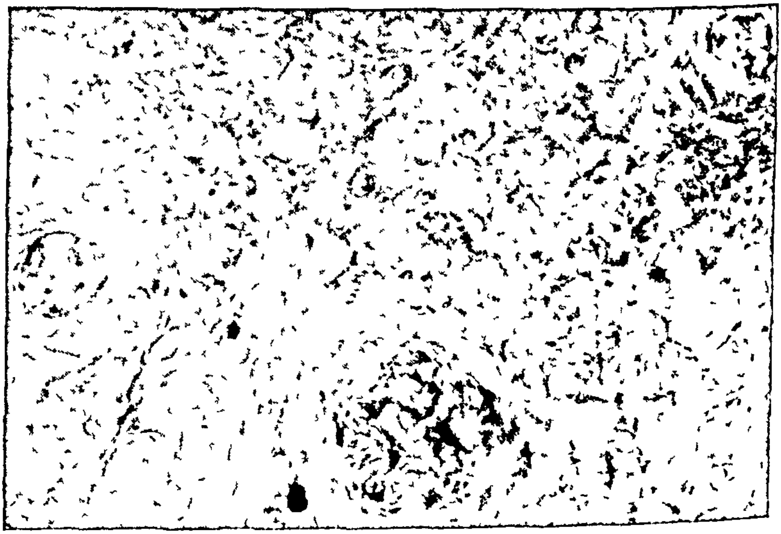

Fig 3 Sudan X250 Granulomatous mass in middle ear Cells in palisades sheets field of eosinophil leukocytes and histiocytes, giant cells partly *of foam cell character*

The *inner ear* remained free of invasion (see Fig 2) The otic capsule was intact in spite of penetration of the layers of the surrounding portions of the temporal Destruction started in the operculum, widening the space occupied by the saccus endolymphaticus

Between the carotid canal and the tip of the cochlea, and sitting athwart the cochlea like a cap, a focus was located of a character which can be best described as angiomatic or spongiotic (see Figs 6 and 7) It was sharply demarcated against the vicinity, and near the tip penetrated the cochlear capsule, stopping only outside the endosteal layer It consisted of dense conglomerates of small caliber vessels between the osseous lamellae The conglomerates did not completely replace the connective tissue of the marrow spaces and did not proceed to form blood lakes by confluence or cause bone destruction The connective tissue surrounding the vessels was best demonstrated by the Sudan stain showing perhaps, a relation to the basic disease

The fissular region contained a single large block of cartilage

The cochlear modiolus was completely filled, the nervous apparatus continuous The membrane of Reissner was distorted, in places bulging and, again, torn as by overstretching The tectorial membrane was displaced, without doubt artificially The organ of Corti (see Fig 8) was sufficiently preserved to show it to be not reached by the morbid process, and was of normal elevation with its elements well demonstrated inclusive of the hair cells and the hairs Guttulate formations in the tunnel and elsewhere within the organ of Corti were doubtless of artificial origin and not stained distinctly by Sudan The breaking open of the medial wall of the papilla of Corti with outpouring of cells and loss of the hair of the inner hair cell has been explained elsewhere (Kelemen, 1956) as originating during the fixation Nothing abnormal was seen in the vestibular end organs, parts of the vestibular endolymphatic space showed perhaps some degree of dilatation

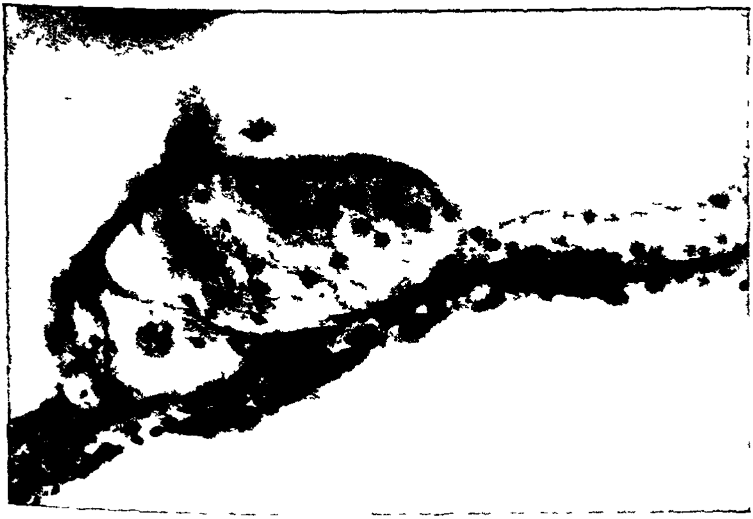

Fig 8 Hematoxylin-eosin X400 Organ of Corti in the middle coil Changes such as rupture of its medial wall and the guttulate formations are artificial Elevation tunnel well outlined cells show that in vivo the organ was normal

The inner *acoustic meatus* was free, with a few small psammomas in its dural lining In its anterior wall a granulomatous focus part of the penetrating mass, did not reach the surface of the canal wall

The *petrous apex* was completely invaded (see Fig 2) but by a very different material from that found in the middle ear All marrow spaces were tightly filled by a mass made up of fibrocytes and bundles formed by coarser or more delicate fibers The degree of vascularization was not excessive The bony septa still containing well stained osteocytes

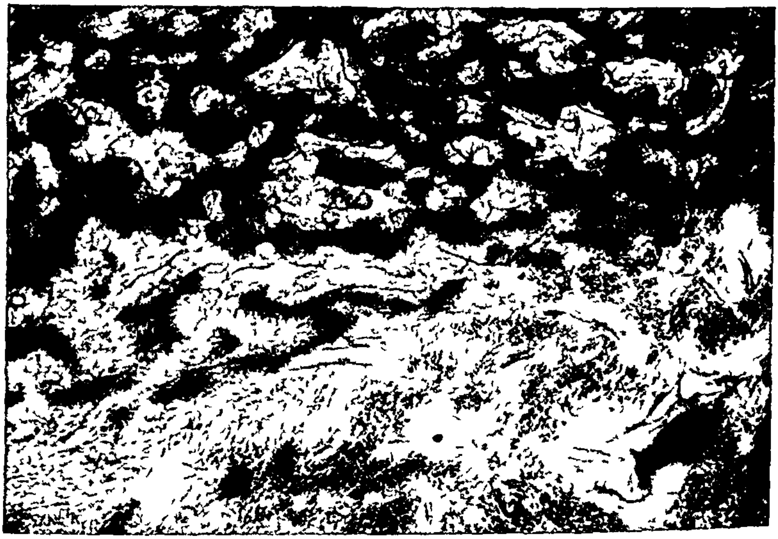

Fig 6 Heidenhain-Mallory X250 Upper half Spongiotic focus at tip of the cochlea Lower half Normal labyrinthine capsule of the apical coil

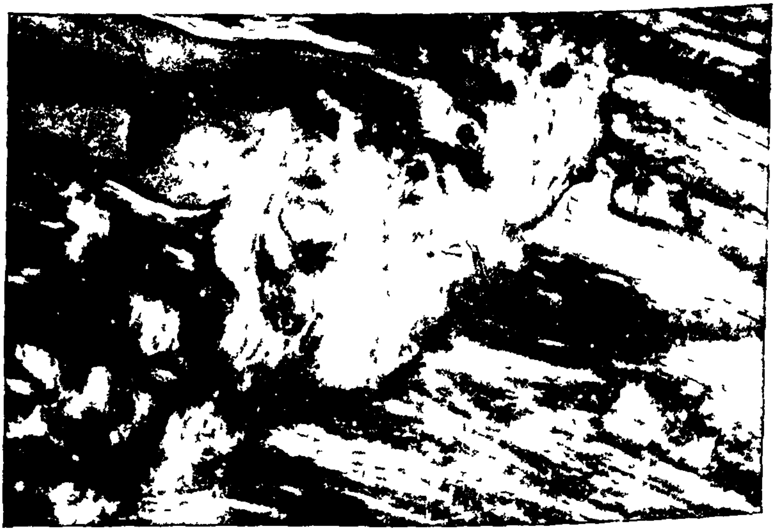

Fig 7 Unstained polarized light X100 Sharp boundary between the spongiotic focus and the normal labyrinthine capsule

of the septa of the air cells The otic capsule, with its content, remained intact Around the cochlear tip a sharply demarcated angiomatic focus was observed in the bone

The findings must be discussed within the framework of *a* the case history; *b* comparable reports on pathology in the hearing organ, *c* case reports without complete temporal bone analysis, and *d* general knowledge on lipid granulomatosis of the bones

a In the present case history no exophthalmos was noted It is known that other constituents of the syndrome, too, may be lacking Steurer (1952) reported absence of exophthalmos *and* diabetes insipidus Both, exophthalmos and diabetes insipidus, depend on pressure of the advancing mass, the direction and progression of which are irregular and unpredictable

Non-involvement of the nervous apparatus of the inner ear depended probably on mechanical protection by the resistant otic capsule

The role of the measles virus, found in autopsy material, is hard to evaluate, the symptoms were probably covered by the severe basic condition Measles may have been at the root of the intercurrent otitis media which left signs of chronicity the rosary of submucous cysts (Kelemen, 1958), the hyalin mass in the niche of the round window, the closed tympanic perforation (in case there was any present at all) Doubt regarding the direct effect of the virus on the sensory structures arose when no pathologic changes as found by Lindsay and Hemenway (1954) could be observed

b The case of Druss differed from the present one in the lack of invasion of the middle ear; the latter was traversed merely by fibrous strands, and contained a small amount of purulent exudate On the other hand the dura was diffusely infiltrated, while here it was thickened, but uninvaded The two cases exhibited identical features in the osseous parts Druss described the marrow spaces throughout the temporal bone, inclusive of the petrous apex, as diffusely infiltrated with the lipid granulation tissue, which, to a great extent, underwent fibrotic transformation Spindle cells containing were attacked from every side, with their substance ultimately ground up into small sequesters (see Fig 9) In spite of the abundant supply of fibrocytes the latter did not line up into rows of osteoblasts, on the other hand, in eroded lacunas, cells were nesting which could be termed osteoclasts by their content of debris The area of fibrous replacement, going around the otic capsule, reached the base of the high mucous membrane of the middle ear (see Fig 2)

The walls of the *carotic canal* remained intact, the pericarotic plexus showing engorgement The *dura* was very thick but without signs of an acute inflammation The *nervous* apparatus (VIIIth nerve and its ganglia, the facial, the Gasserian ganglion) was not invaded

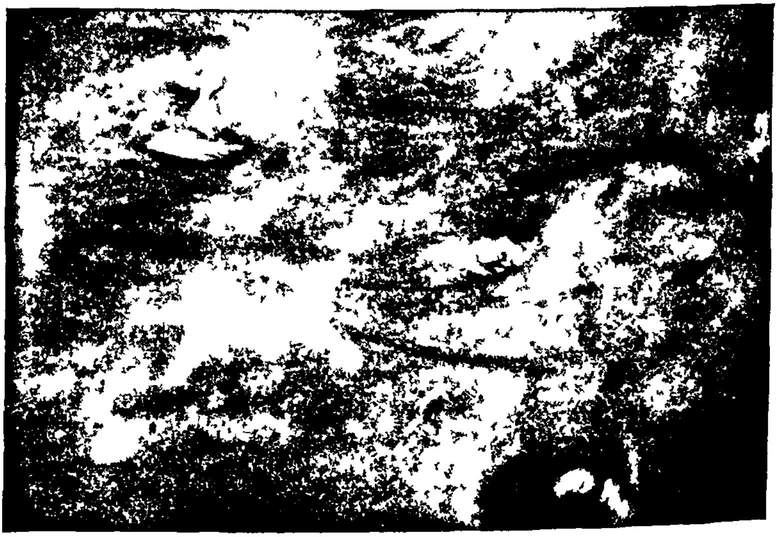

Fig 9 Unstained polarized light X50 Petrous apex filled by fibrous mass in which smallest sequesters are suspended

COMMENT

The foregoing gives histopathological findings in a hearing organ which participated in a generalized lipid granulomatosis involving the scalp, other skin regions, skull, pituitary, both lungs, epicardium and lymph nodes From the triad (albeit only moderately typical) skull defects, diabetes insipidus and exophthalmos, the latter was absent Remissions, concomitant otitis media, and measles in the terminal period were recorded Granulomatous masses filled the external and the middle ear, causing deep destruction of the bone, in the petrous apex complete fibrotic replacement was present, with sequestration c Reports on comparable cases, but without complete histologic analysis of the temporal, contain numerous instances of successful X-ray treatment with arrest or healing up to roentgenological normalcy of the affected bones Shea (1938) emphasized and was supported in his conclusion by Schall, that surgical interefeience should be avoided Here, the relatively benign course of the Hand-Schueller-Christian phase with multiplicity of clinical and histological features may have been on its way but did not develop sufficiently fast to counteract the acute and fatal Letterer-Siwe phase

Schuknecht and Perlman found that while the labyrinthine capsule is rather resistant to invasion by carcinoma, this is not the case in Hand-Schueller-Christian lesions Nager mentioned that participation of the labyrinthine capsule in this disease was assumed by X-ray findings, with a tentative localization in the periosteal layer Schuknecht and Perlman, discussing differential diagnosis, stated that a very active eosinophil granuloma may produce pictures identical with osteomyelitis of the skull Steurer's case was diagnosed, preoperatively, as a cholesteatoma Hertle (1956) reported two cases of frontal bone affection appearing under identical clinical manifestations, one turned out to be osteomyelitis, the other an eosinophilic granuloma Falkinburg, Littleton, Hunt and Horwitz (1959) published the fourth case with bilateral xanthomatous lesion of the mastoids

Histologically, here, absence of any reaction in the form of bone apposition discriminated sharply against osteomyelitis with its exuberant osteogenesis amid the most severe inflammatory-suppurative manifestation, right up to the final sequestration (Kelemen, 1957, Meltzer and Kelemen, 1959) Goodhill found the resistance of the capsule of the bony labyrinth to invasion by granuloma remarkable among destruction of other bones of the body, including the ossicles, the annulus tympanicus, the mastoid and the petrous tip The modiolus of the present case was free of the periostitic reaction to which Brunner (1928) called attention in Gaucher's disease

According to Dingley (1952) the middle ear is bypassed, at least until a late stage, and non-involvement of facial nerve lipid substance were scattered throughout this tissue Contrary to the findings in the present case, there was evidence of new bone formation but no bone resorption around the margins of the marrow spaces

The case of Goodhill presented infiltrations in all pneumatized portions of the temporal bone, including the mastoid and the petrous apex No mention was made of any mass in the tympanic cavity, the infiltration was restricted entirely to the cancellous bone structure and cellular trabeculae of the temporal, with areas where either reticulum cells, or foam (xanthoma) cells, or eosinophils predominated

Filling of the tympanic cavity by granulomatous blocks, suspended in exudate, was seen only in the present case Druss found a perforated tympanic membrane, through this, parts of the content of the middle ear could have been washed out in the course of the histological preparation

The most conspicuous finding in all three cases remains the intact otic capsule with all surrounding portions of the temporal invaded Infiltration of the temporal was described in the two other cases as uniform in character, while here the mastoid and tympanic region were invaded by a cellular-reticular, and the apical region by an entirely different, fibrotic mass Well stained osteocytes pleaded for a relatively recent date of invasion of the petrous tip, but X-ray showed many months before a "non-osseous" destruction of the pyramid

The entirely different nature of the invading mass in different locations emphasizes the recommendation of Dawes (1954) and of others to try to obtain adequate biopsy material, a single specimen may not show all the characteristic features of the disease

To consider fibrosis as a sign of regeneration was here impossible because of lack of new bone formation the mass of fibrocytes, instead of lining up as osteoblasts, behaved like a tumor, *e g*, a fibrosarcoma, destroying the bone by halisteresis

Christian phase, showed, within the temporal, features which could enroll it under either of the two headings For this reason it seems justifiable to retain the general term of "eosinophilic" or, as Goodhill suggested, "histocytic" granuloma "Histocytosis" was adopted here as one of the numerous synonyms offered by Thannhauser

Regarding *etiology*, Thannhauser asserted that, since an infectious cause was not found, a blastomatous origin of the systemic disorder might be considered Here, complete lack of osteogenetic reaction in the petrous tip recalled the type of destruction, with simple "melting away," caused by some tumors There were advocates of the concept to regard Letterer-Siwe lesions as a neoplasm related to leukemia Actually, as pointed out by Szanto and Saphir (1959), the reticuloendothelial system may react with products either of inflammatory or of neoplastic nature, differentiation between neoplasia and reactive hyperplasia is at times difficult or impossible on the basis of histology According to Farber the reaction of the bone marrow to the destructive process may be so striking as to suggest a tumor of the blood-forming tissue It is possible that in the future eosinophilic granuloma, mentioned, as against tumors, as a "tumor-like mass" (Schall and Kelemen, 1959) may attain status as a "true" neoplasm

The peculiar cap-like, sharply demarcated area in the bone athwart the cochlear tip resembled an angiomatous, spongiotic focus Cases of otosclerotic changes demonstrated histologically at a similarly early age are known although very rare The focus found here would represent a spongiotic phase In this connection it is of interest that Brunner observed Gaucher cells in the marrow spaces of an otosclerotic focus

In the *therapy*, X-ray, antibiotics, ACTH, and nitrogen mustard compounds were administered in long courses and were considered responsible for the long remissions The autopsy contained the remark that depletion of the bone marrow in all examined areas could perhaps be ascribed to the long-continued Cortisone This circumstance is of importance and labyrinth may perhaps be so explained On this basis the present case, with full invasion of the tympanic cavity, is to be classified as an advanced one

Labyrinth sequesters were repeatedly found In the case of Chisolm (1954) the middle ear was filled with granulations and a yellowish tissue, the ossicles were absent On this basis—in contrast when comparing with Dingley—the present case, with comparatively intact ossicles, should be classified as a more recent one

Escher (1950), after operation on a two-year-old girl, found deafness and loss of caloric excitability This is one example among many of loss of function through the severe process in the temporal

d General experience offers roentgenological evidence that the temporal frequently is the only affected region of the skull If the yellow-gray polyp appears in the external canal, the condition may be diagnosed from the ear alone Chisolm pointed out that one should become suspicious at any sudden appearance of an aural polyp in a previously normal ear

Within otolaryngologic discussion, Bartels (1950) considered the single symptom-groups as variants of the same disease The most outspoken advocate of a "monistic" theory for the entire group of diseases was Goodhill, who defined histiocytic granuloma of the skull as a clinicopathological entity which may appear in an acute, fatal form known as Letterer-Siwe disease, a subacute stage known as Hand-Schueller-Christian disease, and a chronic benign state known as eosinophilic granuloma He emphasized that the relative frequency of this group should be kept in mind in dubious cases Dawes was one, and cited others, who accepted this continuity on the basis of experience with cases of ear involvement The concept of different pertinent lesions as expressions of the same disease was accepted regarding manifestations in different parts of the organism (*e g*, McKay, Street, Benirschke and Duncan, 1953)

The present case, considered by the pediatricians as on the borderline between the Letterer-Siwe and the Hand-Schueller-massive crust and cerumen, but there is no recurrence, X-ray showing a clean operative cavity

Several data of this history may serve as a corollary to the findings in the case which is the main object of the present discussion the temporal, the only involved bone of the cranial base, even of the entire skeleton, lack of two components of the "classic" diagnostic triad, remarkable resistance of the tissue of the tympanic membrane surrounded by the products destructive to the osseous tissue, penetration to the external canal not through a tympanic perforation but by breaking through the bone of the canal wall, re-conditioning of the sound conduction apparatus after eradication of the offensive material, intact nervous apparatus of the inner ear as shown by the good bone conduction ten years after the operation

SUMMARY

One temporal bone of a two-year-four-month-old girl with eosinophilic granuloma, who died during the Letterer-Siwe and the Hand-Schueller-Christian phases, was examined in serial sections and compared with two others, the only cases previously reported in the literature

The tympanic cavity was filled with exudate and residuum from a concomitant otitis media, with the granulomatous products suspended in these secretions

The granulomatous mass spread to every section of the tympanic cavity and filled the external canal, it penetrated the perianthral and peritympanic cells after destroying their septa Reticular cells, eosinophils in strands and sheets, scattered giant cells and, in distinct groups, foam cells constituted the mass, all suspended in a reticular mesh Near-angiomatous vessel groups formed at many points The petrous tip was completely filled by a fibrous mass, very different from the content of the tympanic cavity, it contained, between the fibers, dense masses of fibrocytes which, however, did not line up to form osteoblastic seams Signs of new bone formation were absent The otic capsule and its contents were intact, as conforms with the two other published cases A well demarcated angiomatic or spongiotic focus in the capsular bone surrounded the apex of the cochlea like a cap

The histopathological findings supported the monistic concept according to which the single groups of symptoms in this sequence of diseases represent only different phases of the identical basic affection

in explaining the invasion of the pneumatic spaces in the temporal, while the otic capsule, poor in marrow, remained unharmed

Regarding *prognosis*, the findings in the temporal had shown healing tendency only in the tympanic membrane, rather peculiarly, between granulomatous masses at both sides and this only if a perforation existed, as the discharge may have escaped via progression through the canal walls This would offer another remarkable instance of higher resistance of soft as against bone tissue Fibrosis in the petrous failed to act as a cicatrization, and here the mass acted more like a tumor Had the patient been operated upon, the greater portion of the temporal might have been found—as in numerous reported instances—in the form of a sequester including the still possibly anatomically intact otic capsule

The short outline of the history of a recently observed case shall be added here in which diabetes and exophthalmos were absent

A 16-month old girl was admitted to the Massachusetts Eye and Ear Infirmary (M G H 708722) with a history of an acute episode at the right ear which developed three months previously with fever, anorexia, listlessness, and a profuse discharge from the ear, a polyp was removed but reformed quickly She was referred for an operation At admission the skin was found to be of a yellow (carotinoid) hue, a raised, yellow brown, papulous area was seen in the left axilla The left ear was clear The right canal was bathed in pus, from which coagulase positive staphylococcus aureus and B proteus were cultured X ray showed complete destruction of the entire mastoid process inclusive of the sinus plate and suggested the possibility of Hand Schueller Christian's disease Complete skeletal X ray revealed no bone lesions WBC 13,400, hemoglobin 8 6 grams per cent, polys 31 per cent lymphocytes 61 per cent, monocytes 1 per cent, eosinophils 6 per cent, basophils 1 per cent She had a moderate anemia, blood cholesterol was within normal limits Modified radical operation (Dr Edgar Holmes) revealed a very firm invasive fibrous tissue beneath the mastoid cortex, occupying the mastoid completely and resembling a tumor The sinus was covered by the fibrous, tumor like tissue The posterior canal wall was almost completely destroyed Polypoid tissue was removed from the epitympanum and the incus was extracted The dura, exposed over the tegmen of the antrum, has shown no discoloration and no pus under pressure was found under it The drum did not show any perforation A skin flap was created from the external canal wall to cover the operative cavity Specimens of the mass were diagnosed histologically as eosinophilic granuloma X ray treatment was inaugurated Eight years after operation the hearing was normal in the left ear and in the right, air conduction showed for the six tested frequencies, 15, 20, 25, 20, 40 and 30 dbs with a practically normal bone conduction At the time of this writing, ten years after operation, the cavity must be cleaned periodically from

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THE FALSETTO. A HIGH SPEED CINEMATOGRAPHIC STUDY.*†‡§

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WITH THE TECHNICAL ASSISTANCE OF
MAURICE LE COVER

Centuries ago the old Italian singing masters recognized two registers of the human voice, the chest and the falsetto or head, the latter two being synonymous. With the invention of the laryngeal mirror it became possible to supplement acoustical impressions with direct observation, and individuals of varying degrees of knowledge, training, and skill recorded their findings and interpretations. The effect of this was to throw the matter of registers into almost hopeless confusion by the introduction of new and often contradictory concepts and terms.

Within the past 100 years the falsetto has been described by vocal pedagogues as a definite register identical with the head voice, as a register distinct from the head voice and situated above it, and then again as below it, and as not being part of the singing voice at all. Since the term falsetto conjures up a variety of musical images, it is imperative that any discussion of the falsetto be prefaced by the author's explanation of what it connotes to him, so that one major source of confusion can at once be eliminated.

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§A sound motion picture illustrating these studies may be obtained from the senior author upon request.

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243 Charles Street (14)

DISTINGUISHED SERVICE AWARD GIVEN TO NEARLY DEAF EAR PHYSICIAN

Dr Kenneth M Day of Pittsburgh, Pa, is recognized by his medical colleagues as one of the most successful ear physicians in the country

He attributes his success to the fact that he has been nearly deaf himself for many years, and has served as a model of what the hearing handicapped can really do

In honor of his service to medicine and mankind, Dr Day received a distinguished service award May 20, 1960, from the Pennsylvania and New Jersey Academies of Ophthalmology and Otolaryngology at a joint annual meeting in Atlantic City, N J, attended by eye, ear, nose and throat physicians from six middle Atlantic states larly the female, a speed of 4,000 frames per second is not enough to allow detailed analysis, furthermore, such a study of the singing voice would seem to require subjects with trained as well as untrained voices, not only for comparative analysis but also in order to assure controlled performances in the fullest possible range and dynamics of the pure falsetto

This report will evaluate conclusions drawn from the films of the Bell Telephone Laboratories in the light of additional high-speed photographic evidence It will also clarify other features of the falsetto which heretofore have not been demonstrated These include *1* the mechanism of the so-called falsetto "break", *2* the nature of vocal cord activity within the falsetto itself during changes in pitch and intensity, *3* the means by which the trained singer effects smooth transition from the fundamental register to the falsetto

METHOD OF INVESTIGATION

Motion pictures of the vocal cords of selected vocalists were taken by indirect laryngoscopy at 6,000-8,000 frames per second with a Wollensak Fastax camera The technique, which is the subject of a separate communication,[4] constitutes a radical departure from that devised by the Bell Telephone Laboratories in that the laryngeal mirror is freely manipulable rather than fixed The elusive falsetto "break" was captured by utilizing film in 400 rather than the usual 100-foot rolls Since this allows three-and-one-half to four seconds instead of only one second in which to photograph a phonatory sequence, it was possible to encompass the "break" or any other isolated phenomenon Voices were recorded simultaneously by a microphone applied to the neck over the thyroid cartilage

The subjects were voice students in varying degrees of training from the department of music at the University of Southern California and professional singers of the authors' acquaintance The vocalists were required to produce a legitimate falsetto, possess a larynx capable of easy visualization, and be able to tolerate a laryngeal mirror These criteria were enough to exclude many otherwise vocally qualified

Fundamental changes in the mechanics of phonation occur at points throughout the singer's range These are manifest either by an audible "break" or "crack" in the relatively untrained singer, or by a less violent shift in the more experienced singer, as he attempts to accommodate to the physiological and/or the acoustical demands of the new pitch

One such production change is most apparent as the voice enters its upper reaches and assumes a less complex and a lighter quality For the purposes of this discussion the falsetto is defined as *that portion of the voice which encompasses this upper singing range*

The vocal cords in falsetto vibrate so rapidly that the actual nature of their movements can only be conjectured on indirect laryngoscopy, nonetheless, observers in the last century, handicapped as they were by relatively primitive methods of visualization, agreed on two aspects of the falsetto which more refined methods of examination have proven to be substantially correct first, that in the falsetto a smaller amount of thyroarytenoid substance is thrown into vibration, second, that in the falsetto, the vocal cords are activated by a less powerful air blast Any further considerations prior to the era of high-speed photography soon bog down in a morass of controversy, and the interested reader is referred to excellent presentations by Mackenzie[1] and Negus[2]

It was not until the Bell Telephone Laboratories[3] investigated the mechanism of the larynx by high speed photography that real objectivity was possible According to the brochure accompanying their motion picture the films show that in the lower register the thyroarytenoid muscles strike against each other in phonation with varying force whereas in the falsetto only the margins vibrate, and complete closure is usually not attained It is also inferred from this study that there is no significant difference between the production of the falsetto and that of a low intensity sound

Superb as these high-speed motion pictures are, their scope in the study of the falsetto is limited since camera speed did not exceed 4,000 frames per second, and only untrained voices were studied In the higher reaches of the falsetto, particu-

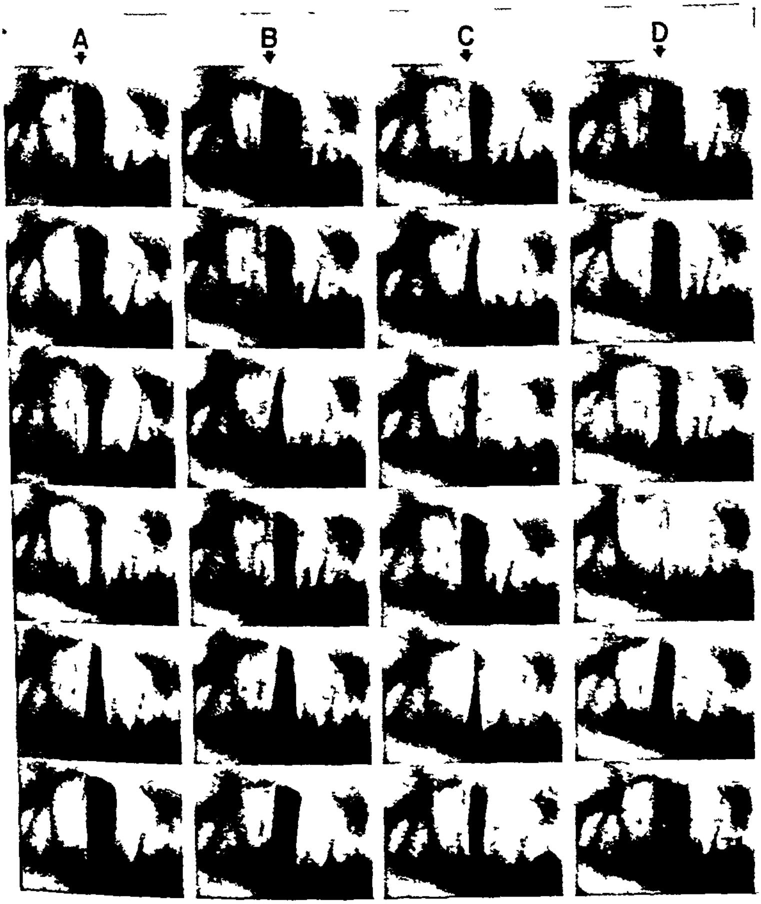

Fig 1 Falsetto break At the moment of transition from fundamental to falsetto or vice versa, the vocal cords are not under control and vibrate without any definite pattern

participating in the vibratory activity This is the picture of pure falsetto The same occurs in reverse as the voice passes from falsetto to lower register

What transpires in this sequence is easily deduced In the uppermost reaches of the fundamental register the vocal cords are in a state of maximal tension Opposing them are the antagonistic cricothyroids which have approximated their points of origin and insertion almost to the utmost Tension is so great that further elevation of pitch by extension of this subjects Of those screened, 15 men and 17 women proved acceptable, and their efforts form the basis of this report

FINDINGS

The Falsetto "Break"

One of the aspects of the voice which has eluded analysis is the so-called falsetto "break," a term applied to the sudden change in quality of the voice as it passes from the lower register to the falsetto and vice versa A prime object of vocal training is the covering of this "break" so that a smooth transition may be effected High-speed films at the moment of transition reveal a sequence which is constant in both sexes at varying levels of intensity and the same whether the "break" occurs on a glissando or on one pitch Variations in appearance are related only to the degree of vocal skill

In ascent or in transition on the same pitch (see Fig 1) the vocal cords are first seen vibrating in the manner characteristic of the lower register, striking each other regularly along their full length and participating with their entire mass in vibratory activity, but very taut because the pitch is high As the "break" is reached, the movements become irregular and without any discernible pattern, the vocal folds wobbling loosely without touching each other, resembling banners waving in a breeze The picture is one of complete chaos The vocal cords of the untrained vocalist may actually come to a halt as the air stream momentarily stops while the neophyte strains to negotiate the "break" In the trained vocalist this period of lack of control is short-lived, and the manner in which the "break" is avoided altogether in the glissando will be discussed later

After a varying period of irregular movement, a quite different pattern of vibration emerges The vocal cords again move in phase and regularly but with a changed overall appearance They vibrate at the same or approximately the same rate but touch each other lightly or not at all, moreover, their margins are quite thin, and the main mass of the thyroarytenoid muscles lies laterally and does not appear to be

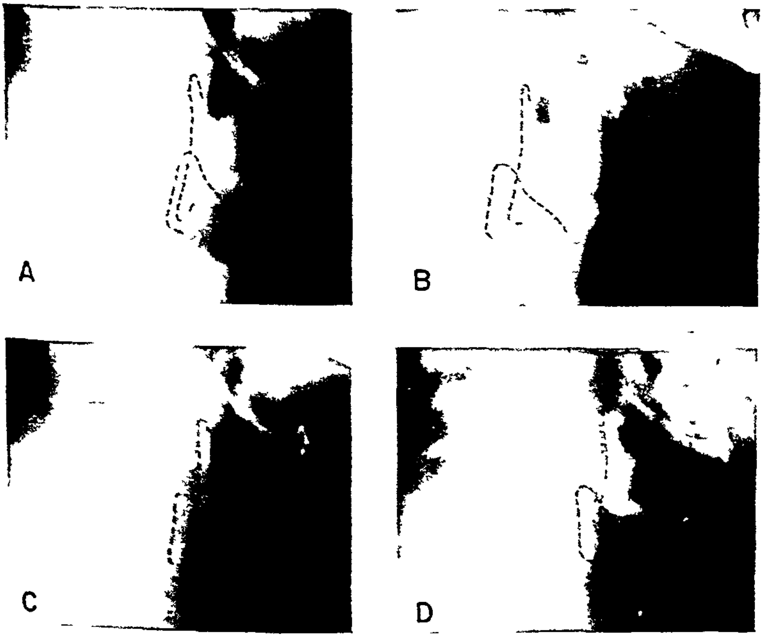

Fig 3 Lateral X-rays of the larynx in a trained singer The glottic chink elongates in passing from low to high fundamental registration not at all in the falsetto break and little or none in the falsetto Legend A—Low fundamental B—High fundamental C—Low falsetto D—High falsetto

state of tonic contraction sufficient to withstand the air-stream yet not enough to bring them together or at best only lightly in the midline Only the margins now vibrate This presents a thin, poorly supported resistance to the air column and, as a result the cordal edges are forcibly separated in each vibratory excursion before they are able to make any or more than light or partial contact with each other

Lateral X-rays of the neck in low fundamental, high fundamental, low falsetto, and high falsetto reveal that there is considerable elongation of the glottic chink in passing from low to high fundamental There is, however no further lengthening in transition to falsetto and little or none within the falsetto itself (see Figs 2 and 3)

TYPES OF FALSETTO ACTION

Some vocalists presumably have no falsetto, and one is same mechanism is impossible, and a quick muscular readjustment takes place The thyroarytenoids relax and in so doing are blown about desultorily The glottic chink in the poorly trained vocalist is prevented from being elongated precipitously at this moment from unopposed cricothyroid pull, because the latter muscles are already in a state of almost maximal contraction from having previously counteracted the thyroarytenoids

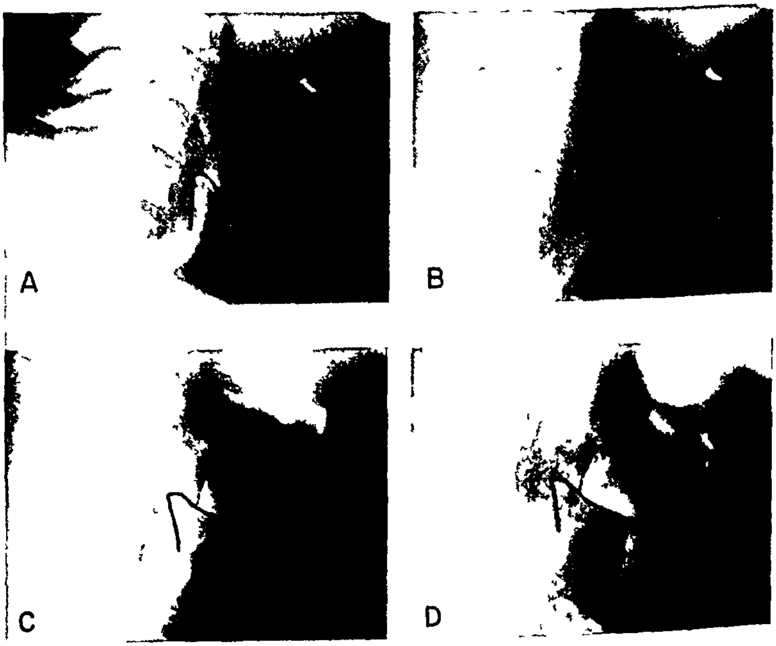

Fig 2 Lateral X-ray of the larynx in an untrained singer Maximal elongation of the glottic chink occurs in passing from low to high fundamental registration very little in the falsetto Legend A —Low fundamental B —High fundamental C —Low falsetto D —High falsetto

After a variable period of uncoordinated or poorly coordinated movements, the pure falsetto vibratory pattern appears The main mass of the thyroarytenoids now lies laterally and no longer vibrates It would be incorrect to assume that they are completely relaxed because if this were true, they would continue to be blown about as in the "break" or as is often seen at the conclusion of phonation They are rather in a

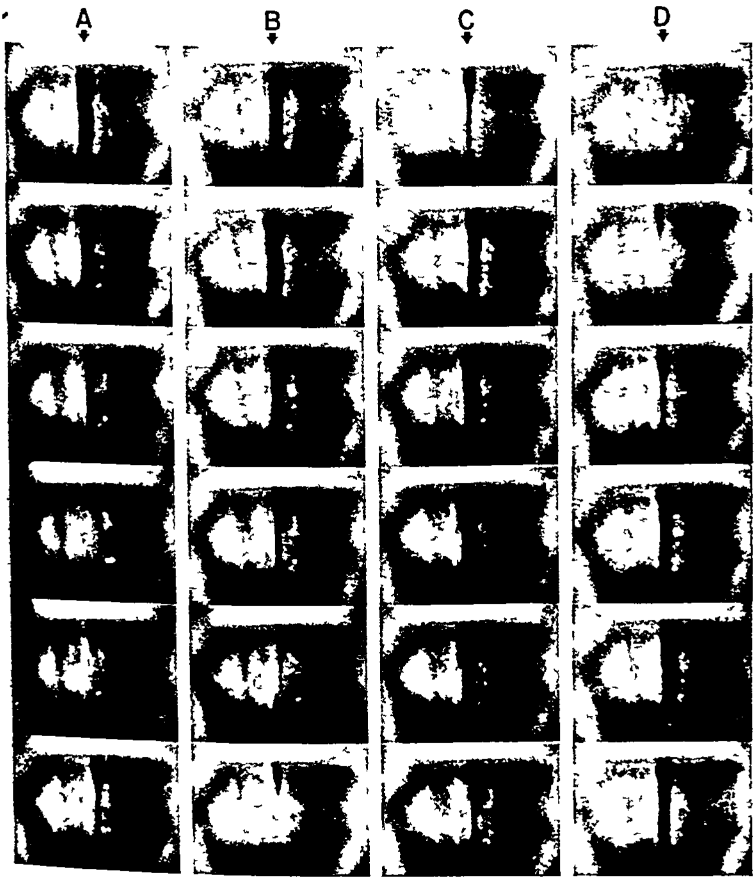

Fig 4 Closed-chink falsetto mechanism The vocal cords touch each other in vibration but there is relative inactivity of their lateral portions

is relatively less active, principle participation being by the medial fibers

In well-trained voices, particularly the female, because of superb control of the thyroarytenoids, there is sometimes difficulty in differentiating this "closed-chink" action from the upper levels of the lower register, and only the tape recorder will suggest how the tones were produced Even then it may be impossible because the skill of some vocalists is so great that the listener is deceived and cannot at all be certain of elicited with difficulty, if at all Others have a falsetto but never use it or do so only occasionally and for special effects Still others have a wide range and are capable of singing in the falsetto with exquisite delicacy and beauty

The physical basis of the falsetto is revealed by high-speed photography to be an astonishingly complex series of intra-laryngeal muscular adjustments Common to all of them and distinguishing the falsetto from the fundamental register is the lesser amount of thyroarytenoid substance thrown into vibration by the air column By maintaining a high degree of tension in the medial marginal fibers of the thyroarytenoids and by relative relaxation of the main muscular mass so as to avoid its directly confronting the air blast, the higher pitch and thinner quality of the falsetto are produced There are several ways in which this is accomplished, and, despite much overlapping, three basic patterns of vibratory activity can be identified One or two or all three may be present in any one voice

The first of these has already been described in connection with the falsetto break (see Fig 1), and is characterized by vibrations in which the vocal cords touch each other infrequently or not at all This "open-chink" mechanism can never be confused with any part of the fundamental register and to see it in a high-speed sequence is to know that the subject must be singing falsetto Since the main mass of the thyroarytenoid muscles is selectively relaxed, only the thin cordal margins are thrown into vibration by the lessened air pressure from below This particular mechanism is complete within itself, and the entire falsetto range may be encompassed by it at soft and moderate intensities Sometimes noted in this action is an anterior to posterior wave-like rolling or "rocking horse" movement of the vocal cords in which, within the same vibratory phase, the posterior halves of the vocal cords are separating as the anterior are coming together

The second and most common of the vibratory patterns in those voices studied is one in which the margins of the vocal cords strike each other as in the fundamental register (see Fig 4), however, the main mass of the thyroarytenoids air pressure is required to push them laterally, and a greater drop in air pressure occurring at this instant allows the vocal cords to make contact again The cycle is repeated at high frequency down through the falsetto

In a number of singers, particularly females, a phenomenon was noted in high falsetto and occasionally in high fundamental registration which has not been previously described This is horizontal ridging on the upper surfaces of both vocal cords, the light reflexes at times being so pronounced (see Fig 5) as to resemble a stepladder Frequently associated with this are irregularities of the cordal margins (see Fig 6) giving rise to a scalloped appearance during vibration This

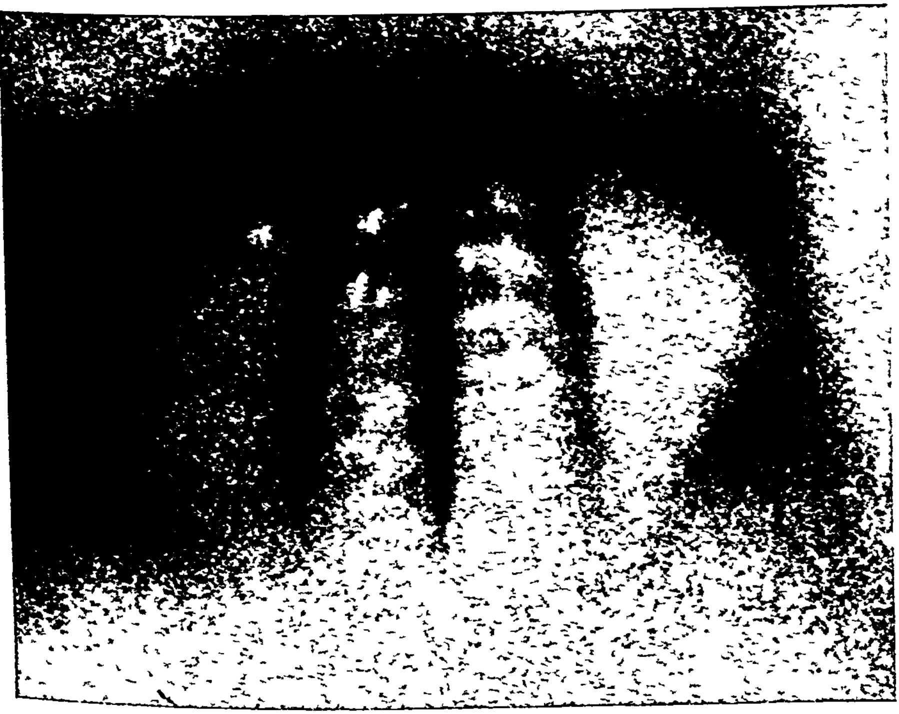

Fig 5 Ridging in a female vocalist singing high C At high frequencies mucus not usually seen on the superior surfaces of the vocal cords is sometimes aligned in visible parallel strands which reflect the light and cause a stepladder appearance

the registration For the most part, however, they can be distinguished on film

Actual measurement of the glottic wave vibrations by high-speed photography in both head and chest registration were recorded by Timcke, et al,[5] and vocal cord movements within each vibratory cycle described in terms of an Open Quotient and a Speed Quotient These formulas are a real contribution in providing a framework for objective analysis of high-speed film sequences A detailed consideration of our films by this method, which requires careful attention to specific pitch and intensity, will be the subject of a separate communication

Suffice it to say here that the vocal cords in the "closed-chink" type of falsetto action appear during projection at usual running speed to be in contact with each other for a shorter period than at comparable intensities in fundamental registration, and they give the illusion of springing apart more quickly after contact It is as though in the resting state the vocal cords would lie together in fundamental and apart in falsetto

In fortissimo the usual falsetto action is closed-chink, the vocal folds being pushed laterally to such an extent by the blast that they bounce together on the rebound A certain number of these will revert to open-chink when the intensity lessens

A delicate balance may be achieved between the open-chink and closed-chink types of falsetto action, which are really only degrees of the same basic mechanism, the two alternating with each other in producing a single sound This represents the ultimate in balance between tension of the cordal margins and subglottic air pressure In this situation the air stream forcibly separates the apposed vocal folds, but the frequency of vibration is so high that the margins spring back toward each other before enough air has escaped to allow them to come together They are thus held apart until subglottic air pressure again reaches the point where they are blown apart Since they were not approximated tightly in this phase, less

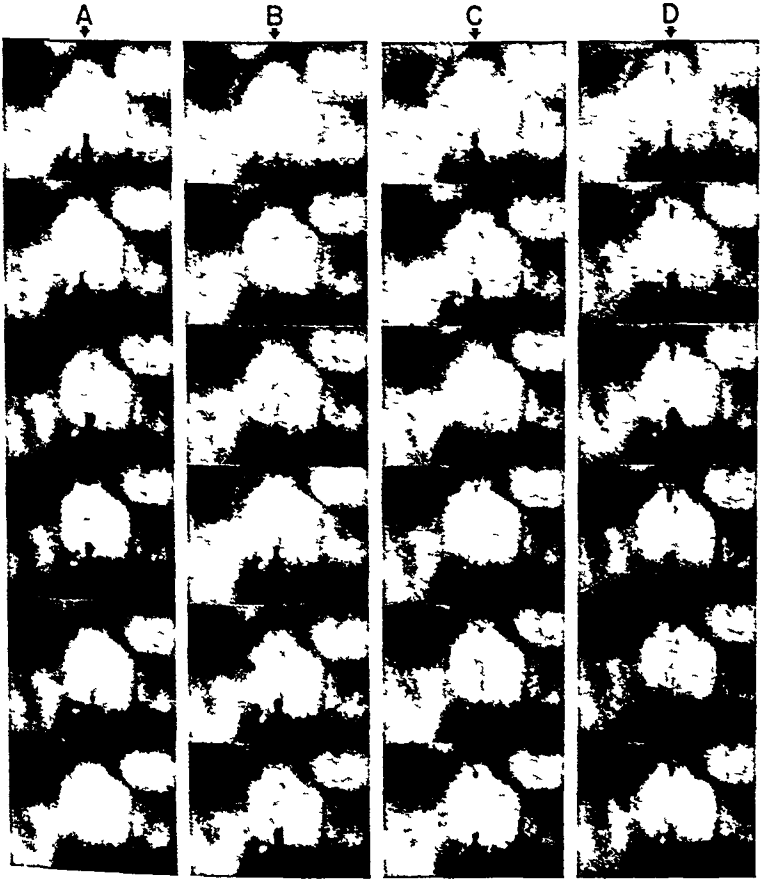

Fig 7 Damping In going through the falsetto to its upper reaches elevation in pitch is produced in some vocalists by approximation of corresponding segments of the vocal cords progressing posteriorly to anteriorly This sequence shows damped cords opening in descent

by the other two mechanisms It is accomplished by progressive closure of the glottis from posterior to anterior as corresponding segments of the vocal cords appose tightly As the vibrating cordal margins become correspondingly foreshortened, the pitch goes up and is finally reduced to a squeak emitted through a pinpoint opening at the anterior commissure (see Fig 7) Beyond this the larynx closes in spasm This type of falsetto action was recognized in the last century and called stop-closure by Mackenzie[1] It was documented by was at first thought to represent an unusual type of configuration occurring at high frequencies, but close study revealed it to be due to mucus In some subjects, at very high rates of vibration, the thin layer of mucus on the superior surfaces of the vocal folds aligns itself in parallel strands which reflect the light As the cordal margins spring apart during rapid vibration, this mucus is left behind momentarily because of its inertia, thereby giving them a somewhat irregular or saw-tooth shape

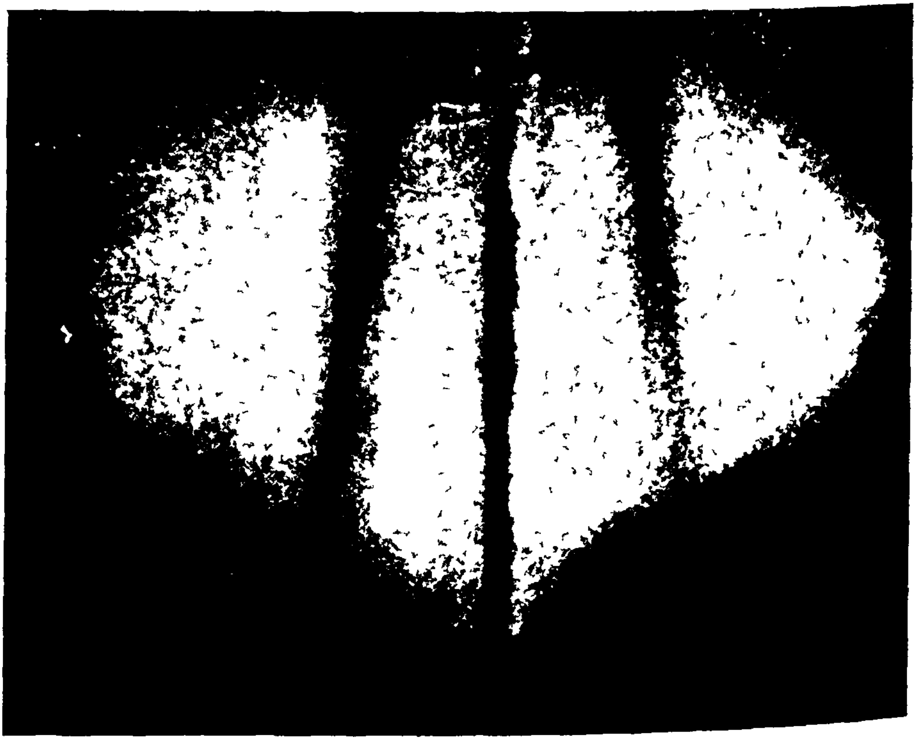

Fig 6 Scalloping in a female vocalist singing high C At high frequencies mucus not visible at lower rates of vibration sometimes produces an irregular appearance of the vocal margins

The third type of falsetto action is stop-closure or damping This becomes operative in the highest reaches of the falsetto in some voices and allows the pitch to rise beyond that possible

COORDINATION

The discussion thus far has related to the falsetto break and the falsetto itself There are some vocalists in whom no break is discernible and whose entire range seems to be encompassed in one register These voices flow smoothly and evenly from full voice to unmistakeable falsetto with no trace of abrupt change in quality Sometimes in "dissecting" this type of voice, as it were, for photographic purposes, years of training can be temporarily undone by forcing the subject to vocalize in such a way as to cause the long concealed falsetto break to reappear Other voices withstand all efforts to reduce them to a more elementary state These observations are not new, but the mechanism by which a smooth transition is effected has only been hypothecated and not previously demonstrated

High-speed photography reveals the muscular coordination by which this is accomplished, and the thyroarytenoids are shown to be capable of the most subtle intrinsic adjustments Starting at either end of the vocal range there is such a gradual change in the appearance of the cordal vibratory pattern that it is necessary to make comparative observations at intervals within the film strip to appreciate them (see Fig 8) In ascending from the lower register at any one intensity, the vocal folds are first seen striking each other broadly, the entire substance of the thyroarytenoid muscles participating in the vibratory activity As the pitch rises, they become increasingly taut, but the entire mass of thyroarytenoid is in motion In the upper reaches of the lower register and in the area of what would be the falsetto break in the incompletely trained voice, the frequency of vibration continues to increase and, in the trained vocalist, there is a decreased activity of the most lateral portions of the thyroarytenoids Continuing into the highest reaches of the voice and into what is acoustically falsetto, there is no sudden abrupt change in the vibratory pattern At any given intensity, movement of the lateral aspects of the cords becomes less but does not cease The glissando scale, or arpeggio is affected so smoothly and with such control that no real transition may be visible the high-speed films of the Bell Telephone Laboratories and termed damping by Pressman[6] This mechanism is utilized in the high falsetto by some male singers and blends imperceptibly with that portion of the voice above that used in singing It is, however, by no means a necessary accompaniment of the high falsetto and was seen much less often in this study than the two previously described mechanisms Most of the male subjects encompassed the entire falsetto with the vocal cords vibrating in their full length, and damping was not seen once in the female Those male voices employing the falsetto with the greatest skill utilized damping at high frequencies

There may be more to damping than has been described, because it is impossible to visualize the larynx of certain male vocalists at these very high frequencies The larynx closes down, and the epiglottis is drawn inward into a covering position as the glottic resonating chamber assumes its smallest possible dimensions In one male vocalist ascending from middle C to F (f″) one octave and a fifth above, the vocal cords did not dampen posteriorly, but the laryngeal orifice was so contracted anteriorly that they were probably squeezed together in that area

Stop-closure or damping effectively raises the pitch by a combination of reducing the functioning length of the vocal cords and immobilizing more of their substance It is presumed to be due to the contraction of controversial interdigitating fibers within the thyroarytenoid muscles, but this remains as yet unproved Since there is an accompanying narrowing of the larynx in its lateral diameter, the explanation may lie in squeezing of the vocal folds by contraction of more laterally situated muscle bundles

It must be emphasized that the terms damping and falsetto are not interchangeable Damping correctly describes only one type of falsetto action and not the major one at that As such it can no more be used synonymously with the falsetto than can a part be substituted for the whole master of his thyroarytenoids and achieves the ultimate in delicacy of muscular control

FACTORS OF PITCH AND VOLUME

Pitch within the falsetto, just as in the fundamental, is primarily a function of tension of the vocal cords Lateral X-rays demonstrated little or no elongation of the glottic chink within the falsetto itself In the trained singer control of the thyroarytenoids is so expert that selective contraction of their fibers will effect elevation in pitch In the untrained voice, however, there is another and decidedly inefficient factor at work Control of the medial elements of the thyroarytenoids is so imperfect that they are inadequate for the regulation of pitch A rise is then accomplished simply by increasing the force of the expiratory air blast, thus forcing the vocal folds to vibrate at a higher frequency even though their state of tension remains unchanged Pitch may be raised an entire octave by this mechanism alone Because of excellent muscular control the well-trained singer may swell from pianissimo to fortissimo on the same pitch while the less expert will experience a rise in pitch

Volume in the falsetto is controlled as in the fundamental register by the force of the tracheal air column In the incompletely trained voice overblowing in the region of the "break" may cause the falsetto to revert to the lower register by throwing the entire muscle mass into vibration The trained singer, however, is in such control of his laryngeal musculature that the vocal cords withstand increased air pressure, whatever portion of the thyroarytenoid muscles participating in the vibratory action simply responding by greater lateral excursion The sound thus produced may be thinner because of a less complex overtone pattern and less powerful because of a smaller mass of moving muscle, but it can still be loud

TERMINOLOGY

Although this study has clarified certain features of the

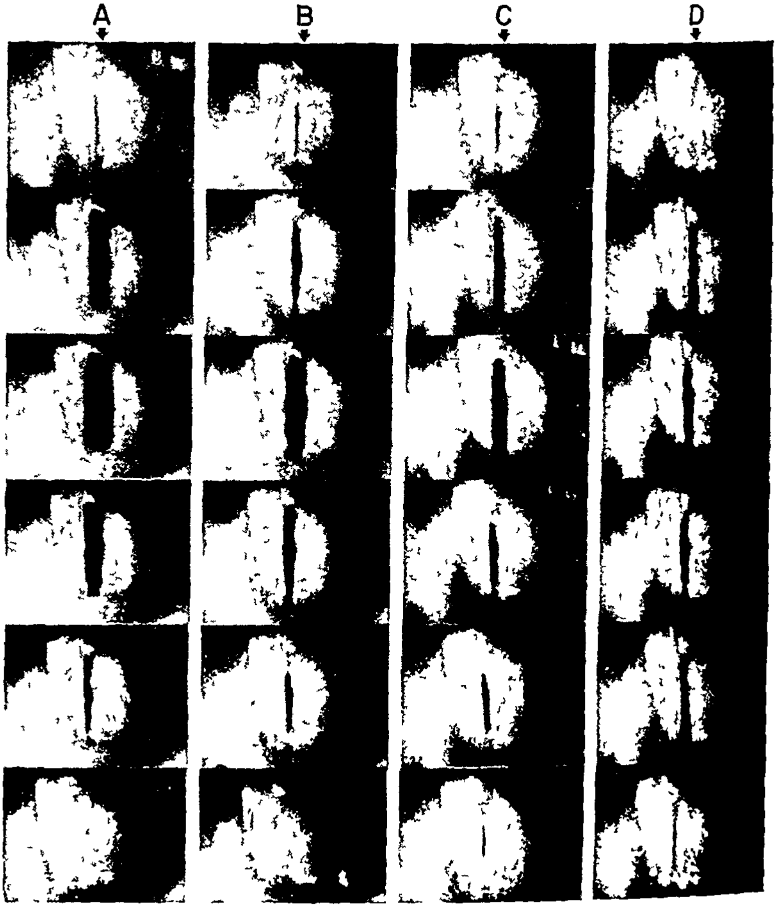

Fig 8 Coordination In well trained singers the voice passes from low fundamental registration to falsetto with no abrupt change in quality or appearance of the vocal cords

In others within the space of a few frames and with such subtlety that it can be easily overlooked, a quick shift from fundamental to falsetto mechanisms occurs The movements are so well controlled that there is no real "break" and a true blending of registers is achieved It is clear that in skilled vocalists upward extension to the higher reaches is affected at any given intensity by expert utilization of less thyro-arytenoid substance The accomplished singer is the complete master of his thyroarytenoids and achieves the ultimate in delicacy of muscular control

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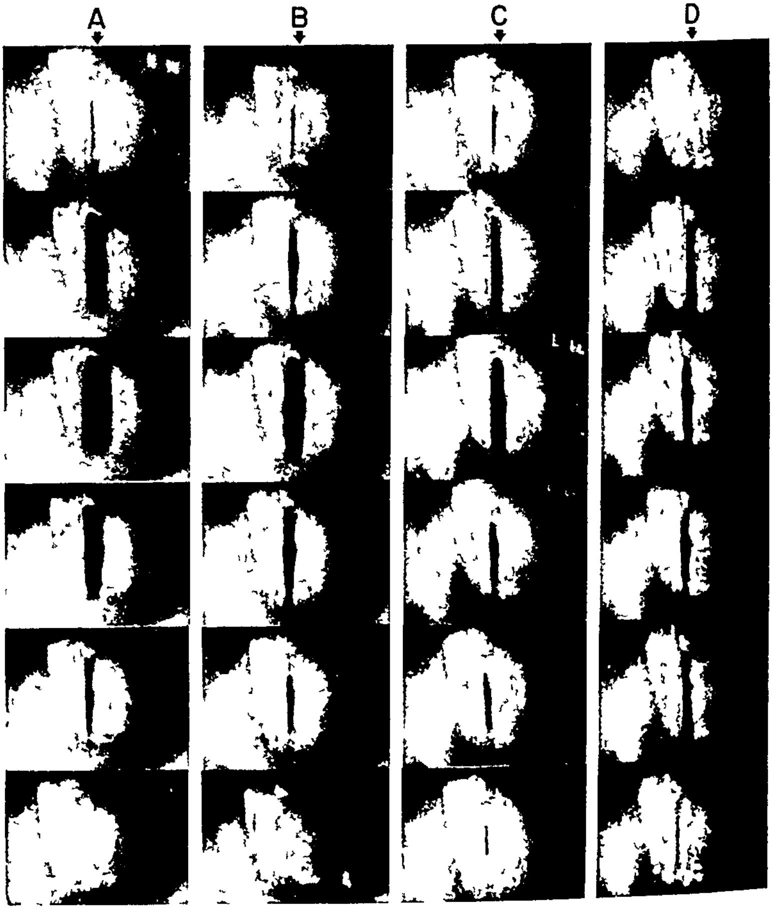

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SUMMARY

The falsetto refers to the upper part of the singing range situated above the point at which there occurs a change from relatively full and sonorous tones to those of characteristically lighter and thinner quality The various mechanisms by which this is accomplished are complex but essentially of such a nature that the thyroarytenoid muscles are refrained from vibrating in their entirety as they do in the fundamental register Three basic patterns of vibratory activity in the falsetto have been identified by high-speed photography The first is the open-chink, in which the cordal margins do not or only occasionally touch each other in vibration, the second is the closed-chink, in which they do make contact, and the third is damping, in which pitch is raised by progressive approximation of corresponding segments of the vocal cords usually from posterior to anterior The first two mechanisms are by far the most common and are accomplished with the vocal cords vibrating in their entire length just as in fundamental registration The distinguishing feature of the falsetto is decreased activity of the laterally situated mass of thyroarytenoid musculature In damping there is also an actual shortening of the vibrating elements of the cords

The degree of vocal training is of prime importance in determining both the acoustical qualities of the falsetto and its appearance in high-speed photography In making transition from the fundamental register to the falsetto in incompletely trained voices, high-speed photography reveals a period of muscular dyscoordination, the length and degree of which is directly related to the vocalist's skill In the advanced vocalist this so-called "break" is not heard nor is it seen in film sequences, selective contraction and relaxation of the thyroarytenoid muscles effecting a smooth transition from the lower register to the falsetto This selective control continues throughout the falsetto so that the entire vocal range may falsetto, it does little to solve the vexing problem of terminology Laryngologists and voice teachers lament the continued use of such terms as chest register, head resonance, head tones, etc , which have long ago been shown to be without physiologic basis These terms are bad enough from a scientific standpoint, but more than that, they do not have uniform esthetic connotation within the vocal profession itself For example, a soprano singing F below high C and, from an acoustical standpoint, utilizing the falsetto, will remark "I don't know what you mean by falsetto In continuing upward I simply use more head resonance " A second vocalist will then comment, "No, those high tones are obviously falsetto " This is typical of the varying interpretations of a nomenclature in general usage for several hundred years

It is possible to employ these terms, unscientific as they are, in the untrained voice because cleavages are clearly audible In the trained voice, however, it is another matter High-speed films in this study have repeatedly demonstrated that the entire vocal range may consist of only one register in the sense that all tones are produced by the same basic mechanism Controlled selective contraction and relaxation of the thyroarytenoid muscles will effect a graduated and smooth transition from 100 per cent utilization of the thyroarytenoids in the so-called lower register to a range of perhaps 10 to 60 per cent in falsetto Using superbly coordinated vocal musculature as raw material the expert vocalist then achieves coloring and further blending by skillful use of the supraglottic resonating chambers The result is an artistic effect which confounds all efforts to reduce the voice to terms which are descriptive of its primitive stage

Too many variables are at work for a single terminology to satisfy everyone at this time It would seem to be more important for greater knowledge and understanding of modern laryngeal physiology to filter through all levels of vocal pedagogy so that there can at least be a meeting of minds on factual ground If this can be accomplished, nomenclature may well take care of itself, for it will then evolve through a

PRIMARY ADENOCARCINOMA OF THE UVULA

Report of a Case

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Primary adenocarcinoma of the uvula is a rarely encountered tumor of the oral cavity. In a 50-year survey of the literature, Stout[1] found no cases of adenocarcinoma in his reported series of 28 tumors of the uvula. New and Childrey[2] reported 357 cases of malignant tumors of the tonsil and pharynx over a 13-year period, 1917-1930, but there was no mention of primary adenocarcinoma of the uvula in this series. In 41 cases of adenocarcinoma of the oral cavity, Watson[3] listed the soft palate as a primary site in three cases. Martin[4] reported 16 adenocarcinomas of the palate, but only four originated from the soft palate. The authors[3,4] did not mention the uvula as a primary site in these soft palate adenocarcinomas. Havens and Ye[5] discussed primary malignant tumors of the uvula, but all seven cases reported were squamous cell carcinoma. Tremble and Lockhart[6] state, "malignant tumors of the soft palate are relatively common, however, those with their primary site of origin in the uvula are rare."

The origin of adenocarcinoma of the uvula is the mucous glands. Glands of the mucous type predominate beneath stratified squamous epithelium; however, beneath pseudostratified ciliated columnar or respiratory epithelium, seromucinous glands predominate.[7] Much confusion has existed between the relation of adenocarcinoma of the true salivary glands and adenocarcinoma of the glands in the upper respiratory tract and oral mucous membrane. This confusion is due to the fact that these neoplasms with similar gland structure

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appear to be an extension of one basic mechanism Variations in vocal skill are associated with such wide differences in acoustical properties of the voice and physiologic behavior of the vocal cords, that it is impossible at this time to encompass vocal registration in a single descriptive terminology

The findings of this study suggest new areas for experimentation and speculation on the part of the laryngologist, vocal therapist, and voice teacher The behavior of the vocal mechanism during phonation in the falsetto points, for example, to the possibility of corrective vocal exercises utilizing the falsetto in a comfortable tessitura from piano to mezzoforte For the voice teacher there is evidence from this study that it is not only possible but also desirable to develop the falsetto in the normal singing voice, and that a smooth transition from the fundamental to the falsetto register is attainable with proper training

CONCLUSIONS

1 A study of the falsetto is reported, based on high-speed motion pictures taken by indirect laryngoscopy at 6,000-8,000 frames per second in vocalists of varying degrees of training and skill

2 The mechanism of the falsetto is reviewed and observations recorded and interpreted in the light of this photographic evidence

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may originate from either serous, mucous, or glands of the mixed type Sharnoff and Lisa[8] call attention to the differentiation that should be made regarding the cylindromas of salivary gland origin and the "true" adenocarcinomas that originate from mucous glands McDonald and Havens[9] classified 122 cases of adenocarcinoma of the nose, throat and mouth as distinct from cylindromas They graded these 122 tumors by the Broders method The authors[9] regarded Grade I and II adenocarcinoma as related to the mixed tumors

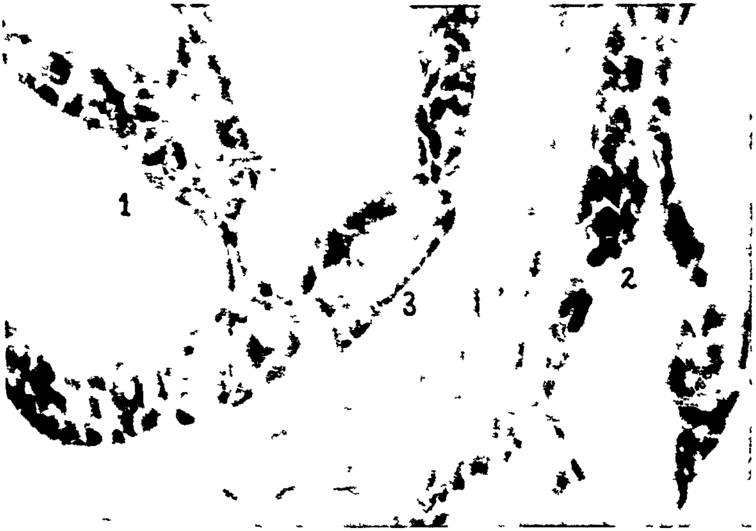

Fig [illegible] High power view showing mitosis at (1) hyperchromasia at (2) nucleoli at (3)

Forty-two adenocarcinomas of Grade III and IV in this group were considered to have an origin different from that of cylindromas and papillary adenocarcinoma of the salivary glands Mixed tumors that originate from the mucous glands and the salivary glands can be distinguished from these "true" adenocarcinomas despite their similar microscopic structure Mixed salivary gland tumors are composed of epithelial elements, spindle cells, cartilage and myxoid tissue, in addition to the atypical glandular structures present The acinar

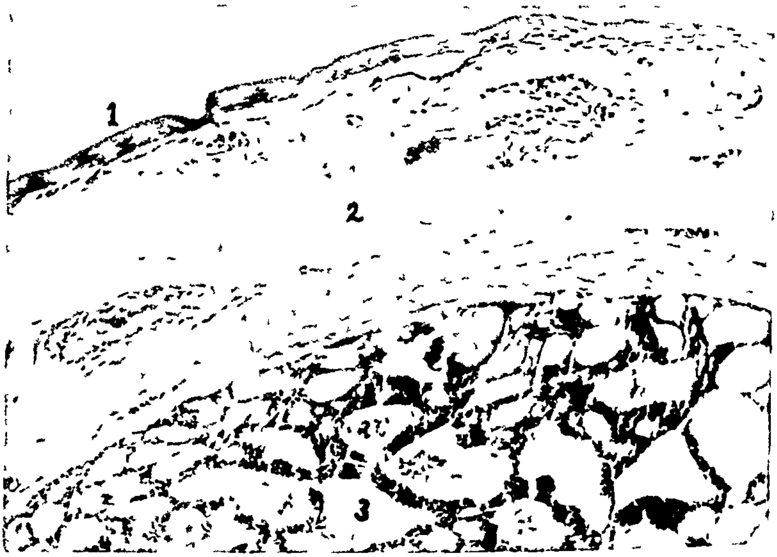

Fig 1 Low power view of the tumor showing (1) stratified squamous epithelium of the uvula (2) submucosal layer (3) adenocarcinoma showing atypical glandular structure

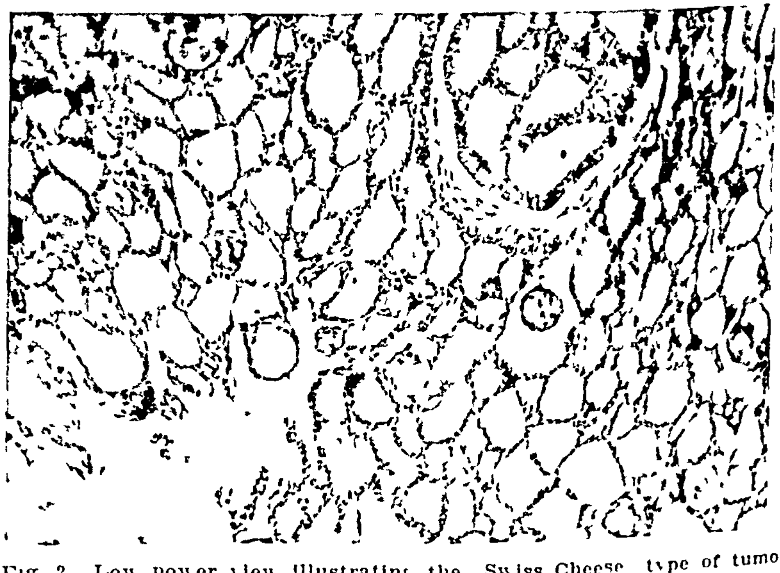

Fig 2 Low power view illustrating the Swiss Cheese type of tumor composed of atypical glands some of which contain colloid

rarity of these tumors is emphasized One additional case is presented

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RECOGNITION AWARDS

Hans von Leden, M D, and Paul Moore, Ph D, of Northwestern University recently received the Gold Medal of the Illinois State Medical Society and the Hektoen (Bronze) Award of the American Medical Association for original investigation These Awards were granted in recognition of their research on the mechanics of the crico-arytenoid joint spaces of the atypical glandular structures present in the "true" adenocarcinomas may not be seen in all mixed tumors

The following case is presented as an example of primary adenocarcinoma originating in the uvula Such tumors are infrequent, and only a few cases have been reported in the literature

REPORT OF A CASE

The patient, a 55 year old white male, had noticed a slowly growing "lump" on his uvula for four months No other symptoms were present Laboratory studies were within normal limits The patient was advised to have the tumor removed without further delay On May 3, 1956, the entire tumor was removed under general anesthesia without incident The patient was discharged the following day The tumor measured 1 5x1x1 cm on gross examination Pathology report by Dr Andrew A Eggston was as follows Microscopic sections reveal a surface of stratified squamous epithelium which has an ulcerated area, around which some lymphatic inflammatory reaction is present, some mucous glands surrounded by inflammatory reaction as well as trabeculae of collagenous fibrous tissue are also present The outstanding features are seromucinous glands that show marked cellular proliferation The cells form atypical glandular structures and show anisocytosis, hyperchromasia and mitosis The entire structure is that of cylindromatous or "Swiss Cheese" type of adenocarcinoma Diagnosis Adenocarcinoma of the Mixed Seromucinous Gland type

The above case is asymptomatic and free of disease over three years after surgical removal of the tumor

COMMENT

Primary malignant tumors in the uvula are rare Otolaryngologists performing tonsillectomies have an excellent opportunity for routine examination and palpation of the uvula Histological examination should be performed in all cases where the uvula may be removed for any reason The functions of the uvula were described by Richardson and Pullen[10] They considered the uvula of more importance in deglutition than in speech As illustrated by the above case, removal of the uvula apparently does not seriously impair the swallowing function

SUMMARY

A discussion of adenocarcinoma of the uvula is presented, together with a selected review of the literature The relative tween and drum or graft and footplate The prosthesis consisted of a polyethylene strut with a flared end In using this single strut I found it difficult to maintain its correct position The strut can easily slip out of position because of the difficulty in maintaining a tight connection between the bony footplate and the soft tissue of the graft Some of the oval window niches are quite shallow and the strut can slip out of position easier than with a deep niche The tight fit necessary to maintain the proper position between the graft and bony footplate makes extrusion of the strut through the graft a very likely possibility

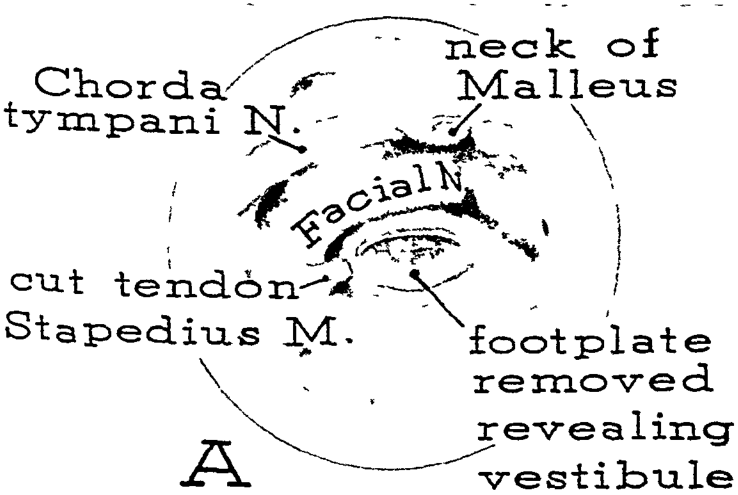

Fig 1

To overcome these difficulties, I have been using the following procedure with gratifying results in "Type IV" tympanoplasty technique

After completing all the necessary removal of infected tissue and diseased bone, the oval window niche and footplate are treated exactly as in performing a Shea[4] fenestration of the oval window The promontory is saucerized if necessary and the footplate of the stapes is removed, even if it is mobile

THE CROSSBAR (POLYETHYLENE) PROSTHESIS FOR THE IMPROVEMENT OF HEARING

Initial Report[*][†]

G DOUGLAS HAYDEN, M D ,

Richmond, Va

To accomplish the objective of a tympanoplasty procedure, various techniques have been described since 1950 Type IV tympanoplasties have consistently given the poorest results in hearing improvement Type IV tympanoplasty, as described by Wullstein,[1] is where erosion of the crura of the stapes has occurred, leaving only an intact mobile footplate The tympanic membrane or graft can not be brought into contact with the footplate for sound transformation to occur as in a Type III tympanoplasty in which a healthy mobile stapes is present

Wullstein's technique in Type IV tympanoplasty has been to leave the mobile footplate exposed directly to the sound waves while protecting the round window with a graft This procedure is based on the physiological principle that there must be a difference in the time and phase relationship by the two windows for stimulation of the cochlea Besides the technical difficulties in maintaining this difference between the two windows, the end result of improved hearing left much to be desired In trying to improve his results, Wullstein has reported the use of an oval ring made of "Polavit" fitting in the oval window niche between the mobile footplate and tympanic graft acting as an acoustic transmitter Apparently, these results were not too encouraging

Since then, Shambaugh[2] and Guilford[3] have reported improved results with the use of a polyethylene prosthesis be-

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[*] Presented at a meeting of the Tidewater Ophthalmological and Otolaryngological Society Portsmouth Va May 25 1960

[†] From the Department of Otology Rhinology and Laryngology Medical College of Virginia

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The No 50 polyethylene strut is not likely to extrude unless one makes it too high

The size of the crossbar strut can be varied When there is no bridge, as in radical cavities, the No 50 strut should be slightly shorter than in operations in which the bridge is preserved If the strut is too high it tends to cause atrophy and perforation of the drum and graft

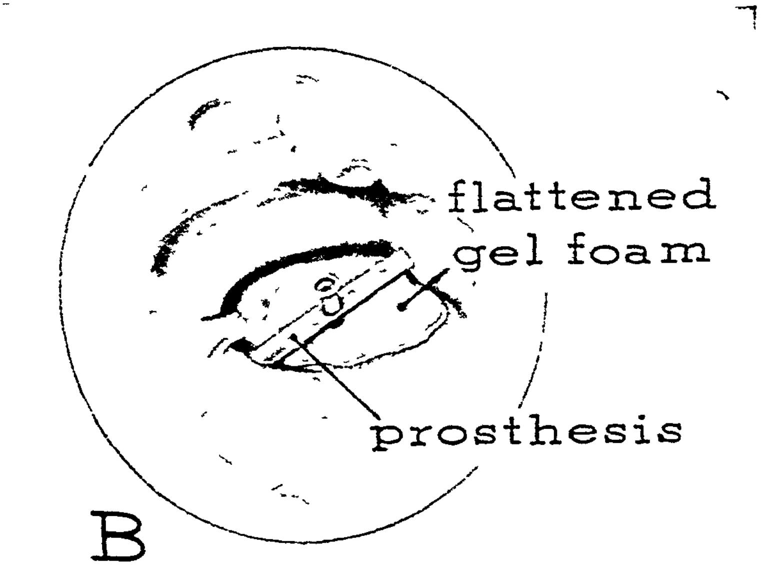

Fig 3

The same prosthesis has been used in fenestration of the semicircular horizontal canal failures with success The improved hearing in every case has been as good or better than a regular revision of the fenestrated canal could hope for

TECHNIQUE

A good-sized flap of skin is elevated in the fenestrated cavity and the stapes exposed A fenestration of the oval window procedure is then performed A flattened piece of (see Fig 1) A flattened piece of gelfoam (or a vein graft) is placed over the oval window A previously-prepared crossbar polyethylene prosthesis (see Fig 2) is then inserted between the oval window and graft The crossbar consists of a No 50 polyethylene strut with one end flared and the oval window end slightly tapered, going very loosely through a piece of No 205 polyethylene tubing (see Figs 3 and 4) The No 205 tubing acts as a stabilizing influence and keeps the acoustic transmitter (No 50) in place

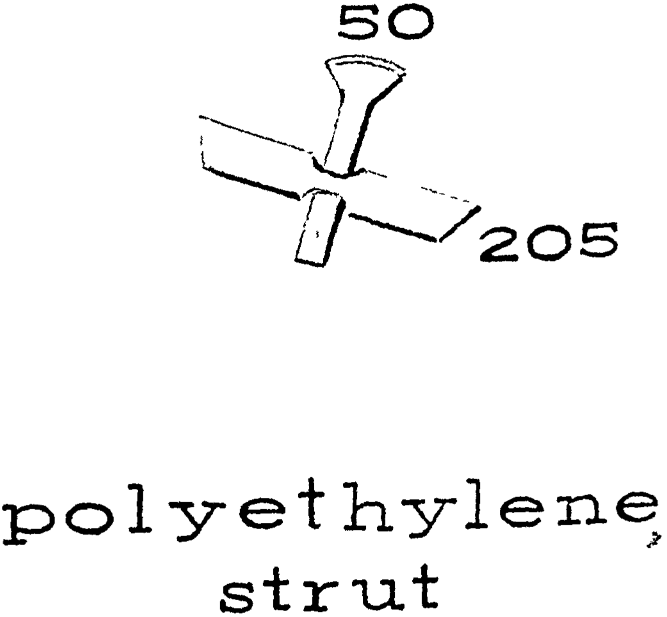

Fig 2

Removal of the footplate, even if it is mobile, makes for a much better maintenance of position of the prosthesis, and improved hearing results because of better motility instead of a more static condition as in the Wullstein oval ring of Polavit and who still have a good cochlear nerve response but who failed to regain or maintain useful hearing

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gelfoam is placed over the vestibule and a crossbar prosthesis is fitted into position between the tympanic membrane and the oval window opening, supported by the flattened gelfoam In an occasional case, one may find it necessary to use a small skin graft if the flap is too fragile to elevate properly making it impossible to close the middle ear without it I have had encouraging results with seven straight cases in the past six months using this method

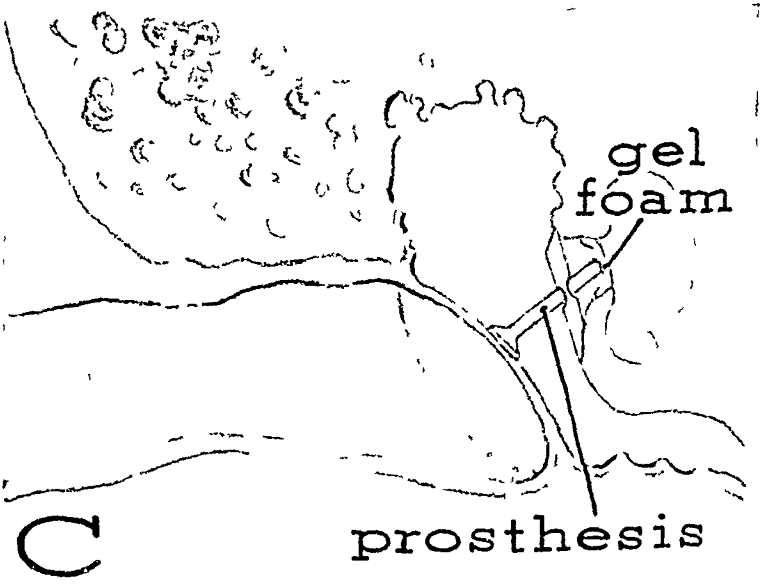

Fig 1

SUMMARY

A polyethylene prosthesis with a stabilizing bar can act as a good transmitter for the improvement of hearing in Type IV tympanoplasties The maintenance of its position and the improvement of hearing can be enhanced by removal of the footplate even when mobile

The same type of prosthesis has also proved successful in improving the hearing in certain patients who have previously undergone a fenestration of the semicircular horizontal canal

The present observation is extremely rare: it is the second case reported in the medical literature, the first case having been presented by Lawson Whale, laryngologist to the Hampstead General and the London Temperance Hospitals and was published in the *British Medical Journal*, 2 987, Dec 10, 1921

Studying the embryology of the thyroid gland one can realize the genesis of these cases The thyroid gland is formed very early in the human embryo (with 18 somites only), before the development of the neck In this phase of evolution the thyroglossal duct is related to the heart, arterial trunk and the sketch of the esophagus Not until the seventh week does the neck begin to assume its structure

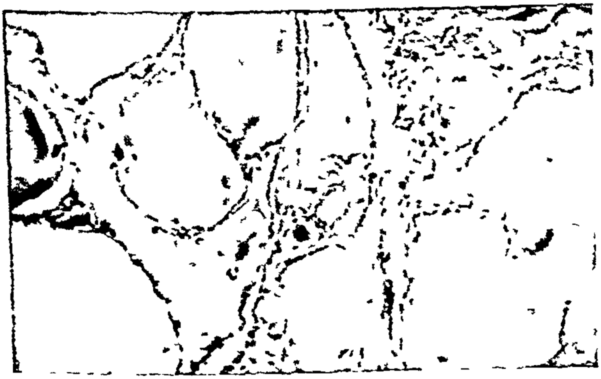

Fig 1 Photomicrograph showing the thyroid tissue found in esophagus (X100)

In some aspects our case resembles Whale's observation The tumor in both cases was seen as a benign lesion, like an esophageal papilloma I believe that frequently these thyroid nodules can be infected In Whale's case the infection process was serious The symptomatology presented by his patient suggested to him the possibility of esophagus perforation and infective mediastinitis because the patient told him that she had swallowed a fish-bone In my case the symptoms of infection were not so serious, but they were evident and were revealed by the odinophagy and the bleeding of the aberrant

ESOPHAGEAL NODULE OF THYROID TISSUE *†

GABRIEL PÔRTO, M.D., F.I.C.S.

Campinas, Brasil

The paper describes an extremely rare case and is only the second one published in the medical literature.

CASE REPORT

A 56 year old male peasant, living in São João do Itaguacu, Estado de São Paulo, Brasil, came for consultation, March 31, 1959.

The patient told me that one month prior to this visit he had felt a little pain on swallowing. Nose, mouth, pharynx and larynx were normal. The thyroid gland was not enlarged; in fact, it was difficult to locate by palpation. X-ray examination showed a normal esophagus.

Esophagoscopy was done on April 1 using Jackson's tube, 7 mm., and by this means a tumor was discovered attached to the midline of the cervical esophagus. The tumor was bleeding, and, after aspiration of the blood was clearly seen. It was about the size of a pea, moriform in contour, suggesting an esophageal papilloma. By means of a forceps it was easy to remove the small tumor entirely for biopsy.

The histopathologic examination was made by Dr. Monteiro Salles, and his report was: ectopic nodule of the thyroid gland.

Functional test of the thyroid gland by I^{131} (Isotopo Diagnosis) was made by Dr. Ubajara C. P. Correa, April 8, 1959. His conclusion was hypofunction of the thyroid gland according to the basal metabolism. Lugol solution, thyroid extract, mineral salts, vitamins, especially vitamin B_{12} were prescribed by Dr. José Estevan Teixeira de Camargo, April 8, 1959.

The patient returned to the office, June 11, 1959, having recovered from the esophageal symptoms. On the following day an esophagoscopy was done; no abnormality of the esophagus was observed.

COMMENTS

This case can be named an esophageal ectopic thyroid or an esophageal aberrant thyroid as both denominations mean the same condition. I prefer esophageal nodule of the thyroid, because thyroid tissue found in the esophagus obviously must be aberrant or ectopic.

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*Presented at the VIII Meeting of the Brazilian Society of Oto-rhino-laryngology and Bronchoesophagology in Porto Alegre, Sept. 1959.

†From the Department of Otolaryngology, Instituto Penido Burnier, Campinas, Brasil.

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thyroid These cases usually have no symptoms, they can be discovered only when the thyroid tissue is infected

In both cases reported in the literature the treatment was easily carried out and the small thyroid nodules were completely removed through the esophagoscope

SUMMARY

One case of esophageal nodule of thyroid is reported The tumor was found in the esophagus cervical and was completely removed through the esophagoscope This observation is extremely rare, it is the second case published in the medical world literature The first one was presented by Lawson Whale in the *British Medical Journal* (2 987, Dec 10, 1921)

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SARCOIDOSIS IN OTOLARYNGOLOGY

Report of Eleven Cases. Evaluation of Blind Biopsy as a Diagnostic Aid.[*][†][‡]

JACK ALLAN WEISS, M.D.,
Chicago, Ill.

The recognition of upper respiratory tract lesions of sarcoidosis presents a challenge, as the changes are frequently indeterminate in appearance, especially in their initial stages. An expanding interest and constant awareness on the part of the otolaryngologist is necessary to achieve an increasing accuracy of diagnosis. Together with lesions of the skin and eye, manifestations of the disease in the nose and throat are easily inspected and readily available for removal of tissue for microscopic examination. From a group of apparently unrelated conditions and syndromes, sarcoidosis has been established as a systemic disease with protean manifestations in various combinations. Though largely involving the reticuloendothelial system, it also affects almost every other structure and organ in the body.

It is the intent of the author to present a description of the general aspects of sarcoidosis, to review the reports of occurrence in the field of otolaryngology, and to add a group of observed cases. In addition, data will be submitted on a

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[*] Submitted as Candidate's Thesis to the American Laryngological, Rhinological and Otological Society, Inc., 1959.

[†] Awarded citation of "distinguished excellence" by American Laryngological, Rhinological and Otological Society, Inc., 1960.

[‡] From the Department of Otolaryngology of the Chicago Medical School and the Michael Reese Hospital.

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series of blind biopsies in established cases of sarcoidosis, with comment on the indications for and validity of this technique as a diagnostic procedure

Lindsay and Perlman have listed comprehensively the reasons for the concern of the otolaryngologist with this chronic granulomatous disease "*1* Frequency of lesions in the nose and throat as indicated by clinical and autopsy reports This may have etiological significance *2* Lesions in these locations are readily accessible for biopsy, also, they may precede the cutaneous lesion *3* Sarcoid lesions in the nose and throat may cause notable or severe symptoms *4* Cervical adenopathy or salivary gland enlargement may be directed to the laryngologist for his diagnosis and management *5* As one of the few chronic systemic diseases of undetermined etiology which affects the nose and throat it deserves study"[1]

It is hoped that the otolaryngologist will be stimulated to continued study of this bizarre disease, which is within his sphere of activity, and avail himself of the opportunity to detect a systemic condition by its local manifestations Because of the increasing prevalence of this pathologic process, it is important that he be familiar with it, as he may be requested to perform random biopsies to confirm a presumptive diagnosis of sarcoid lesions elsewhere

The nature of the disease has been succinctly stated by the Second Conference on Sarcoidosis held in Washington, D C, October 21, 1956, under the auspices of the Division of Medical Sciences of the National Academy of Sciences, Division of the National Research Council This expression represents the current consensus in the United States

"Sarcoidosis is a systemic disease, or group of diseases, of undetermined etiology and pathogenesis Histologically, it is marked by the presence of epithelioid-cell tubercles, showing little or no necrosis Varying types of inclusions in giant cells may be present, but are not pathognomonic A similar histological picture may be found in certain other diseases, especially in infectious granulomas and in beryllium poisoning Clinically, the disease most frequently involves lymph nodes, lungs, skin, eyes, liver, spleen and phalangeal bones The course is usually chronic and constitutional symptoms vary markedly More specific symptoms, when present, are related to the tissues and organs involved

"The intracutaneous tuberculin test is frequently negative, but a positive test does not controvert the diagnosis Hyperglobulinemia and leucopenia are common and hypercalcemia, hypercalcinuria, elevated phosphatase and eosinophilia are variable but sometimes significant features of sarcoidosis

"The diagnosis of sarcoidosis is based upon the above clinical features associated with a compatible histological picture, providing beryllium poisoning and infectious processes of known etiology can be excluded Spontaneous clinical recovery, with or without recognizable fibrosis, may result, or sarcoidosis may persist for years with varying functional alteration of the tissues or organs involved, or the disease may follow a progressive course, ending fatally "

ESSENTIAL CHARACTERISTICS OF SARCOIDOSIS

A detailed description of each aspect of systemic sarcoidosis is not germane A general discussion will be presented to understand the lesions in the otolaryngologic areas and evaluate their significance in regard to the total picture of the disease Longcope and Pearson characterize it as a "chronic, infectious granuloma, spreading slowly from one organ and tissue to another with few serious constitutional symptoms, and an erratic unpredictable course which may exacerbate, remain stationary or regress, heal by resolution or proceed to fibrosis with either minor residuals or severe symptoms depending on localization" The disease occurs at all ages but principally between the twentieth and fiftieth year The disease is more prevalent in females, there is a notable predisposition for the Negro race,[23] and there is a geographical predilection for the rural areas of Southeastern United States and the Scandinavian countries, which is inexplicable at present

The features of the process have been stated by various authors [2-8] Others have contributed comprehensive discussions of series of cases ranging from 34 to 300 in number [9 16] McGovern and Merritt systematically describe sarcoidosis in children [17] Carvalho's lengthy exposition has a bibliography of 1,544 references, dating from 1875 to 1952 [18]

Historically, sarcoidosis has developed through the correlation of various apparently unrelated signs and symptoms * Many names are associated with its evolution Hutchinson in 1875 gave the first clinical description,[101] while Boeck in 1899 first employed the term sarcoid and suggested its generalized nature [19] Good historical accounts are given by various authors,[6,9,14,20,21] Clinical syndromes, later to be consolidated with the systemic disease, include lupus pernio,[22] Besnier (1899) , osteitis tuberculosa multiplex cystica,[23] Jungling (1909) , uveoparotid fever,[24] Heerfordt (1909), and Mikulicz's disease[25] (1892) Schaumann is credited with associating the cutaneous and visceral lesions, thereby firmly establishing the systemic nature of sarcoidosis [26,27]

The etiology of sarcoidosis is an unresolved, long-continued controversy, especially its relationship to tuberculosis [14] The major concepts include

1 An independent disease, probably a granuloma of specific but unknown cause, either bacterial, viral, protozoal, etc [28,29]

2 Tuberculosis, in a modified or anergic form, with a hyperimmune reaction destroying the bacilli [3,22,30 31] (The noncaseating tuberculosis of Pinner [2])

3 A group of granulomatous diseases of known etiology fungal, tuberculoid leprosy or beryllium toxicity

4 A response to chemical intoxication, disordered lipid metabolism, or an exogenous or endogenous toxin [32]

Increasing current evidence indicates that tuberculosis is an unlikely cause Localization of sarcoid lesions has been variously reported as lung and lymph glands (peripheral, hilar and mediastinal), 90 to 100 per cent, skin and eyes, 20 to 25 per cent, bones 15 to 25 per cent, spleen about 20 per cent, liver about 12 per cent, and less than 5 per cent in the salivary glands, respiratory mucosa, heart, central nervous system, skeletal muscle, kidney and elsewhere

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Observers comment on the striking contrast between the degree of anatomical involvement of an organ, demonstrated by clinical and roentgenographic signs, and the mildness of the symptomatology[13] Local symptoms and signs may not be manifest until organ involvement is rather extensive Complaints in a group of 160 patients reported by Longcope and Freiman[14] occurred in the following number of cases cough 46, ocular 40, malaise 35, loss of weight 33, shortness of breath 30, skin lesions 29, enlarged lymph glands 21, abdominal pain 18, no complaints 16, expectoration 10, parotid swelling 8, night sweats 10, hoarseness 7, fever 7, swelling of hands and feet 6, chest pains 6, joint pains 6, and facial paralysis 3

Roentgenologic aspects of sarcoidosis are prominent The lungs are almost always involved, showing as a rule, a bilateral, diffuse localized or patchy interstitial infiltration, with miliary nodules, followed later by massive fibrosis Considerable enlargement of the hilar and mediastinal glands is frequent

Osseous lesions are quite common In combined statistics of X-ray manifestations from various sources, Holt and Owens ascertained an incidence of 15 per cent in 279 patients[33] Other reports range from 17 4 per cent to 25 7 per cent[13,16 34] Localization is principally in the small bones of the hands and feet Although foci have been reported in almost every bone in the body, involvement of the nasal and facial bones is quite rare Roentgenograms of the phalanges, metatarsals and metacarpals of the involved bones show round, radiolucent "punched-out" areas due to cystic destruction This appearance is almost pathognomonic The primary seat of attack is the medulla, with extension into the cancellous bone causing thinning and bulging of the cortex Invasion of the Haversian system results in a reticular pattern of destruction[14] authors [2-8] Others have contributed comprehensive discussions of series of cases ranging from 34 to 300 in number [9 16] McGovern and Merritt systematically describe sarcoidosis in children [17] Carvalho's lengthy exposition has a bibliography of 1,544 references, dating from 1875 to 1952 [18]

Historically, sarcoidosis has developed through the correlation of various apparently unrelated signs and symptoms Many names are associated with its evolution Hutchinson in 1875 gave the first clinical description,[101] while Boeck in 1899 first employed the term sarcoid and suggested its generalized nature [19] Good historical accounts are given by various authors,[6 9,14,20 21] Clinical syndromes, later to be consolidated with the systemic disease, include lupus pernio,[22] Besnier (1899) , osteitis tuberculosa multiplex cystica,[23] Jungling (1909) , uveoparotid fever,[24] Heerfordt (1909), and Mikulicz's disease[25] (1892) Schaumann is credited with associating the cutaneous and visceral lesions, thereby firmly establishing the systemic nature of sarcoidosis [26,27]

The etiology of sarcoidosis is an unresolved, long-continued controversy, especially its relationship to tuberculosis [11] The major concepts include

1 An independent disease, probably a granuloma of specific but unknown cause, either bacterial, viral, protozoal, etc [28,29]

2 Tuberculosis, in a modified or anergic form, with a hyperimmune reaction destroying the bacilli [3,22 30,31] (The noncaseating tuberculosis of Pinner [2])

3 A group of granulomatous diseases of known etiology fungal, tuberculoid leprosy or beryllium toxicity

4 A response to chemical intoxication, disordered lipid metabolism, or an exogenous or endogenous toxin [32]

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Laboratory data are not specific but provide some information. The hemogram is not diagnostic. Leukopenia or eosinophilia may occur. Total serum proteins are usually increased, with inversion of the albumin-globulin ratio. Serum calcium and alkaline phosphatase are increased in a few patients. The Kahn test is negative. Coccidioidin, histoplasmin, and blastomycin skin tests are negative, the tuberculin test is usually negative. Hyperglobulinemia occurs in about 25 per cent of cases. Hypercalcemia and hypercalcinuria may be present. Examination of sternal bone marrow yields no diagnostic information beyond the exclusion of the blood dyscrasias.

The gross pathology is represented by multiple, minute, pale, pinhead nodules which are discrete at first but may become confluent to form larger nodules or masses.

The histologic structure is the same in all body tissues. The basic unit is the hard (noncaseating) tubercle consisting of round focal collections of epithelioid histocytes. The cells have pale-staining vesicular nuclei, acidophilic cytoplasm and well-demarcated walls.[35] The nodule has a thin peripheral rim of lymphocytes. Multinucleated giant cells, of the foreign body or the Langhans type are conspicuous (see Fig. 1). These often show intercytoplasmic inclusions which are compatible with, but not pathognomonic of, sarcoidosis. They occur in other granulomas such as leprosy, leishmaniasis, brucellosis and berylliosis. The asteroid body of Wolbach[36] appears as a stellate shape with spiculated extensions (see Fig. 2). Schaumann's body is concentrically lamellated, double-contoured and deeply basophilic staining.[30] The centers of the nodules occasionally show a slight fibrinoid necrosis, but caseation does not occur. The process may resolve at times with disappearance of the cells. More often the lesions go on to a varying degree of fibrosis and hyalinization. Excessive scarring and replacement of normal structure accounts for functional impairment of the tissue or organ affected.[37-38]

James has cogently expressed the general attitude toward treatment in his comment: "It is difficult to dogmatize on the management of sarcoidosis, a disease whose cause is un-

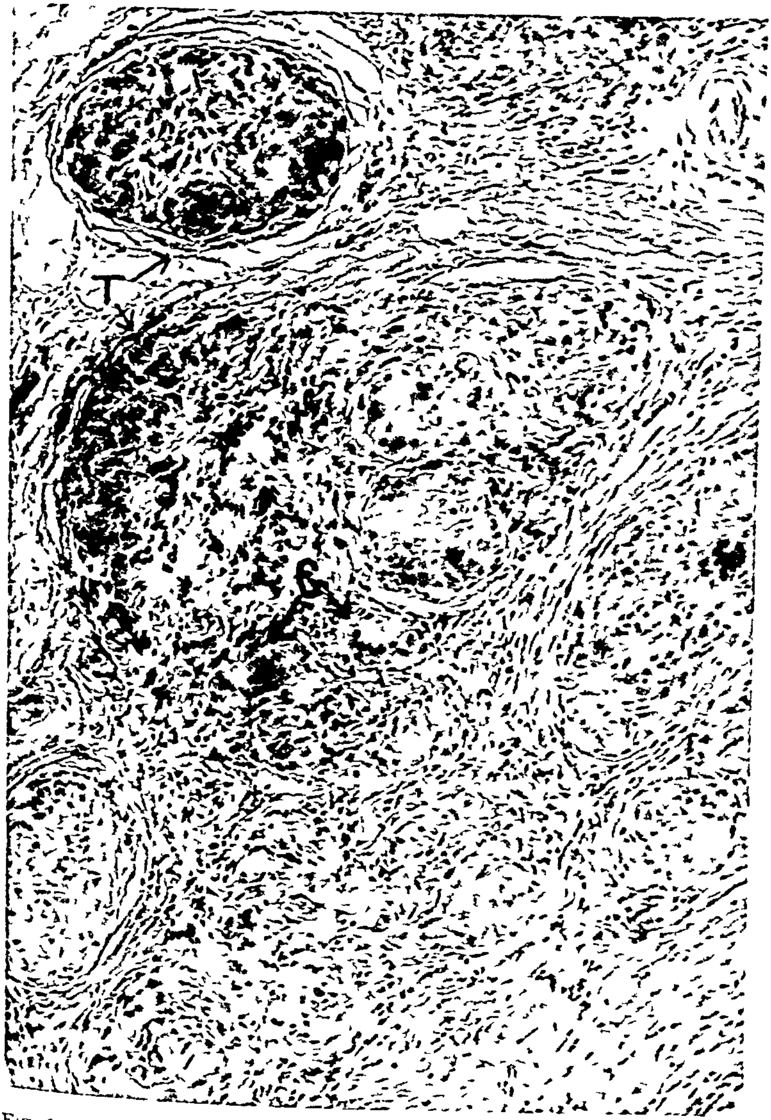

Fig 1 S S Case 2 Cervical lymph node (X100 H and E stain) showing almost total replacement of normal architecture by sarcoid tubercles (T) The lesion is moderately aged or regressed with an increase in fibrosis hyalinization and giant cells (G)

Fig 2 S S Case 2 Giant cell (X600 H and E stain) containing a stellate asteroid body of Wolbach (A) Intercytoplasmic inclusions are characteristic but not pathognomonic of sarcoidosis

determined, whose clinical manifestations are protean, whose natural history is unpredictable and whose specific therapy is unknown "[11]

Middleton[6] and others assert that treatment is empiric and hard to evaluate as progress of the disease is slow, and spontaneous remissions and resolutions occur In the past, arsenicals, iodides, tuberculin, irradiation, gold salts, chaulmoogra oil, Calciferol-D, promin, diasone, antibiotics, nitrogen mustard and other agents were tried with no success

Siltzbach assessed the effect of corticosteroid therapy in active lesions in 13 patients He found varying degrees of subjective and objective improvement The greatest value was evinced in the "suppression of acute organ-threatening and life-threatening effects of the disease," even though relapses followed cessation of treatment, and " decisive evidence that the disease is fundamentally affected by the drug is lacking", however, substantial information is afforded by his serial post-treatment biopsy studies, which exhibit definite regression in the histopathologic appearance of the lesions in contrast to pre-treatment specimens [39 40]

Citron also accepts these drugs as currently the most valuable, regretting the fact that short-term therapy does not produce permanent improvement and the required long duration is associated with known hazards [41]

Carr and Gage reviewed the world literature and found conflicting statements regarding prognosis [4] They traced 90 per cent of 109 sarcoid subjects in their own series and found 93 per cent alive after five years The prognosis is most favorable in those cases with a scarcity of symptoms and general good health Tuberculosis as a complication presents a special hazard with an incidence of 10 per cent as stated by Freiman[42] and 25 per cent according to Riley [9]

The concept of the benign course of sarcoidosis has undergone considerable revision Longcope and Freiman[14] gave a mortality rate of 14 per cent in 142 patients while Riley[9] and Reisner[13] each reported 25 per cent At present it is estimated that the outcome may be poor in 15 per cent to 20 per cent of the cases due to superimposed tuberculosis, pulmonary fibrosis with cor pulmonale, myocardial lesions with cardiac failure, central nervous system lesions, pituitary gland involvement and nephrocalcinosis, also, severe uveal tract fibrosis may cause blindness

MUCOUS MEMBRANE AND ASSOCIATED LESIONS

General Considerations

Lesions of sarcoidosis in the mucosa of the upper respiratory tract and the tracheobronchial tree are in the special province of the otolaryngologist The collected data from several large series of cases, including both clinical and autopsy observations, give a localization in the respiratory tract (including the salivary glands) in only 27 of 935 proved cases of sarcoidosis [9-14,16,43] This is an incidence of about 3 per cent It is very probable that the prevalent impression of a low incidence is erroneous, as one can readily infer from a perusal of various reports that examination of these areas is not done routinely This applies with equal emphasis to postmortem data, which rarely comment on the larynx or the nasal and oral cavities

On the other hand, Lindsay and Perlman[1] observed lesions in nine of 50 patients (18 per cent) This study is a noteworthy exception to the paucity of respiratory tract lesions as indicated above One may surmise that a plausible explanation would be the orientation of the observers, insofar as laryngologists obviously seek for and discover more occurrences in their own field

An even more striking contrast is afforded by an examination of the continental European literature Pautrier (France) discussed various mucosal lesions[44,45] and these were emphasized by Schaumann's notable early investigations from Sweden [26 27,30,46] In his monograph based on personal experience and an extensive review of the literature, Leitner (Switzerland) concluded that mucosal lesions are rather frequent and tonsillar involvement occurred in about one-half of the cases [47] Gravesen's monographic exposition in 1942 gave a 14 per cent incidence in 734 collected cases[34] Reyn found a 35 per cent incidence in 14 patients,[48] while Lomholt reported 60 per cent in 60 patients[49] The last three are from Denmark, Boeck's pioneer studies were in Norway It is interesting to speculate why sarcoidosis with mucosal lesions is more replete in the Scandinavian countries Is it due to more thorough study of the nose and throat or to a unique variant of the disease?

The importance of mucosal lesions in the respiratory tract primarily is due to the fact that they are easily accessible for biopsy This may be of particular value in establishing a diagnosis in obscure symptom complexes, furthermore, this importance is emphasized by the occasional circumstance of their concurrence as the only other lesion with a visceral involvement of the lungs, liver, or spleen

That mucosal lesions are of more than intrinsic interest is reflected in the attitude of several authoritative observers who have advanced the heuristic concept that the respiratory tract may be the portal of entry Ulrich[50] and Wille[51] speculated about the nose, Longcope and Freiman[14] suspected the lungs, Schaumann[46] implicated the tonsils, Gravesen, however, did not agree with this theory[34]

While sarcoidosis of the mucous membrane is usually associated with generalized lymphadenopathy the two manifestations are not related in pathogenesis Glandular enlargement is not necessarily in the drainage area of the lesions[14]

Specific Locations of Lesions

For clarity, mucosal lesions will be classified anatomically, although affected sites are frequently multiple in most patients While an accurate incidence cannot be established, from a review of the literature it appears that the most frequent locations are the nose and tonsils, the laryngeal components are secondary, and the remaining areas are occasional

Nose

The nasal tip is a rather frequent site for a cutaneous lesion, at times also involving the alae, columella or dorsum [2,53]

The process starts as multiple, elevated nodules which are 1 to 5 mm in diameter, discrete, yellowish, dry and scaling Increase in size by merging results in a conspicious large lobulated granuloma, bluish-red in color (later changing to brown) with a hyperemic areola, sharply differentiated from adjacent normal skin (see Fig 3) The very slow growing mass is soft, compressible, smooth and neither painful nor tender Bleeding or ulceration does not occur There is no continuity or sequential relationship between cutaneous and mucosal lesions of the nose, indicating the independence of involvement of these tissues

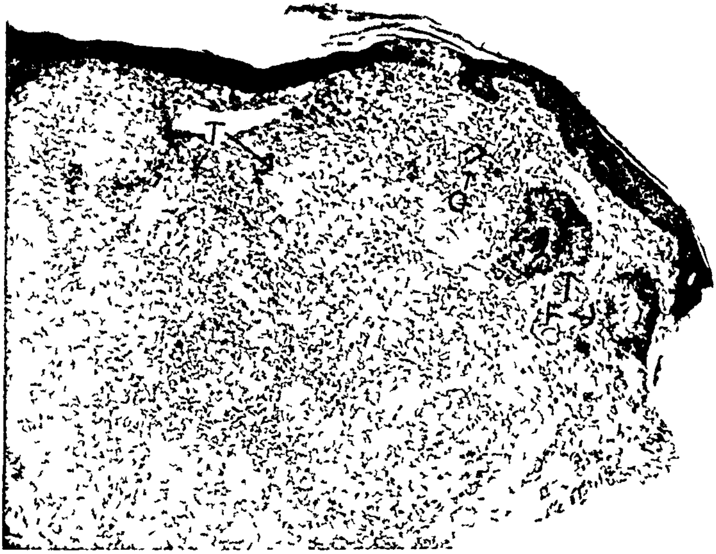

Fig 3 F T Case 1 Skin of nasal tip (X75 H and E stain) showing diffuse infiltration of dermis by typical hard (non-caseating) tubercles (T) Note hair follicles (F) and a multinucleated giant cell (G) (See Fig 4)

Nasal mucosal lesions are reported to occur mainly on the septum and inferior turbinate[16, 27, 46, 51] Boeck (1905) described chronic changes in the nasal mucosa in six of his nine cases, however, the only biopsy done failed to show the characteristic histopathology He suggested rhinologic studies[31] Ulrich's two cases were confirmed by biopsy[50] Klingmuller

(1907) made the early observation that mucosal lesions for a variable time may be not only the initial but also the sole apparent evidence of sarcoidosis[55]

Symptoms of nasal involvement may be slight or absent, but nasal discharge, discomfort and a variable degree of obstruction to respiration are common The changes may be limited to a catarrhal inflammation, lacking any distinctiveness[45] Microscopic examination may reveal the histologic structure of sarcoidosis, even without gross lesions or symptoms This is the rationale of "blind biopsy," to be discussed later

Lomholt[49] and Bainwater[76] identify the nasal lesions as follows

1 Disseminated tiny nodules, 1 mm to 4 mm in diameter, single or grouped, grayish-white, opaque, with a hyperemic border The nodules are firm but the mucosa is soft, bleeding easily on palpation They arise in the submucosa and are best observed after shrinking of the mucosa by a vasoconstrictor solution Later they gain the surface forming a yellowish wrinkled plaque with mucosal hypertrophy

2 Catarrhal rhinitis, with turgescence, serosanguinous or mucopurulent discharge, desquamation, erosions, crusting, and occasional ulceration

3 Hypertrophy and fibrosis occur in the end stage, with thickening of the septum and turbinates, and occasionally a polypoid mass, causing nasal blocking

Wille's concept embraces extension from the nasal mucosa to the lacrimal sac via the naso-lacrimal duct, with less frequent migration to the pharynx, oral cavity and larynx[71]

The pathologic process may eventually resolve or proceed to fibrosis or atrophic rhinitis The latter, with perforation of the cartilaginous septum, has been reported by Bainwater[illegible] and by Fletcher[57]

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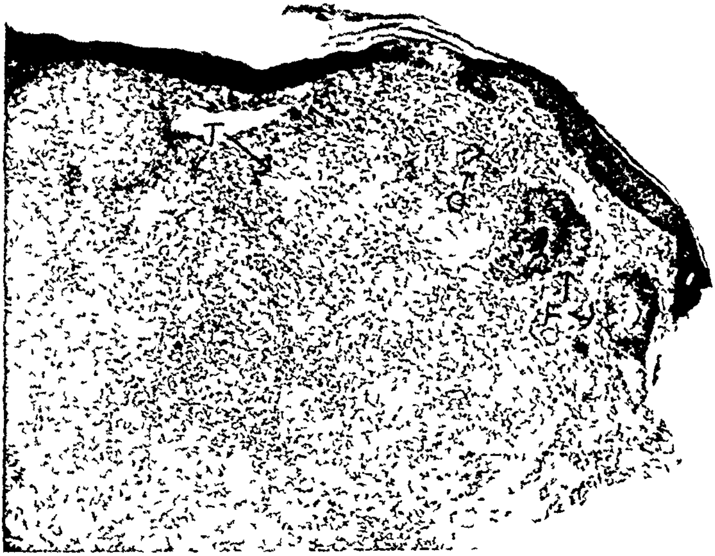

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Larynx

Laryngeal involvement is stated to be definitely less frequent than nasal, but Lindsay and Perlman described granulomas of the epiglottis in three of their nine cases and infiltration of the cords in another[1]

Bailey's patient had a pea-sized granuloma of the soft ventricle, causing hoarseness It was still present six years later Pulmonary lesions were then noted, though not found originally He concluded that laryngeal involvement in sarcoidosis may be primary without evidence elsewhere[55]

Orso was of the opinion that his case was limited to the larynx, as general examination was negative[59]

Schubert and Neuss described nodular masses, edema and infiltration of the epiglottis[60]

In 1940, Poe's patient, a woman aged 41, presented multiple sarcoid lesions of the skin, nose, septum, mandible and hard palate with nodular infiltration of the epiglottis, aryepiglottic folds, and trachea, sparing the vocal cords[52] In the succeeding ten years the laryngotracheal masses had increased to an obstructive size requiring several excisions of proliferative tissue via direct laryngoscopy and eventually necessitating thyrotomy and tracheotomy

Treble in 1958 reported a similar case in which a polypoid lesion of the cord was removed Subsequent enlargement of the lesion required tracheotomy on two occasions Under Prednisone treatment the laryngeal lesions disappeared but lesions elsewhere progressed[61]

Because of massive obstruction of the larynx in his patient, a permanent tracheotomy was done by Rosedale[62] Gravesen[34] and Ulrich[50] also recorded laryngeal lesions

Shea found a nodular tumor in the cartilaginous larynx, not involving the vocal cord, in a woman with hoarseness of four months' duration Removal was done via laryngofissure and a window in the thyroid cartilage Microscopic examination revealed sarcoidosis[63] cases[1] Similar lesions have been noted by other observers—James,[11] Weeden and Beckh,[10] Reisner,[13] and Fletcher[57]

Palatine Tonsils

Lesions in the tonsils are said to be second in frequency to those of the nasal mucosa, but several observers consider them the most common In his extensive investigations, Schaumann was impressed with the frequency of tonsillar involvement, reporting a positive biopsy in each one of his series of 21 cases[16] Clinical evidence was usually absent except for hypertrophy This observation of the regular existence of lesions in the tonsils was one indication to him of the systemic nature of sarcoidosis and constituted a valuable pathognomonic sign

Barnwater,[56] Lomholt,[49] and Pautrier[46] reported tonsillar lesions Leitner thought they occurred in 50 per cent of all cases The experience of Gravesen in finding them in 64 per cent of 70 patients, with or without gross changes, supports Schaumann's conviction Further verification of this position may be derived from Reisner's declaration that "the tonsils being a part of the lymphatic system, there occurs a natural predilection of sarcoid for these tissues" It was his belief that thorough investigation would probably reveal more cases of involvement of the mucosa of the upper respiratory tract despite the absence of clinical symptoms[13]

Most American observers report a much smaller incidence of tonsil involvement In nine cases Lindsay and Perlman report one tonsillar lesion and two pharyngeal lesions[1] Rubin and Pinner[43] list one tonsil lesion in 44 autopsies collected from the world literature up to 1944 Again, the reservation arises as to how often the tonsils were inspected Longcope and Freiman, in their authoritative monograph evaluating 160 cases, designate "pharyngeal involvement" as occasional, with no specific mention of the tonsils[11] James similarly fails to include tonsillar lesions in a series of 150 cases[11] It is apparent that the actual incidence must lie somewhere between the extremes of the 0 per cent of James (England) and the 64 per cent of Gravesen (Denmark)

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Further confirmation of this belief is provided by the experience of Citron In 70 cases of suspected sarcoidosis with pulmonary lesions shown by clinical and roentgenographic examination, he was able to aid the diagnosis in four patients by bronchoscopic biopsy[41]

Snider and Mackler examined 23 patients with proved sarcoidosis, all of whom had intrathoracic signs of the disease as well as diverse lesions elsewhere. Bronchoscopy showed no visible lesions in five cases, hyperemia of the mucosa in two, and mucosal thickening in 16 Random biopsy was not done in five, was negative in four and was positive in 14 cases In three of the positive group the mucosa was grossly normal It was significant that in three other cases the bronchus was the only site to yield a positive result They were convinced that granulomatous involvement of bronchial mucosa, readily accessible for biopsy via bronchoscopy, is more common than heretofore supposed and that a specific appearance is usually absent

Nasopharynx

Lesions in this area would probably arise from the lymphoid tissue of the adenoid and might be expected in association with involvement of the tonsils, as a component of Waldeyer's ring, however, such localization is rarely reported, or probably overlooked Lindsay and Perlman noted small granulations in the vault in three of their nine cases[1]

Larsonn's interesting experience at the Radiumhemmet in Stockholm is impressive In patients with generalized sarcoidosis, presumably undergoing irradiation treatment for cutaneous or other manifestations, he observed ten instances of nasopharyngeal lesions The incidence cannot be given as the number of cases observed was not stated By nasopharyngoscopy solitary, circumscribed, reddish-yellow, adenoid-like, lobulated nodules were observed, usually in the midline The larger masses resembled tumors and caused some discomfort and symptoms of nasal block and Eustachian tube obstruction

A summary of the data in several reports[11-13] discloses four laryngeal lesions in 345 cases, about 1.2 per cent. Despite this low incidence, laryngeal lesions are potentially hazardous as proliferative granulations may become obstructive.

Trachea and Bronchi

Although lesions in the trachea might be anticipated with pulmonary involvement, they are infrequently reported. Rubin and Pinner listed two instances in 44 autopsies.[13] In cases of laryngeal involvement, extension to the trachea appears to occur at times in the cases cited above.[52,60] Tracheal lesions were present in three of the nine cases in Lindsay and Perlman's collection.[1]

Longcope and Freiman point out that the smaller bronchi are always involved in pulmonary form of the disease.[11] Lesions of the major bronchi, infrequently reported in the past, have received more attention in recent years.[17,18]

Benedict and Castelman (1941) observed the larger bronchi to be reddened and edematous with bleb formation and partial stenosis. Biopsy revealed the characteristic microscopic structure. The only other manifestation was a supraclavicular mass of glands.[61]

Olsen's case in 1946 had pulmonary infiltration and paralysis of the IXth and Xth cranial nerves. There were no cutaneous lesions or obvious lymph glands available for biopsy. The diagnosis of sarcoidosis was made by microscopic examination of a smaller nodular lesion of the right main bronchus.[65]

Siltzbach and Sones record two cases with findings of partial stenosis of a main bronchus. Biopsies of the thickened mucosa surprisingly showed the typical granulomas.[66] They quote five other cases in which microscopic evidence of sarcoidosis was obtained by bronchoscopy, although a gross lesion was present in only *one* subject. These authors make the pertinent comment that the incidence *of sarcoidosis in the major bronchi is undetermined* as few cases have had endoscopic examination and fewer of these have had tissue re-

revealed the structure of a seromucinous, branched tubo-alveolar gland with sarcoid granulomatous infiltration [71]

Despite lesions on the septal mucosa, microscopic examination of cartilage removed by Poe during a submucous resection of the nasal septum failed to reveal any evidence of involvement [72]

Oral Cavity

Occasionally lesions occur in the soft palate and buccal mucosa Hard palate lesions have been seen as an infiltrative process [31, 73] Stomatitis has been noted [60] Lingual lesions are exceedingly rare Rubin and Pinner cited one instance of a tongue nodule discovered at autopsy [73] Leitner was able to collect only three cases [47]

Nasal Accessory Sinuses.

Lesions in the sinuses might be expected to follow those in the nasal mucosa The occurrence is rarely reported, perhaps because symptoms do not direct attention to the sinuses In some instances stated extension is presumptive, based on the combination of nasal lesions and roentgenographic changes

Lindsay and Perlman made a definite diagnosis of maxillary sinus involvement by a positive biopsy of polypoid tissue in the nose emerging from previously made naso-antral windows [1]

In Bordley and Proctor's case roentgenograms showed dense clouding of the left antrum with bone destruction in the left ethmoid and frontal Mucosa removed by ethmoidectomy showed sarcoid structure [70]

Because of bilateral maxillary sinus clouding on X-ray, Livingstone performed a bilateral Caldwell-Luc on a patient with sarcoidosis involving the eyes, lungs and peripheral lymph glands The hyperplastic mucosa removed revealed sarcoidosis [74]

Uveoparotid Fever and Mikulicz's Syndrome

Ocular lesions are mentioned as they are in an associated

Lateral roentgenograms demonstrated altered soft tissue contour The diagnosis was confirmed by biopsy Five of the patients also had nasal lesions, three had tonsillar lesions and three had swelling of the parotid glands[68] Again we note the marked disproportion in incidence of lesions compared to most of the American reports

Nasal and Facial Bones

As previously stated, these bones are rarely affected, however, Holt and Owens urge a detailed roentgen examination of nasal bones in patients with skin lesions of the nasal dorsum as the process may produce a painless, non-tender destruction of bone[33]

Rubin and Kling reported a case of an infiltrating tumor of the septum, which was thickened and studded with minute nodules The mass extended into the skin and caused erosion of the nasal bones, demonstrable on X-ray examination Biopsy of the tissue confirmed sarcoidosis The nasopharynx was not involved This case was considered unusual as the earliest involvement seems to have been limited to the nose[69]

Kistner and Robertson studied a case with a nodular lesion of the nasal tip The nose had gradually widened Biopsy of a nasal bone showed a proliferative sarcoid granuloma, invading the osseous trabeculae[53]

Bordley and Proctor encountered a destructive lesion involving the left middle turbinate and ethmoid complex Two successive intranasal anterior ethmoidectomies were done The second showed pus and new sclerotic bone which on microscopic examination revealed a granuloma "suggestive of sarcoid"[70]

In Poe's case, a roentgenogram of the mandible showed a circular area of translucency compatible with the destruction caused by sarcoidosis[52]

Kalman and Mallet recorded a most unusual case of sarcoidosis occurring in an aberrant mixed salivary gland rest in the maxilla Roentgenograms portrayed a cyst as a radiolucent area Microscopic examination of the excised tissue originating in the tympanic cavity and projecting into the canal in a patient with chronic otitis media. On biopsy this presented the microscopic structure of sarcoid.[81] Gravesen also alluded to tympanic cavity involvement in rare instances.[34]

Central Nervous System

The central nervous system is not spared in sarcoidosis. Invasion of the cerebrum, cerebellum and meninges and pars intermedia of the pituitary gland has occurred. Lesions in the brain stem may cause paralysis of the second, fifth, seventh, eighth, ninth, tenth or twelfth cranial nerves.[14,60,82]

Colover reviewed 115 collected cases of central nervous system involvement in sarcoidosis.[83] He noted facial paralysis in 58 cases, unilateral in 36 and bilateral in 22. Recovery was the rule, though partial residual impairment did occur. That involvement was central in origin and not due to local parotid infiltration was evidenced by the observations that it could occur in the absence of parotitis, that there was no direct relationship between the degree and duration of the parotid swelling and the functional impairment and that there was an associated loss of taste in ten of the cases. The next most frequently affected cranial nerve was the optic. Vagus and glossopharyngeal nerve involvement occurred in 29 patients. Laryngeal paralysis, usually unilateral, appeared in nine cases. The acoustic nerve was involved in 12 cases, with perceptive deafness in eight and vestibular branch disturbances in four.

REPORT OF CASES

The current series comprises 33 cases of sarcoidosis* in which the diagnosis has been established by a correlation of compatible clinical signs and symptoms, characteristic radiologic changes, positive microscopic evidence from one or more tissues and, usually, a positive Kveim cutaneous reaction. There were also compatible laboratory data in most cases, including negative skin tests for fungal diseases. To this group has been added three cases with previously diagnosed oto-area, which must be included because of the availability for biopsy. They may occur early and at times be the initial sign of the disease. James gives an incidence of 25 per cent in 200 cases[75] while Longcope and Freiman aver that it may range from 9 per cent to 64 per cent.[11] The most common manifestation is iridocyclitis, followed in frequency by keratitis, conjunctivitis and episcleritis.

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*Twenty-eight cases were from the Sarcoid Clinic (Dr Johann Bornstein, Chief) of the Chest Service (Dr David Radner, Director) of the Michael Reese Hospital. Five were from the Cook County Hospital, Chicago.

In 1909, Heerfordt contributed the classical description of the uveoparotid fever syndrome: *1* fever, *2* parotid enlargement, *3* uveal tract disease, and *4* facial paralysis, usually bilateral and transient.[6] Its etiology was later identified with sarcoidosis by the investigations of Bruins-Slot,[76] Pautrier,[11] and Longcope and Pierson.[73] In 1936, Folger analyzed 60 collected cases.[78]

Wilson[77] reviewed 31 reported cases of facial paralysis in uveoparotid fever and presented a successful decompression of the facial nerve in his patient.

Mikulicz described his syndrome in 1888 as a chronic, afebrile, painless, bilateral enlargement of the lacrimal glands and one or more salivary glands, customarily the parotids. There is an associated dryness of the conjunctivae and oral cavity.[25] An etiology of sarcoidosis has been ascribed to an estimated 15 to 20 per cent of these cases. Wheeden and Beeck[10] found Mikulicz's syndrome in one out of 34 cases, while Reisner's incidence was two in 35 cases.[13]

Pautrier found salivary gland involvement uncommon.[11] Parotid swelling without combined involvement was observed by Reisner in four of 35 cases[18] and by Riley in four of 52 cases.[9]

Ear

Involvement of the ear is usually a cutaneous condition. Boone and Coleman[79] and Poe[80] report essentially the same findings of pinhead or larger nodular infiltrations of the skin of the auricle with some enlargement and distortion but without involvement of the external auditory canal or the tympanic membrane, however, Ziegler described a hard polyp

6 (P B)	F	52	N	Peripheral glands – *Cutaneous –* Liver – Central nervous system§	Epiglottis§ *Parotid, bilateral§*	* (Also, conjunctiva –, gastrocnemius muscle –, scalene fat pad –)	–	–
7 (E H)	F	30	N	Lungs§ Liver* Mediastinal glands§ Peripheral glands –	Bronchus* Nasal mucosa*	*	*	–
8 (V T)	F	28	N	Cutaneous§ Lungs§ Hilar glands§ Phalanx§ Conjunctiva*	Nasal mucosa* Soft palate§ Hypopharynx§	§	§	§
9 (R S)	F	36	N	Peripheral glands§ Cutaneous† Hilar glands§	Soft palate§ Larynx† Septum† Ethmoid* Lacrimal glands§ Nasal bone*	§	§	§
10 (R J)	F	35	N		Nasal tip† Tongue*	§	§	§
11 (E A)	M	22	N	Pulmonary§ Hilar adenopathy§	Bronchus*	§	§	§

Code for Biopsies * positive, – negative § not done, † indeterminate granuloma

TABLE I

Respiratory Tract Lesions in 11 Cases of Sarcoidosis

Case	Sex	Age	Race	General Lesions	Respiratory Tract Lesions	Kveim Test	Blind Biopsy	
							Turbinate	Tonsil
1 (F T)	M	37	N	Cutaneous nasal tip* Peripheral glands§ Lungs§	Nasal septum§ Epiglottis§ Hard palate* Nasopharynx§ Atrophic rhinitis§	*	–	–
2 (S S)	F	15	W	Peripheral glands* Spleen§ Liver§ Lungs, hilar glands§ Mediastinal glands§	Tongue§ Bronchus*	*	§	§
3 (T W)	F	22	N	Cutaneous* Liver§	Uveoparotid syndrome§ Nasal alae§ Facial paresis, unilateral§	§	–	*
4 (I L)	M	45	N	Lungs* Peripheral glands† Liver†	Soft palate*	*	–	–
5 (S W)	F	50	N	Lungs§ Peripheral glands*	Bronchus*	– 1956 * 1958	–	§

lows: peripheral lymphadenopathy of the cervical, axillary and epitrochlear glands, lungs clear on auscultation, spleen and liver not palpable. The tonsils were not remarkable. The posterior pharyngeal wall was covered with a tenacious, greenish mucopurulent secretion. The nasal tip exhibited a soft, smooth lesion about 2.5 cm in diameter. It was bluish red in color with a hyperemic areola, well differentiated from the normal adjacent skin (see Fig. 4). The lesion was painless, nontender, compressible and slightly elevated. The nasal mucosa appeared red, infiltrated and covered with viscid secretion and odorous crusts, which left a bleeding surface when removed. The middle and inferior turbinates were small. The appearance resembled an atrophic rhinitis. A small perforation was present in the cartilaginous septum. In the vault of the nasopharynx could be seen a granuloma about 4 mm in diameter. The hard palate contained a granular infiltration about 1.5 cm in diameter. The epiglottis was studded with many whitish miliary nodules. Its rim was infiltrated and showed a "turban like" thickening similar to that seen in tuberculosis. The left arytenoid was edematous and the left vocal cord hyperemic, but there was no impairment of mobility. The eyes showed no gross changes. The parotid, submaxillary and lacrimal glands were not enlarged.

Prednisone therapy was started at 30 mg. daily and reduced to 20 mg. daily after three months. In October, 1958, he was admitted to the hospital because of an acute pulmonary infection superimposed on the sarcoidosis and chronic diffuse pulmonary emphysema. This responded to chloromycetin, humidification and antispasmodic aerosol therapy.

Laboratory Data: Urinalysis revealed a moderate albuminuria with 20 white blood cells per field. (Intravenous pyelogram was normal.) Serum calcium 8.6 mg. per cent, serum phosphorus, 3.9 mg. per cent; serum glucose 91 mg. per cent, serum urea, 10 mg. per cent, serum creatinine, 1 mg. per cent, serum potassium, 5.3 mg. per cent. Liver profile tests were normal. Serum total protein, 8 gm. per cent, serum albumin 2.1 gm. per cent, serum globulin, 5.9 gm. per cent (inversion of albumin-globulin ratio). The hemogram was not noteworthy except for a 14 per cent monocytosis. The Wasserman reaction was negative, the Kahn 2+. The Kveim reaction was positive. Blind biopsies of the tonsil and inferior turbinate were negative.

On last examination in August, 1959, the palate lesion had regressed and there was less nasal crusting. Little change could be noted in the appearance of larynx except for the absence of the hyperemia of the left cord. X-rays of the lungs were as before. He was still on Prednisone 20 mg. daily and stated that he had gained weight, felt good, had less cough and expectoration and was working regularly.

Comment: The nasal tip is a frequent location for an infiltrating plaque lesion of the Boeck's sarcoid variety of cutaneous sarcoidosis. The "atrophic rhinitis" degeneration of the nasal mucosa is somewhat common, the septal perforation less so. The multiple mucosal lesions in the nose, hard palate, nasopharynx and larynx evinced some response to corticosteroid therapy.

Case 2: S. S., a white female, aged 15 years was first seen in 1954 with a 'lump' in the tongue of about two months' duration. She complained of cough and some 'annoyance" on swallowing. Examination revealed a non tender nodule 1 cm. in diameter, in the mid portion of the tongue laryngologic lesions* (Cases 8, 9, 10), making a total of 36 cases

Thirty-two of the patients were Negro 28 females and four males, while four were white three females and one male The disproportionate high incidence in the Negro race and in the female sex is in accord with statistical data in other larger series

In the 33 unselected cases lesions in the nose and throat and associated areas (salivary glands and tracheobronchial tract) were found in eight patients, an incidence of 24 per cent (see Table I)

Conceding that statistical data in a very small series are not necessarily accurate, nevertheless it may be considered definitely indicative Our clinical experience fulfills the prediction of some observers that comprehensive routine examination of the respiratory tract in sarcoidosis would disclose a greater number of lesions than was formerly expected This includes gross lesions as well as unsuspected involvement revealed by blind biopsy This observation receives further corroboration from the comprehensive bronchoscopic studies of Snyder and Mackler as summarized above[67]

Case 1 F T , a Negro male, aged 37 years, presented symptoms of nasal crusting, partial obstruction of breathing, a moderate non productive cough and hoarseness of two years' duration A purplish swelling of the tip of the nose had started seven years ago as a "pimple" and had progressed slowly There was no bleeding, ulceration, pain, or tenderness No other cutaneous lesions were seen He had received intramuscular injections of streptomycin twice daily for ten weeks as treatment for tuberculosis

In March, 1958, he was admitted to the Chicago Municipal Tuberculosis Sanitorium with this presumptive diagnosis Roentgenograms of the chest revealed irregular mottled infiltrations of both upper lung fields, with radiolucent areas in the apices The Mantoux test was negative in 1 100 dilution Numerous sputum stains and cultures, and gastric washing cultures were negative for tubercle bacilli Fungal skin tests (histoplasmin, coccidioidin and blastomycin) were negative X rays of the hands and feet did not show any osseous changes The diagnosis of sarcoidosis was made by a microscopic study of the nasal tip lesion (see Fig 3)

In April, 1958, he was admitted to Mandel Clinic of the Michael Reese Hospital for further management The essential findings were as fol-

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*From the University of Illinois Research and Educational Hospital (by courtesy of Dr Francis L Lederer) and the Illinois Eye and Ear Infirmary (by courtesy of Dr Frederick Pollock)

The presumptive diagnosis was benign tumor possibly fibroma or rhabdomyoma

She was admitted to the Sarah Morris Children's Hospital of the Michael Reese Hospital On preoperative examination the spleen was found to be 4 cm below the costal margin and the liver 1 cm below The parotid, lacrimal and submaxillary glands were not enlarged A moderate lymphadenopathy was present, involving the cervical, inguinal and axillary nodes X-rays of the chest showed massive mediastinal and hilar lymphadenopathy with diffuse interstitial infiltrations of the lungs Because of polyuria, enuresis and urinary findings of cylinduria with a trace of albumin, an intravenous pyelogram was done This exhibited an enlarged left kidney with a filling defect in the calyx Laboratory data was non contributory, including sternal marrow puncture

Material for a Kveim test was not available Biopsy of a cervical node showed a microscopic structure consistent with sarcoidosis Ziehl-Neelsen stain was negative for acid fast bacilli and the periodic acid Schiff stain did not reveal any fungi (see Fig 1) Bronchoscopy revealed diffuse granularity of the mucosa of the major bronchi A biopsy was positive for Boeck's sarcoid (see Fig 5) In 1958 a positive Kveim test was obtained

On last examination in July, 1959 the only notable finding was a hyperemic nasal mucosa which bled easily on palpation with a probe The patient had no disturbing constitutional symptoms, and was doing well on corticosteroid therapy The tongue nodule had largely receded, however, it has varied in size in relation to the intervals of omission of therapy

Comment In this established case of generalized sarcoidosis, with lymph gland, pulmonary, liver and spleen involvement, the tongue nodule in all probability is a sarcoid lesion The renal pathology may also be suspected to be due to sarcoidosis

Case 3 T W Negro female, aged 22 years came to the Cook County Hospital in March 1958, because of pain and progressive decrease in vision in the right eye Two years previously she had had a febrile illness of one month's duration with bilateral facial swelling followed by left facial paralysis A similar episode occurred in February, 1958 Essential findings were as follows cutaneous lesions, plaques and subcutaneous nodules over the extremities and trunk multiple yellowish nodules 1 to 4 mm in diameter, over the cheeks, lips and nasal alar margins The right eye showed a granulomatous uveitis with a posterior synechia fibrosis of the lens capsule and a secondary glaucoma Corticosteroid therapy was started The left parotid gland was enlarged and indurated The left side of the face showed a residual paresis The liver was 2 cm below the costal margin the spleen was not palpable

The nasal mucosa contained no definite lesions except a spider-web capillary dilatation The tonsils pharynx, larynx and nasopharynx showed no noteworthy changes Biopsy of the skin revealed sarcoidosis Biopsy of the right inferior turbinate was negative, but tissue taken from a tonsil exhibited sarcoid histopathology

Comment This case exhibits a cutaneous sarcoidosis and a recurrent uveoparotid fever syndrome It also illustrates the

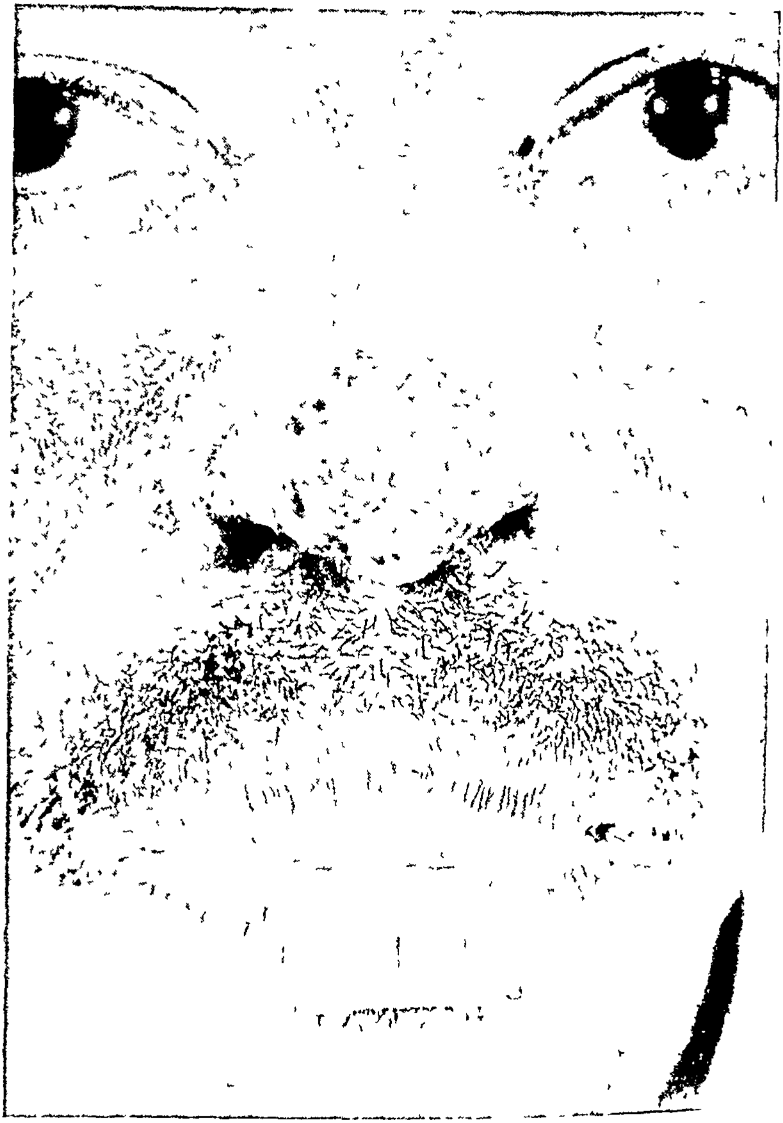

Fig 4 F T Case 1 Nasal tip manifestation of sarcoidosis The lesion is an erythematous infiltrating plaque soft compressible and slightly elevated The color is bluish-red with a hyperemic areola well differentiated from the adjacent skin (See Fig 3)

finding of microscopic sarcoidosis in the tonsils on blind biopsy in the absence of a recognizable gross lesion

Case 4 J L, a Negro male, aged 45 years, had been treated for tuberculosis for six months at a municipal sanitorium on a regimen of streptomycin, ioniazid and para aminosalicylic acid because of symptoms of loss of weight, fatigue, and dyspnea on exertion X-rays of the chest showed a diffuse, patchy, infiltration of the upper two thirds of each lung There was no cough or excessive sputum Tubercle bacilli could not be demonstrated Scalene node biopsy in January, 1957, was inconclusive Liver aspiration biopsy in April 1957 revealed a granulomatous lesion suggestive of sarcoid Intercostal biopsy of the lung in November 1957 showed a microscopic structure consistent with sarcoid

He was referred to Mandel Clinic for management At this time there were no nasal symptoms or swelling of the salivary glands X-rays of the chest showed no change from above, and the lungs were clear to auscultation The tonsils were small The pharynx mucosa was hyperemic and granular The nasal turbinates were enlarged and covered with a thin hyperkeratotic layer The septal mucosa was covered with crusts which left a superficial erosion on removal The soft palate exhibited a patch of discrete yellowish nodules from 1 to 3 mm in diameter The larynx and nasopharynx were clear Since December, 1957 he has been on Prednisone therapy with a dosage varying from 25 to 10 mg daily with occasional periods of cessation The palate lesion has regressed partially Despite the presence of a cough, productive of a thick yellow sputum, he is subjectively improved The Kveim test was positive in March, 1959, but biopsies of the tonsil and turbinate were negative

Comment This case presents sarcoidosis of the lung, liver and mucosa of the soft palate The haphazard localization of the disease in the respiratory mucosa is shown by absence of microscopic evidence of involvement of the nasal mucosa, despite the gross changes of crusting with erosion It also illustrates the necessity of making an early diagnosis of sarcoidosis by any available means to avoid unnecessary intensive antibiotic and chemotherapy

Case 5 S W, a Negro female aged 50 years attended Mandel Clinic in 1955 with a history of fatigue loss of weight, shortness of breath wheezing and cough productive of a colorless sputum She also complained of occasional epistaxis and some obstruction to nasal breathing Chest X ray showed widespread bilateral confluent linear infiltration Tuberculin skin tests were negative and tubercle bacilli could not be found The Kahn reaction was negative Hands and feet showed no osseous roentgenologic change There was a reversed albumin globulin ratio with albumin 2 4 gm per cent and globulin 5 8 gm per cent

Biopsy of a cervical node and needle aspiration of the liver confirmed the diagnosis of sarcoidosis Bronchoscopy in April 1955 revealed a thickening of the mucosa of the left and right main bronchi and a positive biopsy was obtained The Kveim reaction was negative in August 1956 but positive in December, 1958

On Prednisone therapy 20 to 30 mg daily between May 1955 and

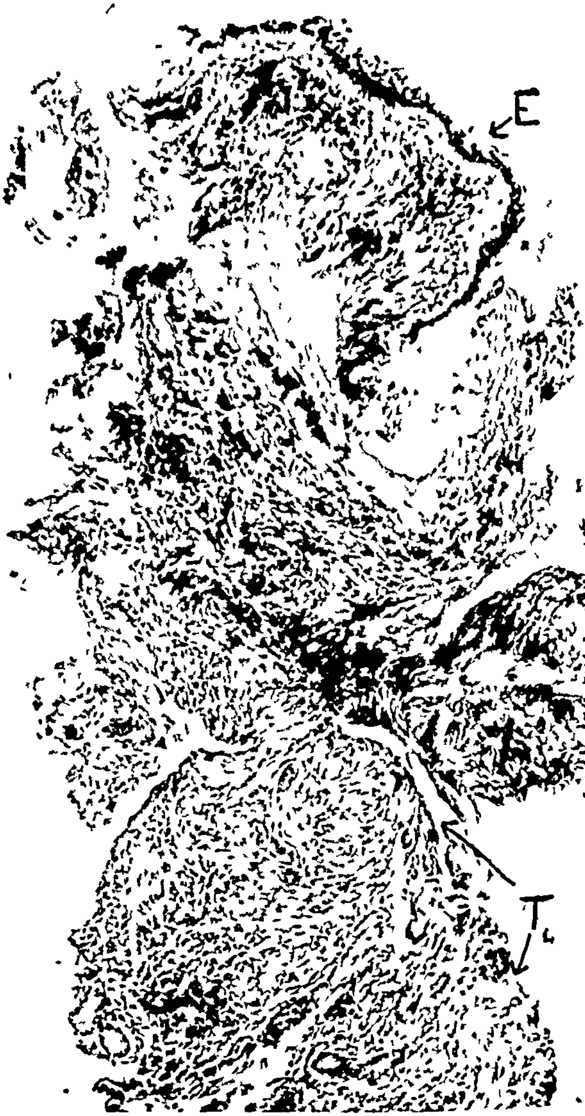

Fig 5 S S Case 2 Bronchial mucosa (X75 H and E stain) showing tubercles (T) in the submucosa Epithelium is at E

the lesions, the recurrent parotitis and the positive Kveim test, make the diagnosis most probable

Biopsy of the epiglottis and parotid is contemplated

Case 7 E H a Negro female aged 30 years had received a long course of antituberculous therapy for ten months, starting in July, 1954, because of fatigability, cough, night sweats and loss of weight Chest X rays showed a bilateral mediastinal and hilar lymphadenopathy with diffuse linear and nodular pulmonary infiltration Microscopic examination of a supraclavicular node showed "caseating tuberculosis" The old tuberculin skin test was 2 plus in 1 1,000 dilution Some clinical improvement with regression of the nodes followed treatment In December 1954, the nasal mucosa was observed to contain multiple pinhead nodules and areas of infiltration over the septum and both inferior turbinates Biopsy revealed a "nonspecific granuloma" In February 1955 liver biopsy was reported as "granuloma, either tuberculosis or sarcoid" Biopsy of the bronchial mucosa was stated to show a tuberculous granulation, but the Ziehl-Neelsen stain was negative for tubercle bacilli Subsequent biopsies of bronchial mucosa and liver showed a granuloma consistent with sarcoidosis Continued intensive studies were negative for tubercle bacilli, except one bronchial lavage which yielded "a few acid fast bacilli" A Kveim test was positive for sarcoidosis A sternal puncture revealed a non specific marrow pattern During 1955 she was continued on a reduced antituberculous regime, but the response was not characteristic as a new infiltration appeared in the right upper lobe on X ray

On examination in 1958, X-rays of the chest were normal in appearance, and patient was generally well Biopsy of the tonsil was negative in July 1959 Despite the absence of gross changes in the nasal mucosa biopsy of the inferior turbinate was positive

Comment This case presents an arrested tuberculosis (cervical lymphadenitis and pulmonary) and a generalized sarcoidosis It shows the not infrequent clinical, albeit not etiological, relationship between the two diseases

Case 8 V T, a Negro female aged 28 years, came to the Illinois Eye and Ear Infirmary in 1953 with a complaint of nasal discharge, occasional epistaxis, swelling of the lids of the right eye and "lumps" in the skin Examination revealed thickening of the right upper eyelid with follicular hypertrophy of the tarsal conjunctiva There was no involvement of other ocular structures The cervical glands were moderately enlarged Subcutaneous nodules about 1 cm in diameter were present over the extremities and trunk The nares were crusted and excoriated The nasal mucosa was hyperemic, bleeding easily on touch Small discrete submucosal white nodules were located in the right inferior turbinate The uvula soft palate and anterior wall of the hypopharynx were the site of superficially eroded infiltrations The larynx and the tonsils appeared grossly uninvolved The lungs were clear to auscultation but X rays showed bilateral increased hilar densities X rays of the hands and feet showed a well defined radio translucent area in a phalanx Biopsies of the tarsus and the inferior turbinate revealed a microscopic structure consistent with sarcoid

Comment The circumscribed area of bone destruction in a

June, 1956, the patient gained weight and felt generally well This has been continued to date with occasional intermissions Examination in March, 1959, revealed a peripheral lymphadenopathy The liver was palpable The tonsils were large, but otherwise not remarkable The oral cavity, nasopharynx, and larynx showed no change The turbinates were pale, boggy and covered by a thin adherent filmy membrane Biopsy of the right inferior turbinate was negative

Comment The bronchoscopic biopsy was positive early in the course of this case The Kveim test was negative in 1956 despite preceding positive biopsies of the liver, bronchus and scalene node

Case 6 P B , a Negro female, aged 52 years, had been under observation since 1950 at Mandel Clinic for a multiplicity of symptoms fatigue, repeated epistaxis, recurrent parotid swelling, and neurological signs including intention tremor, hypoesthesia of the right side of the body, absent knee jerks, cerebellar ataxia, and decreased deep tendon reflexes, etc The essential findings in repeated admissions to Michael Reese Hospital were as follows

March, 1950 Recurrent bilateral parotitis Right serous otitis media with marked polypoid swelling of tissue around Eustachian orifice as visualized by nasopharyngoscopy Palpable spleen and liver Chest X ray showed right hilar density Boeck's sarcoidosis was suspected and corticosteroid therapy instituted which reduced the parotid swelling

April, 1951 Febrile episode with parotid swelling, axillary, cervical and inguinal lymphadenopathy, and pigmentation of the skin

November, 1953 Episode of pneumonia Bilateral parotid swelling

December, 1954 Submaxillary glands swollen and tender No ocular pathology

January, 1955 Right parotid swelling with fever Repeated epistaxis Hemogram shows leukopenia, thrombocytopenia, a relative monocytosis and a normochromic, normocytic anemia Hyperglobulinemia present Fungal and tuberculin skin tests were negative

Examination in March, 1959, was as follows Nose mucosa is hyperemic with dilatation of vessels which bleed easily on palpation The turbinates are turgescent and lightly crusted The tonsils are absent The oral cavity and nasopharynx show no changes The larynx is normal except for a number of pinhead white nodules on the rim of the epiglottis This is a presumptive sarcoid lesion The parotid glands are enlarged but not tender

During this elapsed period biopsies were negative from axillary and cervical nodes, skin, scalene fat pad, gastrocnemius muscle, tonsil, inferior turbinate, conjunctiva, and liver aspiration on three occasions, however, the Kveim reaction was positive in June, 1959

Comment The many neurological findings, including signs of pyramidal tract and cerebellar abnormality, constitute a diffuse central nervous system involvement compatible with sarcoidosis This, plus the bizarre multiple localization of

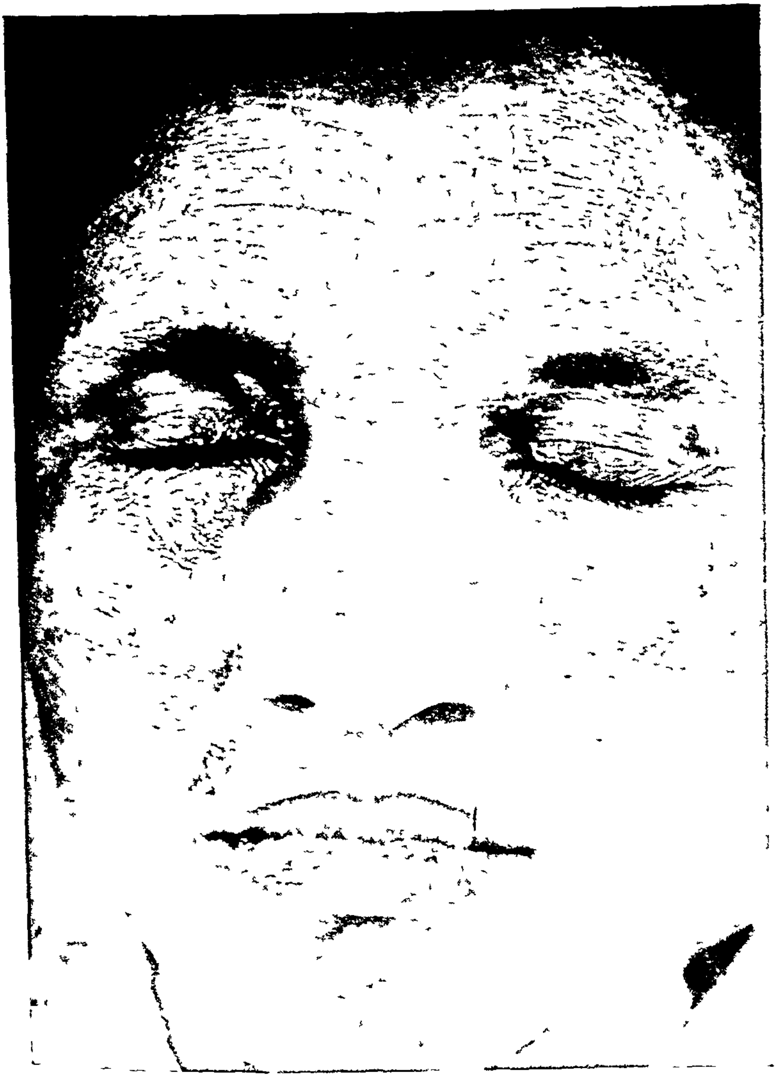

Fig 6 R S Case 9 Nasal deformity with widening of dorsum due to proliferative granuloma involving the ethmoid sinus middle turbinate lateral wall and nasal bones Tissue removed at lateral rhinotomy showed invasion of ethmoid mucosa and nasal bone. (See Fig 7)

small bone of the hand is almost pathognomonic of sarcoidosis, especially in association with the assorted lesions in the conjunctiva, skin, respiratory mucosa and lungs, however, the histologic evidence obtained is still important for confirmation of the diagnosis

Case 9 R S , a Negro female, aged 36 years, attended the Illinois Eye and Ear Infirmary in March, 1954, because of blockage of the nose Examination disclosed a generalized lymphadenopathy Subcutaneous nodules were present on the arms and face Both lacrimal glands were palpable There was no facial paralysis or parotid swelling The tonsils showed evidence of chronic inflammation The nasal mucosa appeared turgescent and infiltrated Small white nodular lesions were seen on the soft palate On direct laryngoscopy, a nodule 1 mm in diameter was excised from the left ventricular fold Microscopic examination showed a non specific granuloma In May, 1954, a local tonsillectomy was performed and a cervical node was removed, again the tissue study showed non specific chronic inflammation, negative for fungi and acid fast bacilli Laryngeal cultures were negative for fungi, yeasts and tuberculosis A supra orbital cutaneous nodule was excised in January, 1955 Microscopic study showed a granulomatous tissue X rays of the chest portrayed clear lung fields but increased bilateral hilar density Views of the hands and feet showed no changes Sinus views revealed clouding of the frontals and left antrum, the latter returned pus on irrigation

In May, 1957, bilateral nasal polypi were noted, associated with a swelling of the nasal dorsum and thickening of the turbinates and the septum This gradually progressed to a stage of nasal deformity with widening of the bridge (see Fig 6) X rays showed soft tissue increase with apparently intact nasal bones Biopsy of the septum and the dorsal swelling again showed an indeterminate granuloma On the basis of the X ray changes in the lung and the several biopsies, the diagnosis of Boeck's sarcoid was strongly presumptive

In May, 1958, a left lateral rhinotomy and left ethmoidectomy were done, removing a firm, fibrous, tumor like mass including part of the nasal bone, middle turbinate and lateral nasal wall Microscopic examination showed invasion of the ethmoid mucosa and the nasal bone by tubercles of a chronic granuloma consistent with sarcoidosis (see Fig 7)

Comment This case presents multiple lesions in the skin, lungs, lacrimal glands, larynx, lymph nodes, ethmoid sinus and nasal bone The two latter sites are very uncommon localizations

Case 10 R J , Negro female, aged 35 years, came to the Research and Educational Hospital of the University of Illinois in January, 1957, because of a roughened, painless area on the tip of the nose which had slowly developed in the preceding two years There were no nose and throat complaints The eyes were normal General physical examination was negative The pharynx, tonsils, nasopharynx and larynx were normal X rays of the chest and hands showed no changes The columella and alar margins showed numerous brownish red tiny papules, conglomerate in some areas (see Fig 8) Biopsy was reported as a chronic granuloma,

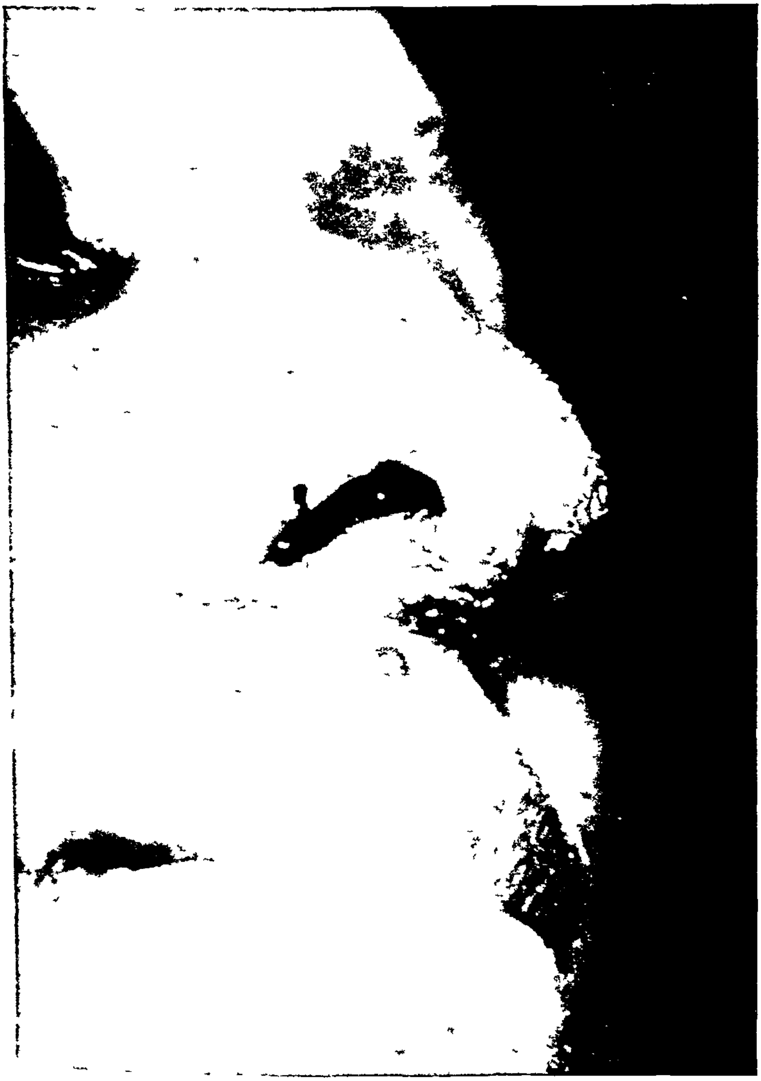

Fig 8 R J Case 10 Lesion of columella Groups of brownish-red pinhead papules conglomerated in some areas Ulceration was absent The nasal mucosa was not affected

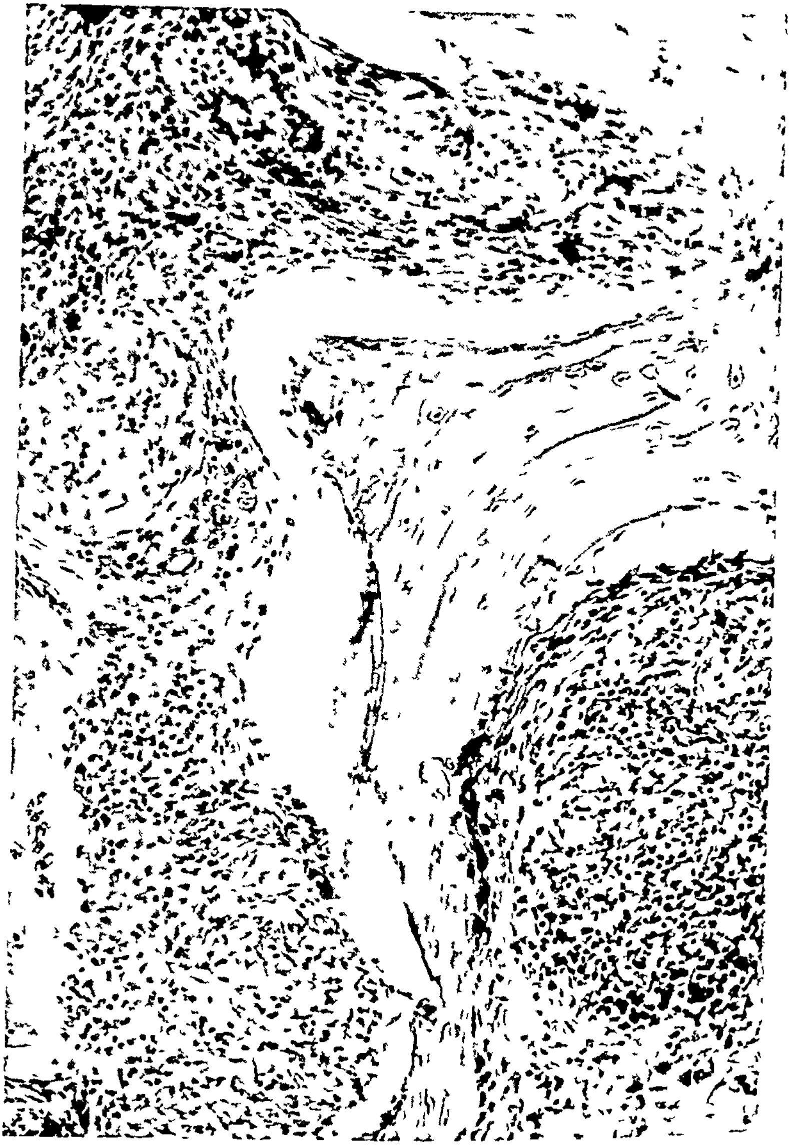

Fig 7 R S Case 9 Nasal bone (X100 H and E stain) with granulomatous infiltration between osseous trabeculae

in the early active period than in the late chronic stage of fibrosis

James emphasizes the importance of early diagnosis to anticipate and prevent serious irreversible changes He finds valuable clues in the age group, 20 to 50 years, in a history of erythema nodosum, episodes of irido-cyclitis or "atypical mumps" and in the presence of asymptomatic roentgenographic changes in the lungs Thorough examination is then indicated to find other signs, including unsuspected hepatosplenomegaly, generalized lymphadenopathy, osseous or nasal lesions [54]

The characteristic, yet not pathognomonic, microscopic structure of sarcoidosis occurs uniformly in any involved tissue Selection of the biopsy site depends on the available sources

1 Cutaneous lesion

2 Scalene or other peripheral lymph node (Lesions in sites 1 or 2 are stated to be present in about one-half of all cases at some time during the course)

3 Needle aspiration of the liver

4 Surgical specimens, such as the spleen or the tonsils

5 Lung biopsy, by the intercostal route or by thoracotomy

6 Bronchoscopy, for lesions in the tracheo-bronchial tree

7 Blind biopsy of the nasal mucosa, tonsil, conjunctiva or gastrocnemius muscle

Selection of one or more of the methods enumerated above for obtaining tissue for microscopic evidence of sarcoidosis should be governed by the judgment of the physician regarding *1* accessibility of the tissue, *2* ease of the procedure with minimal trauma, *3* feasibility in the particular patient under consideration, and *4* statistical probability of positive results Local lesions of indeterminate appearance must be carefully evaluated, especially when they occur in conjunction with undiagnosed pathology elsewhere Lederer's felicitous etiology undetermined, possibly blastomycosis The Kveim test was not done because of lack of the antigen

In April, 1957, a smooth, painless, intralingual nodule, 1 cm in diameter, was found Aralen, 500 mg daily, effected some improvement in the nasal lesion only Biopsy of the tongue revealed a microscopic structure consistent with sarcoid

Comment Despite the surprisingly inconclusive biopsy of the cutaneous lesion, the diagnosis was established by microscopic examination of the tongue, a very rare location for sarcoidosis

Case 11 E A, Negro male, aged 22 years, was one of the patients in the series of Snider and Mackler[67] He presented clinical and X ray evidence of diffuse pulmonary infiltration and bilateral hilar lymphadenopathy Tissue obtained by bronchoscopy from a site which showed mucosal thickening revealed sarcoid granuloma, while a scalene node biopsy was negative The Kveim test was not done

Comment The diagnosis in this case was made by the bronchial biopsy when tissue biopsy was negative

DIAGNOSIS

Varying viewpoints regarding diagnosis are exemplified by Longcope and Freiman[14] who state "The diagnosis is fraught with many difficulties ", while Middleton[6] declares " it should present little difficulty to an alert clinician " The latter takes into consideration the race, age, diverse clinical manifestations, the paucity of symptoms, X-ray changes in the bones and lungs, a negative tuberculin test, the indolent course with variations, the Nickerson-Kveim reaction, while ruling out other conditions, particularly the fungus diseases Characteristic cutaneous lesions are of value when present, particularly to direct search for other manifestations

Most authorities[6,10 11 13 14] are alerted to the possibility of sarcoidosis by a correlation of radiological and clinical features involving a varied combination of lesions, cutaneous, osseous, pulmonary, reticuloendothelial, lymphatic and ocular This pattern stimulates further examination To such a compatible clinical picture it is necessary to add confirmatory data of a characteristic histologic structure in tissue obtained by biopsy, the Kveim reaction, correlated laboratory data, and exclusion of various other diseases The diagnosis is easier roentgenographic evidence, combined with exclusion of other disease possibilities This situation, in his opinion, reveals the true value of the Kveim reaction as a confirmatory test for active sarcoidosis, within biologic limits[88]

Nelson's review of the world literature attests a low incidence of false positive Kveim reactions, ranging from 0 per cent to 6 5 per cent This is acceptable to him on the basis of the universal recognition that no biological test is completely specific[92] On the other hand, Israel and Sones are unwilling to accept the reliability of the Kveim reaction From several studies,[89-91] they conclude that the test in its present form is not sufficiently dependable to justify its use as a substitute for clinical and biopsy methods of diagnosis

Siltzback supports the general acceptance of the test as useful when tissue for biopsy is not readily at hand Having summarized reports of 560 tests from various sources in the literature* and finding an accuracy varying from 65 per cent to 92 per cent, he asserts that the Nickerson-Kveim reaction is a diagnostic aid of high specificity[89]

BLIND BIOPSY

A disadvantage of the Kveim reaction is the elapse of several weeks before a reading can be made An alternative is the blind biopsy, one taken at random, from a tissue which is apparently normal in appearance

The rationale of blind biopsy depends on a strongly presumptive diagnosis of sarcoidosis, the absence of obvious accessible areas (notably cutaneous lesions or peripheral lymph nodes) and on the observation of Siltzback, among others, that many more tissues contain sarcoid changes in the early stage than was previously suspected The status of this procedure has not been definitely substantiated Opinions vary as to the reliability of different sites, each having its adherents

Aspiration liver biopsy is supported by Mather and his phrase—"a strong index of suspicion"—is a valuable admonition for an essential and rewarding attitude.[85]

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*Nickerson 1941 Kveim[86] 1941 Putkonen 1943 Nelson 1949 Danboldt 1951 Siltzbach and Ehrlich[93] 1954 Rogers and Hazerik 1954 James and Thompson[94] 1955 Reed 1956 and Nelson and Schwimmer[95] 1957

If obvious lesions of sarcoid are not available, two other methods may be employed, the Kveim cutaneous reaction or the blind biopsy. The comparative advantages of these will be weighed.

THE KVEIM-NICKERSON CUTANEOUS REACTION

The confirmatory skin reaction for sarcoidosis was developed independently by Kveim[86] and by Williams and Nickerson.[87] It depends on a cutaneous reaction to the intradermal injection of a saline suspension of fresh sarcoid tissue derived from a spleen or lymph nodes. When the disease is active, there ensues in about six weeks, a palpable, purplish papule which on biopsy reveals the characteristic structure. The test is a simple, safe, specific aid to diagnosis, available to outpatients.

The relationships between tissue studies and Kveim reactions in this series are classified into four groups.

I Tissue biopsy positive Kveim test positive—18 cases

II Tissue biopsy positive Kveim test not done—10 cases

III Tissue biopsy positive Kveim test negative—2 cases

IV Tissue biopsy negative Kveim test positive—3 cases

The Kveim test at times presents interesting variations, not always subject to precise evaluation. Group I affords a double confirmation of sarcoidosis. In Group II, the Kveim was omitted, either because it was considered unnecessary or due to a lack of antigen.

Bornstein feels that the timing of the biopsy and/or Kveim reaction is a significant factor in the outcome. A test done concurrently with a positive tissue biopsy would almost certainly be positive as well. In Group III negative Kveim could reflect an inactive phase of the disease process; furthermore, he considers the cases in Group IV to be true sarcoidosis on the basis of the characteristic clinical and

"Although, usually, there are no conspicious clinical findings on the part of the tonsils, this does not preclude the presence of microscopical involvement and in some cases biopsy may be of value for confirmation of the clinical diagnosis "[13]

In the author's series, blind biopsies were done in 32 cases Tissue was removed under topical 10 per cent cocaine anesthesia using a small loop of an electrosurgical apparatus with a combined cutting and hemostatic current In 22 cases specimens were taken from a tonsil and an inferior turbinate In an additional ten cases the turbinate alone was used, making a total of 22 tonsil and 32 turbinate biopsies Positive microscopic evidence was returned in two of the 22 tonsil sections (9 1 per cent) and in two of the 32 turbinates (6 3 per cent) The combined figure of four positive biopsies in 32 patients represents an incidence of 12 5 per cent There was no gross local evidence of the disease in the four patients with positive results These included Case 3 (tonsil) and Case 7 (turbinate) Although in 1954 the latter patient had exhibited

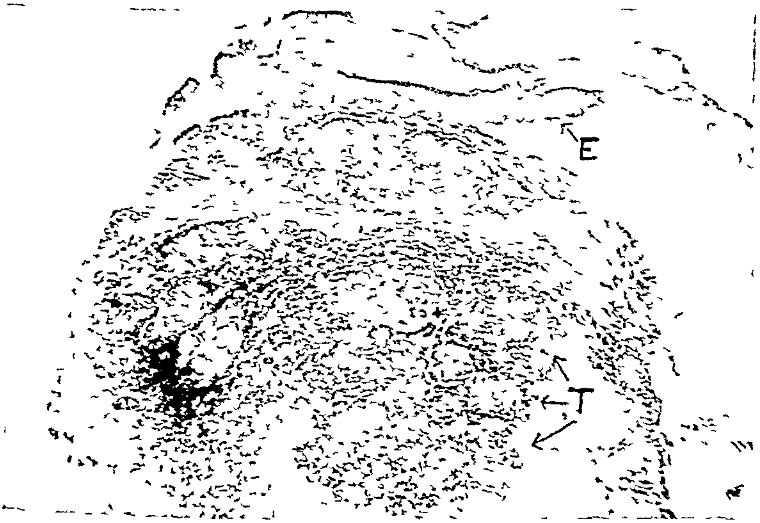

Fig 9 E B Tonsil (X50 H and E stain) blind biopsy specimen showing epithelioid tubercles (T) replacing tonsil structure Note epithelium (E) and a scattering of remaining lymphocytes

associates[96] while Norvitt and his co-workers[97] endorse the scalene node site. James asserts that two-thirds of the patients with sarcoidosis will yield positive results in these two areas.[81] Other locations that have been attractive as a source of microscopic evidence are the bronchi, nasal mucosa, conjunctiva, and tonsil.

Because of its infrequent success, James was not impressed with the value of bronchoscopic biopsy of normal-appearing mucosa, however, various observers have established a difficult diagnosis in some cases by its use.[11,61-66] The importance of this procedure is greatly enhanced by the success of Snider and Mackler in obtaining positive random bronchoscopic biopsies in 14 of 18 cases. They consider that this incidence of 77.7 per cent is high enough to justify further study and wider adoption of the method.[67]

Blind conjunctival biopsy advocated by some authors, was of no diagnostic aid to James[75,84] as he found it to be negative in ten cases of sarcoidosis, including five cases in which sarcoid iridocyclitis was present.

Various reports indicate that the nasal mucosa of the inferior turbinate or septum occasionally yields a positive random biopsy.[10,11,13]

Tonsillar biopsy has been strongly advocated by European authors. Schaumann reported a 100 per cent positive incidence in 21 cases.[16] Gravesen's data indicated a 64 per cent occurrence of tonsillar lesions in 70 patients. For this reason, he considered tonsil biopsy important and advised a large specimen, even preferring tonsillectomy for tissue study.[31] In this connection it is a matter of record that in very rare instances an incidental tonsillectomy, performed for other indications, has fortuitously disclosed the histopathology of sarcoidosis. Schubert and Neuss, in their *excellent review* in 1958, urge tonsil biopsy as an early aid when the diagnosis is occult.[60]

Though finding two positive tonsil biopsies in 50 patients (4 per cent), James dissents from the value of this procedure.[11] To this viewpoint Reisner's contrary *opinion* is pertinent

DIFFERENTIAL DIAGNOSIS

The differential diagnosis often depends on the location of the lesion Among those to be excluded are tuberculosis, Hodgkin's, lymphoma, syphilis, systemic berylliosis, and the fungal disease (blastomycosis, coccidiomycosis and histoplasmosis) Other nasal granulomata may be confusing In lupus vulgaris the miliary nodules are stated by Ulrich[50] and by Wille[51] to be softer on palpation by probe Rhinoscleroma may be recognized by a culture of the bacillus of Frisch (Klebsiella rhinoscleromatis) and the typical microscopic structure[98] In regard to leprosy Lever states that the histologic criteria alone do not provide a differentiation between the two diseases[99]

Israel and his co-workers point out that chronic disseminated histoplasmosis may clinically and microscopically simulate sarcoidosis They emphasize the necessity of utilizing distinguishing criteria, including the histoplasmin skin reaction, serologic tests, and a search for Histoplasma capsulatum by culture of oral and laryngeal secretions and by the Hotchkiss-McManus special tissue stain[100]

SUMMARY

1 The clinico-pathological aspects of sarcoidosis are presented and the otolaryngologic manifestations recorded in the literature are analyzed

2 Eleven cases of diverse sarcoid lesions of the respiratory tract are reported Eight of these were from an unselected group of 33 cases of sarcoidosis, an incidence of 24 per cent Three cases are added of previously established lesions

3 In 32 cases, blind biopsy was done of a tonsil and a turbinate in 22 cases and of a turbinate alone in ten cases Two of the 22 tonsil examinations were positive for the disease (9 1 per cent) and two of the turbinate examinations were positive (6 3 per cent) The combined figure of four positive results in 32 patients represents an incidence of 12 5 per cent of successful blind biopsies

a lesion of the nasal mucosa, in 1959 there was no evidence of sarcoidosis, so that essentially this was a "blind biopsy" The second positive tonsil biopsy was obtained in a Negro female, aged 35, with pulmonary, mediastinal and glandular lesions and a previous scalene node confirmatory biopsy (see Fig 9) The other positive turbinate examination was from a patient with multiple manifestations and a positive Kveim test (see Fig 10)

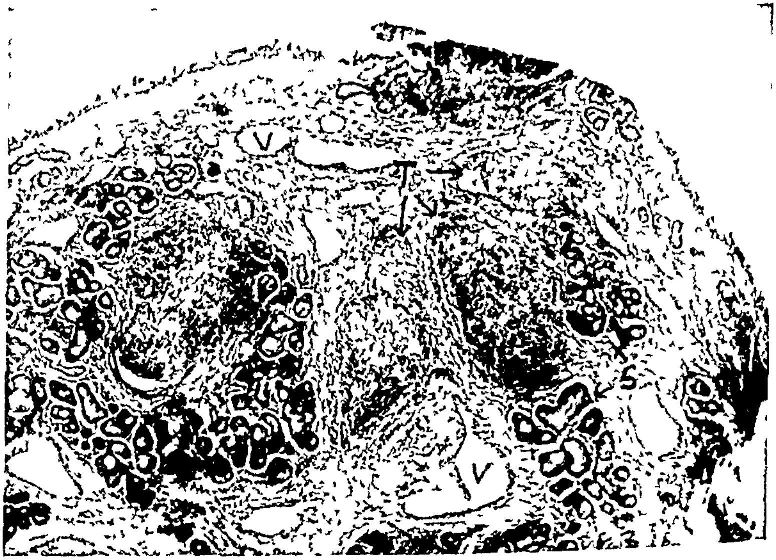

Fig 10 R R Inferior turbinate (X75 H and E stain) Tissue obtained by blind biopsy revealing infiltration of submucosa by discrete tubercles (T) Note epithelium at E seromucinous glands (S) and venous spaces

Despite the modest incidence of positive results, it seems reasonable to employ blind biopsy of the nose and tonsil to expedite the solution of a diagnostic problem These sites (as well as the bronchial mucosa) deserve a high priority in obtaining tissue for study Because of safety, minimal trauma and obvious accessibility they might well be utilized before such procedures as peripheral node excision or liver needle aspiration The probability of early involvement of the respiratory mucosa is an added reason for investigation of this area

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4 On the basis of safety, accessibility and minimal trauma the tonsil and turbinate merit strong consideration as sites for blind biopsy to obtain tissue for microscopic confirmation of a diagnosis of sarcoidosis This applies also to the bronchial mucosa

5 The several combinations of positive and negative tissue biopsies and Kveim reactions in this series are discussed

6 Critical appraisal of the upper respiratory tract and associated areas in the cases studied revealed a higher incidence of sarcoid lesions than has been reported in the American literature

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PARTIAL LARYNGOPHARYNGECTOMY AND NECK DISSECTION FOR PYRIFORM SINUS CANCER

Conservation Surgery with Immediate Reconstruction

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INTRODUCTION

Sacrifice of the voice is not always necessary for the adequate excision of tumors of the hypopharynx and larynx In certain carefully selected cases, lesions of the aryepiglottic fold, medial, lateral and anterior wall of the pyriform sinus, and the lateral wall of the hypopharynx can be excised in continuity with their potential field of spread and the defect closed primarily without sacrificing the larynx

The principles of this conservation surgery is based on the personal experience of the senior author in treating malignancies of the epiglottis[20] Confirmatory data have been obtained by the clinical, and pathologic study of the laryngectomy specimens removed at this institution[19,20]

HISTORICAL REVIEW

Preservation of the voice combined with adequate excision of the laryngeal or laryngo-pharyngeal malignancy has been

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ly controlled” He refers to other series with five-year cure rates of 6 to 15 per cent[15] Murphy[16] says that “an extended surgical procedure is more successful than irradiation in most patients with laryngo-pharyngeal cancer” “ a laryngectomy or laryngopharyngectomy with a lymph node dissection on the involved side should be done” “Radiation therapy will give five-year controls in about 10 to 15 per cent of cancers of the laryngopharynx”

Powers[27] feels that it is not possible to treat the entire neck adequately to a cancerocidal dose of therapy He recommends surgery for the primary with prophylactic neck dissection due to the high incidence of nodes Irradiation can often control the primary, but failure is often on the basis of node persistence

Wood[31] gives a two-year survival of 25 per cent (life-table method) for pyriform sinus lesions

Results are hard to evaluate because the better series frequently include epiglottic lesions, and the poorer series include postcricoid cancer

Surgery of the hypopharynx appears to be giving better results[7,17,28] Pietrantoni[26] gives a five-year survival of 16 per cent for pyriform sinus lesions The senior author has reported 29 per cent five-year survivals for these cases[18] None of these cases had pre- or postoperative irradiation, and all had total laryngectomy and radical neck dissection In 1943, Colledge[6] reported a ten-year survival of six cases of a total of 16

It has been demonstrated that total laryngectomy is not necessary[3,4] in all these cases if the surgeon is willing to undertake the more difficult task of accurate evaluation of the patients and the selection of suitable cases for partial laryngectomy

SURGICAL CRITERIA

To apply this conservative procedure we must have an exact preoperative localization of the tumor, especially the inferior margin We find laryngograms very valuable in confirming the goal of surgeons for many years For purposes of this discussion endolaryngeal or intrinsic laryngeal tumors and the methods used in their treatment, such as laryngofissure or hemilaryngectomy, will not be included We are concerned here only with tumors in the pyriform sinus, on the lateral wall of the hypopharynx, or the aryepiglottic fold, where the vocal cords and the arytenoids themselves are not involved and where the surgical treatment has been total laryngectomy[5,6,12 21,24]

Trotter[30] and Orton[25] have provided the groundwork for conservation surgery of the larynx Alonso[3 4] has been a pioneer in this form of surgery and continues as an outstanding contemporary

Surgery of this area has been described under many names lateral pharyngotomy (Trotter[30]), transthyroid pharyngotomy (Orton[25]), transhyoid pharyngotomy and partial vertical laryngectomy (Alonso[3]) At the time of their initial publication the majority of these procedures had several common features, a pharyngostome was closed secondarily at a later date A neck dissection was not done routinely and anatomic area of excision of the larynx was not clearly defined Trotter's initial conservative surgery[30] was applied primarily to lesions of the lateral wall of the hypopharynx, and the approach was termed a lateral pharyngotomy At this time antibiotics were unknown, and the morbidity and mortality were high This procedure never achieved widespread popularity partly for this reason and partly because of the advent of radiotherapy From that time until approximately 1950, most of these pyriform sinus neoplasms were regarded in the United States as inoperable and were treated by irradiation LeRoux-Robert[11] has advocated combined surgery and irradiation as the initial plan of treatment

Up to the present time in the United States these pyriform sinus lesions have been treated by irradiation, total laryngectomy, or by a combination of both Results of irradiation have not been good[9 13] Del Regato quotes Coutard as having a five-year survival figure of 11 per cent for hypopharyngeal tumors[8] Moss states that lesions of this area are "infrequent-

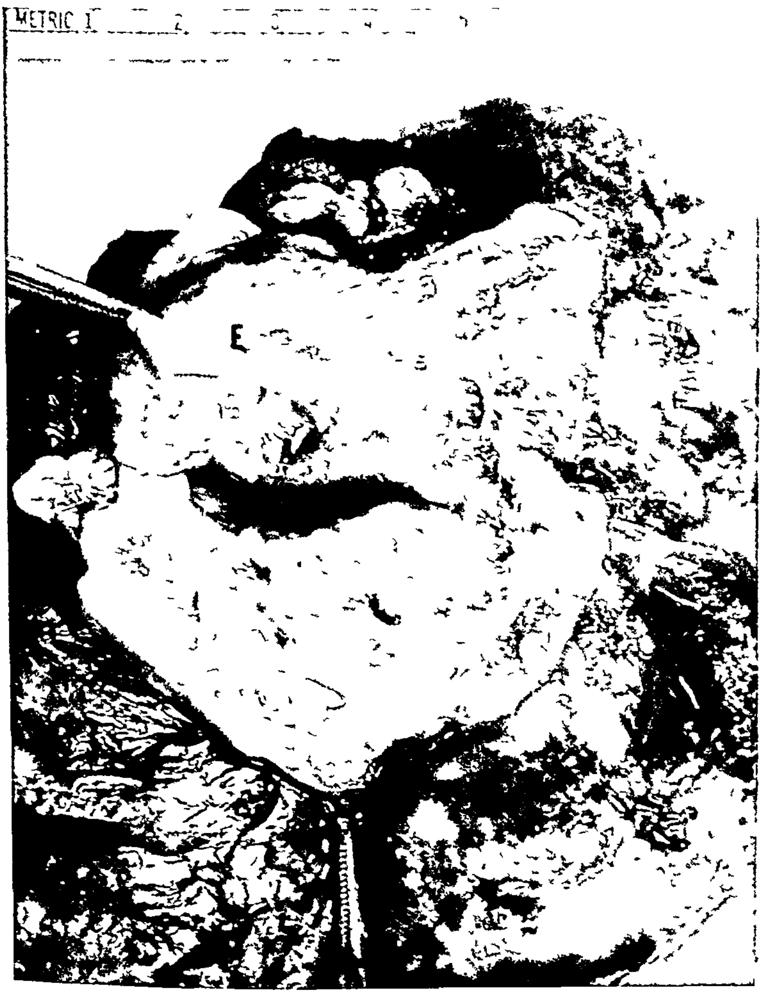

Fig 2 Specimen of a partial laryngopharyngectomy and neck dissection A—arytenoid E—part of epiglottis

This method is applicable to lesions of the aryepiglottic fold, anterior, medial and lateral pyriform sinus and lateral wall of hypopharynx from the pharyngo-epiglottic fold above, to a point just superior to the apex of the pyriform sinus, below It is not advised for lesions involving the lateral or posterior surface of the cricoid We do not include lesions of

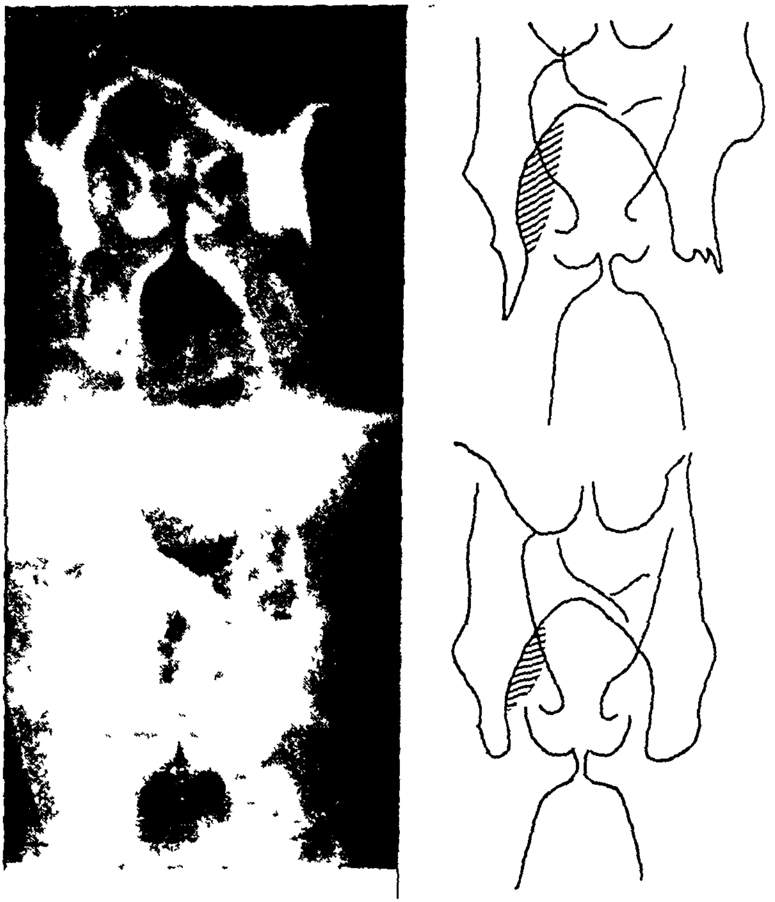

Fig 1 Laryngograms of pyriform sinus cancer Note localized lesion on medial wall of pyriform sinus with a clear apex

our clinical impression that the vocal cords and arytenoids are free of tumor[23] Sometimes a bulky tumor may hang over the glottis and make direct examination impossible Here, laryngograms are very important (see Figs 1 and 2)

In 1956, Jesberg[9] commented on the subject of conservative surgery of the laryngopharynx and partial laryngectomy and stated "until we possess better preoperative and diagnostic aids for measuring the extent of malignant growths it will be impossible to select cases for this procedure" Laryngograms, we feel, are this preoperative diagnostic aid[23]

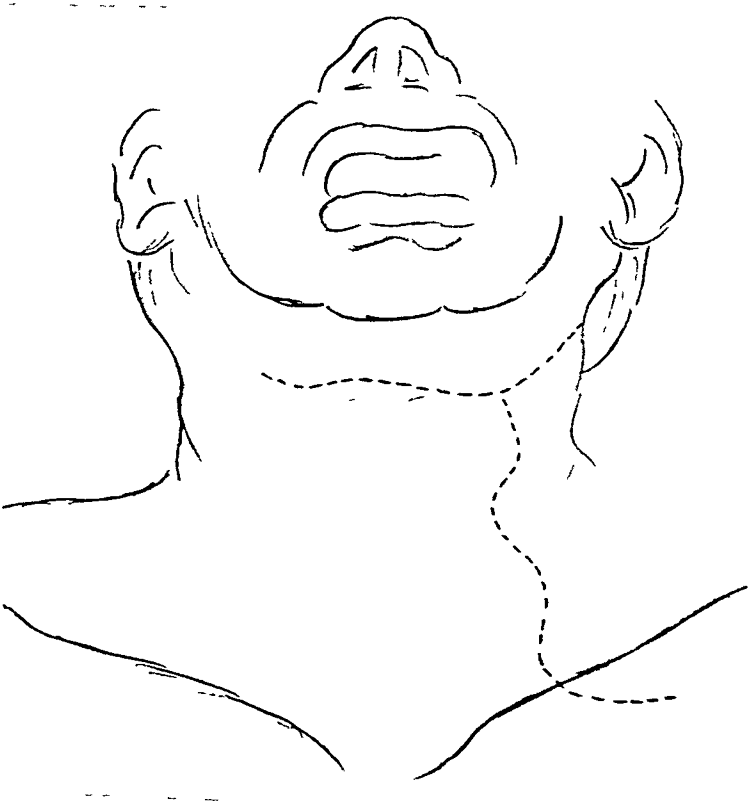

Fig 3 Skin incision

separated down to the level of the mucosa of the pharynx in the anterior and medial vallecula well above the site of the tumor

The sterno-thyroid and thyro-hyoid muscles are transected at the level of the upper border of the ipsilateral thyroid cartilage The external perichondrium is cut horizontally at the same level and elevated to form a flap and to expose the outer surface of the ipsilateral thyroid ala The perichondrium is elevated from the opposite side if it requires partial removal Using the Stryker saw the cartilage is divided in one of two ways, depending upon the site of the tumor (see Fig 4) the posterior wall here as these are to be covered in a subsequent paper [22]

The following criteria must be fulfilled before this type of partial laryngectomy can be performed *1* The true cords and arytenoids must be freely mobile and free of gross tumor involvement *2* The apex of the pyriform sinus must not be involved by tumor *3* There must be no thyroid cartilage invasion *4* Edema of the arytenoid eminence is not a contraindication

Most of the neoplasms in the locations under discussion (upper half of hypopharynx) are exophytic in type and are more amenable to surgery The tumor margin is frequently "pushing" in nature rather than infiltrative, and this seems to better the prognosis [2,29]

A radical neck dissection is done on the involved side in all cases whether nodes are palpable or not The incidence of cervical metastases for pyriform sinus cancer has been estimated from 40 to 60 per cent [10,15] In an operated series the senior author has recently published,[18] metastatic nodes were present in 43 per cent

SURGICAL TECHNIQUE

A nasogastric feeding tube is inserted prior to surgery The patient is prepared and draped while awake A vertical tracheostomy is done under local anesthesia through tracheal rings two, three and four A cuffed endotracheal tube is inserted and general anesthesia begun The usual Y incision is made, but the anterior limb of the Y is made lower and continued to the midline at the level of the hyoid bone (see Fig 3) The customary complete neck dissection is carried out leaving the pedicle attached to the thyro-hyoid membrane and upper thyroid ala The thyroid lobe adjacent to the pyriform sinus is resected with preservation of the recurrent laryngeal nerve The suprahyoid muscles are separated from the upper border of the hyoid on the involved side and the anterior face of the body Using the Stryker saw or heavy scissors the hyoid is divided in the midline, and the ipsilateral half is retracted downward The muscles and fascia are

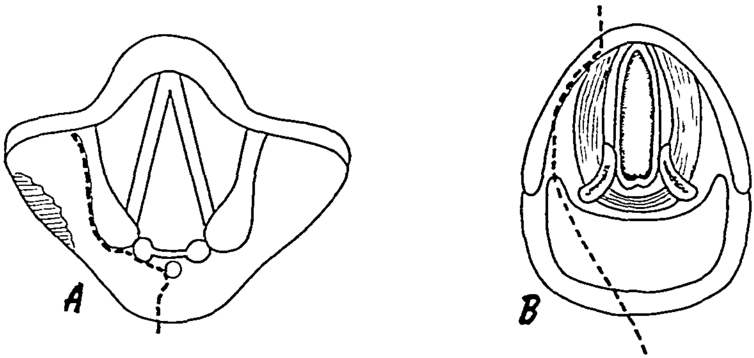

Fig 5 Anatomic area of resection for lateral wall of pyriform sinus A—mirror view B—cross section at level of glottis

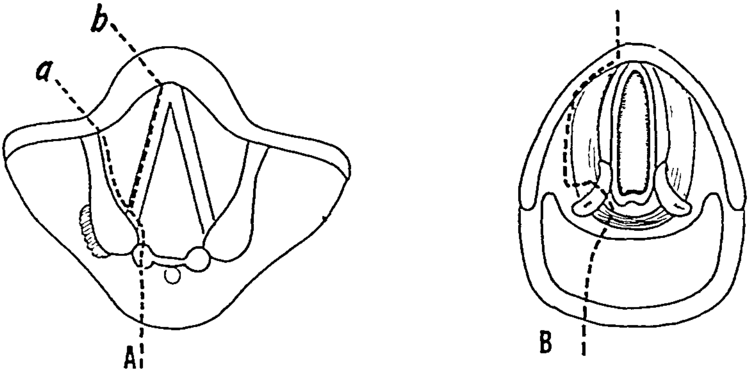

Fig 6 Area of resection for medial wall lesion Aa—in absence of aryepiglottic fold edema Ab—aryepiglottic fold false cord and partial epiglottic resection B—cross section at level of true cords

If the medial wall of the pyriform sinus is involved the cartilage is cut as before, and the aryepiglottic fold, part of the projecting epiglottis, false cord, and entire arytenoid, if needed, are excised with the tumor *en bloc* (see Fig 6) If the arytenoid is freely mobile but edematous it is removed except for the vocal process, and the posterior end of the vocal cord is sutured to the posterior midline of the cricoid (see Fig 7), and the remaining mucosa at the opening of the esophagus is approximated and sutured without tension, but no attempt is made to close the defect entirely with mucosa

For lesions of the lateral wall the ipsilateral cartilage is cut obliquely from the median notch to a point just superior to the inferior cornu

For the other primary sites the cartilage is divided on both sides On the ipsilateral side it is divided as above, but the medial end of the cut is below the notch and just superior to the anterior commissure From this point the contralateral cartilage is divided with saw or heavy scissors and the antero-superior portion freed, leaving the superior cornu intact

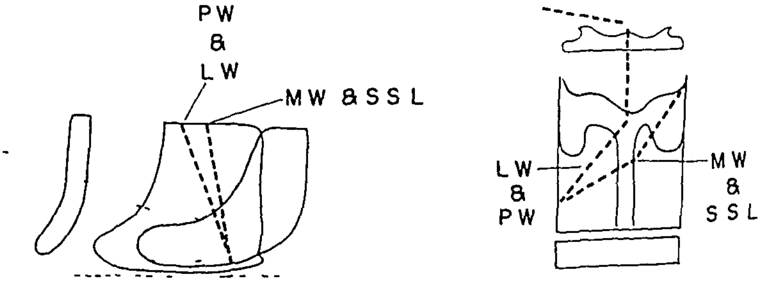

Fig 4 Cartilage transections LW—lateral wall of hypopharynx PW—posterior wall of hypopharynx, MW—medial wall of pyriform sinus, SSL—supraglottic subtotal laryngectomy

The mucosa of the vallecula which was previously exposed is opened and the tumor visualized and inspected but not deliberately palpated to avoid possible tumor spread

If the lesion occupies the lateral wall of the pyriform sinus, the laryngeal vestibule and epiglottis are not disturbed but the ipsilateral half of the hyoid bone and the postero-superior portion of the thyroid cartilage are removed in continuity with the primary lesion and the neck dissection specimen (see Fig 5)

If the lesion involves the anterior portion of the pyriform sinus, the cartilage is divided bilaterally as described above, and the approach through the vallecula is the same, however, the entire epiglottis is removed with the pre-epiglottic space, the aryepiglottic fold, pyriform sinus (anterior, medial and lateral wall), portion of the thyroid cartilage and the ipsilateral hyoid

Malignancies of the lateral wall which involve the posterior wall of the hypopharynx are not included in this discussion because they require a different method of reconstruction and will be covered in a subsequent paper dealing with the posterior wall neoplasms.[22]

In all of the cases of pyriform sinus excision, primary reconstruction of the pharynx is achieved by suturing the base of the tongue to the previously prepared perichondrial flap with silk sutures The margin of mucosa at the posterior border of resection is not sutured, it is tucked inward and the above mentioned stay sutures to the base of the tongue are tightened (see Fig 8) Initially, these cases were closed by the use of a split thickness skin graft sutured to the under surface of the musculoperichondrial flap; however, follow-up revealed that this was not necessary for *small* defects About one-half of each graft over the lateral wall did not take and seemed to prolong convalescence Large pharyngeal operative defects should have a skin graft sutured to the musculoperichondrial flap This avoids secondary partial pharyngeal scar tissue distortion of the pharynx and larynx For resection of the same area, these skin grafted cases had better functional results than those of comparable size without grafts Fascia and muscles are closed with two layers of interrupted fine silk Silk is also used for closure of the subcutaneous tissue and the skin The wound is drained and the pressure dressing applied

The tracheostomy tube is inserted regardless of the location or extent of the excision The patient will be tube fed postoperatively starting 24 hours after surgery, and this is continued until oral feedings are adequate

Ten to 14 days postoperatively indirect laryngoscopy is done and the airway evaluated If the edema has subsided and the airway is adequate, the size of the tracheostomy tube is progressively reduced to permit laryngeal breathing with the tube plugged. If this be tolerated, the patient is permitted to swallow beginning with thick liquid feedings such as gruel Some patients will swallow early but slowly from the beginning, others are much slower When the arytenoid has

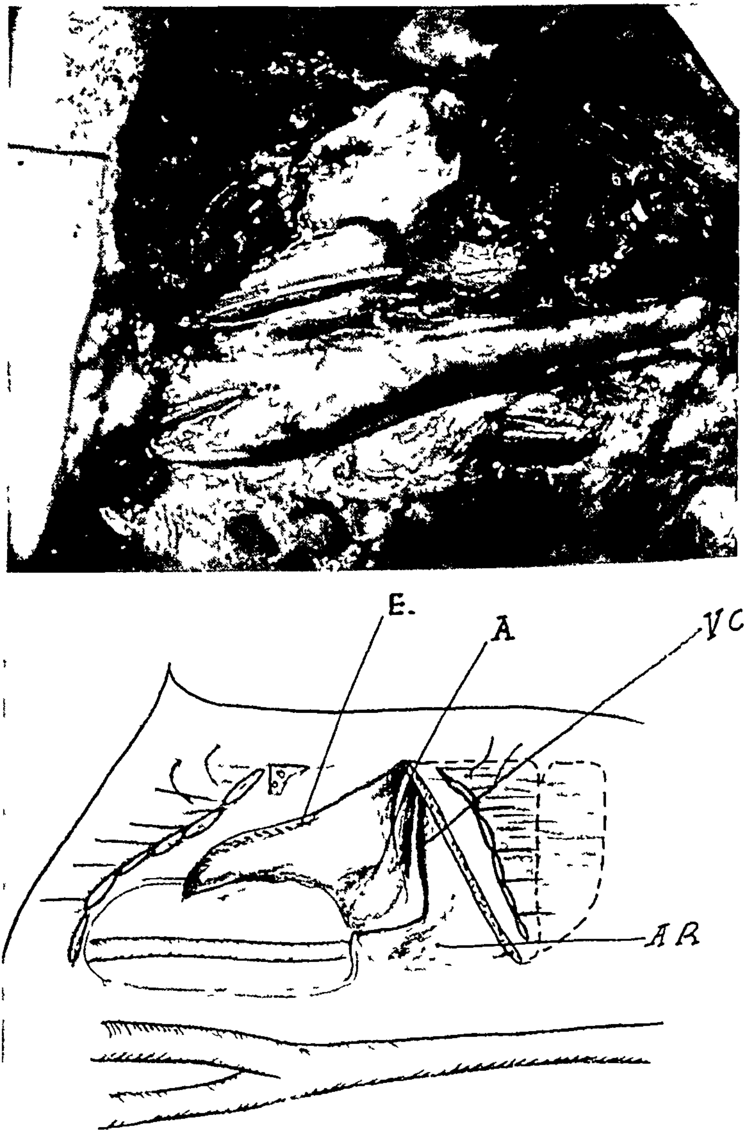

Fig 7 The line drawing is a copy of the operation defect photograph E—part of epiglottis, A—remaining functioning vocal cord VC—flabby remaining cord following removal of arytenoid AR—area defect left by removal of arytenoid with specimen

been removed, swallowing is usually considerably delayed The tracheal tube which has been reduced to approximately No 5 is plugged but never removed until satisfactory swallowing without aspiration has been established The period of rehabilitation may require two to six weeks, and the patient needs constant encouragement When swallowing without aspiration has been established, the feeding tube and the tracheal tube are removed and the tracheostomy wound allowed to close spontaneously (see Fig 9)

CASE PRESENTATION

A small series of 13 patients with pyriform sinus neoplasm is presented Of these 13 cases six were in the lateral wall and seven were in the medial wall Eight had clinically palpable metastatic nodes on one side Four had node metastases that were not palpable i e four of the five patients with clinically negative necks had microscopic metastases

In the six cases a skin graft was used, and hospitalization was prolonged to four to six weeks When it was not used the hospital stay was reduced to an average of three-and a half weeks All were talking and swallowing adequately when they left the hospital At the present time skin grafts are used only for large defects to avoid secondary distortion

The arytenoid was removed wholly or in part in all seven cases of medial wall tumors In one of these cases late postoperative swallowing difficulty was due to gradual scarring causing lateral displacement of the vocal cord and subsequent aspiration This was corrected by replacing the cord in the midline and maintaining this position with a cartilage graft as Meurman[14] has described

Of the 13 cases six were of the “pushing variety and seven were infiltrating[2,29] In one case there was doubt as to whether the lesion in the pharynx was a direct extension of a node metastatic from another site or a primary in the pharynx He is now three years postoperative and there is no sign of another primary

The size of the primary tumors ranged from 2x1 cm to 4x2 cms The longest survival so far is three-and a half years Most of the patients are alive and free of disease one to two years since surgery One man has died since without evidence of persistent tumor or recurrence He developed a second primary in his lung and died following a thoracotomy Only in one instance has there been a local recurrence or persistence of tumor This was an ‘infiltrating type of tumor’ and probably would have recurred if total laryngectomy were performed in this instance (The margin of resection was apparently grossly adequate)

DISCUSSION

For many years the senior author[21] was against conservative surgery for hypopharyngeal and laryngeal cancer on the grounds that the patients’ chances of survival would be jeopardized by anything less than radical excision of the larynx and cervical nodes

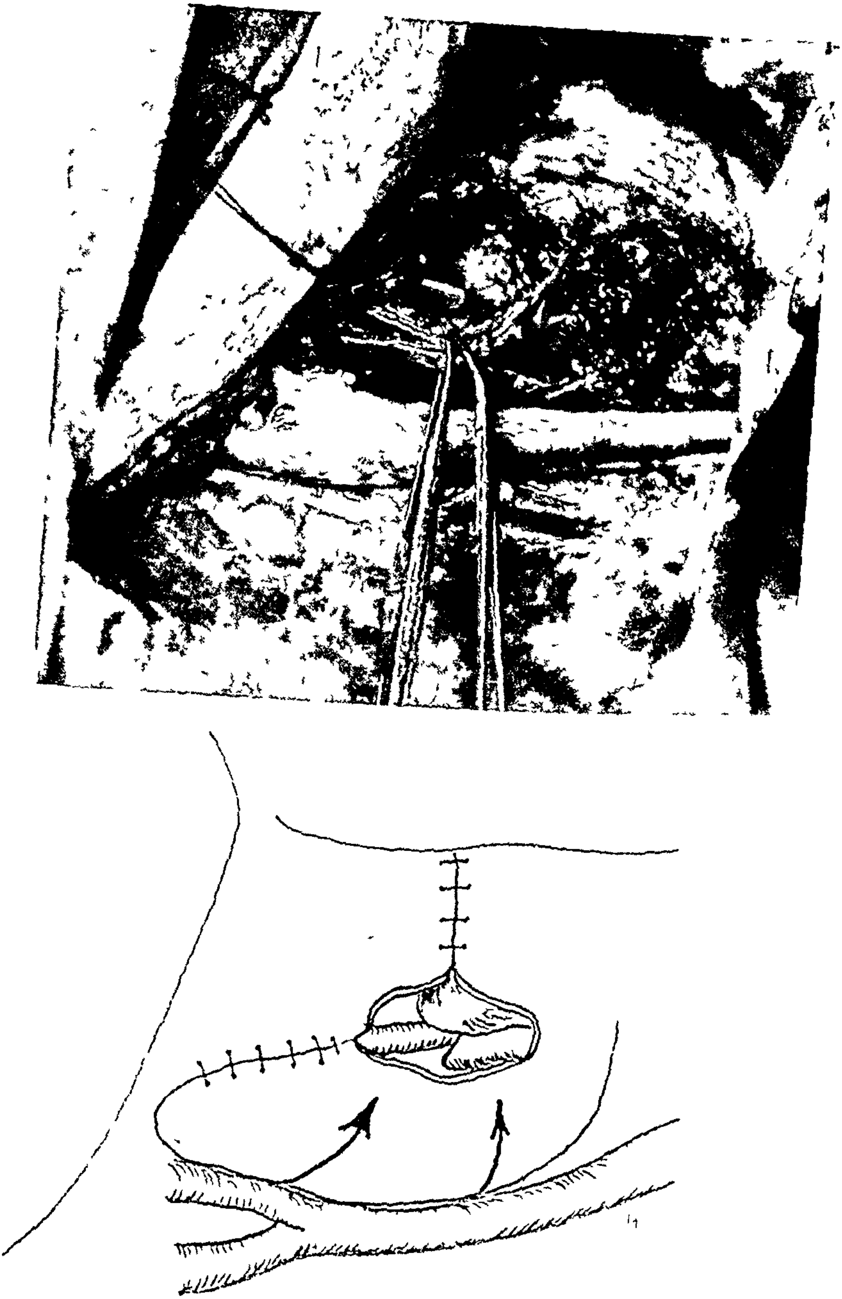

Fig 8 Immediate closure of pharyngostoma This is accomplished by closure of mucosa of tongue with musculo-perichondrial flap of thyroid (M P)

Involvement of the larynx by these tumors is only by direct extension from large tumors or possibly retrograde spread due to plugged lymphatics

On examining the long term results of laryngectomy and neck dissection for these "extrinsic" tumors, it was found that only 32 to 37 per cent survived[18] Conservation surgery that was equally adequate although less radical was attempted several years ago, and the short term results confirm our impression that such conservation procedures do not jeopardize the patient's chance for survival and yet allow him to retain his voice Many of these patients have passed their second year without recurrence in the larynx The development of obvious persistent tumor and recurrences have been in such locations that a total laryngectomy would not have prevented them, hence, we felt that conservative surgery of the larynx with preservation of the voice is possible and advisable with certain selected cases of laryngopharyngeal cancer

Examination of the pathologic specimens shows that edema of the arytenoid has not meant tumor involvement,[29] however, when edema is present the arytenoid is wholly removed to give an added margin of safety (except for vocal process)

When portions of the arytenoid are removed the cord becomes fixed, and it is very important that the cord remain fixed in, or close to, the midline, otherwise aspiration may be a problem This can be corrected by fixing the cord in the midline by suture to the cricoid and by a cartilage graft inserted between the thyroid ala and the internal perichondrium[14] This can be done at the initial surgery or later if it becomes necessary

Tracheostomy is done routinely on all these patients and is left in place until they are able to swallow without aspiration The cough reflex is usually sufficient, but we do not rely solely on this mechanism to maintain patency of the airway The tracheostomy is used as a safety valve to permit suctioning when needed during the process of re-education and readjustment

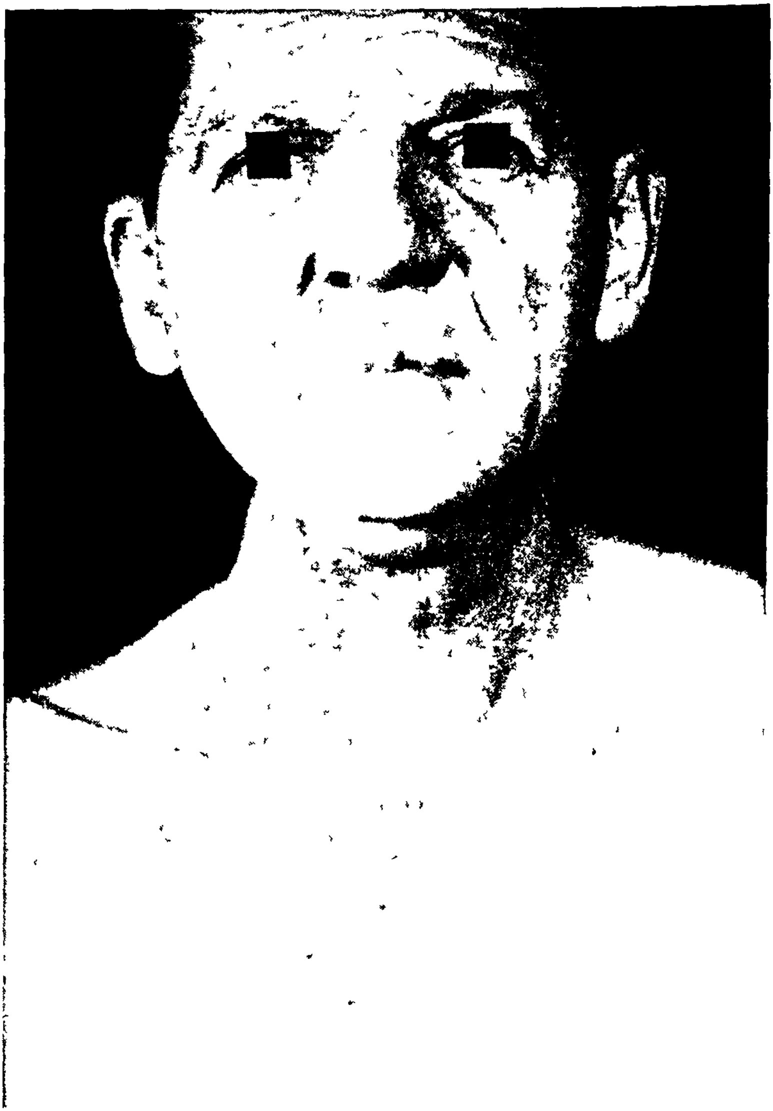

Fig 9 Postoperative photograph of patient

On examining the laryngectomy specimens at this institution it was soon apparent that the arytenoids and vocal cords were completely free of tumor in many of the specimens removed for laryngo-pharyngeal malignancy[29] Study of the lymphatic drainage from the hypopharynx shows that it is lateral toward the nodes and not toward the larynx[15,19,20,29] cure rate should be no better than a comparable series treated by total laryngectomy and neck dissection There should, in this instance, be no need for radical excision, *2* the short term follow-up is very encouraging in that early local recurrences appear to be no more frequent than for total laryngectomy, *3* the extent of area resection of the larynx consistent with preservation of voice and swallowing function includes the pyriform sinus, one arytenoid and any or all of the supraglottic structures

SUMMARY

Partial laryngo-pharyngectomy for carcinoma of larynx and hypopharynx with conservation of the laryngeal function and reconstruction of the pharynx is the treatment of choice for certain carefully selected tumors of medial, anterior and lateral wall of the pyriform sinus, of the lateral hypopharyngeal wall and aryepiglottic fold It is not advised for malignancies involving the apex of the pyriform sinus or the posterior or lateral aspect of the cricoid

Primary closure in these cases is facilitated by the routine performance of a radical neck dissection The postoperative course is more prolonged than for a total laryngectomy, but the preservation of function is well worth the delay A series of 13 cases with short term observations is presented and discussed

CONCLUSION

Partial laryngo-pharyngectomy in continuity with radical neck dissection with preservation of laryngeal and pharyngeal function is recommended as the treatment of choice for certain selected lesions of the larynx and hypopharynx

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We have not used a stent to occlude the larynx The pulmonary complications although frequent have been mild and did not appear to warrant the cord trauma of a stent or the lateral cord displacement that would occur in cases where the arytenoid had been removed, however, the idea merits further consideration Most of the pulmonary difficulty is at the onset of swallowing rather than in the immediate postoperative period, and it is in the latter period that the stent would find its greatest usefulness

This series of 13 cases shows an over-all rate of cervical metastases of approximately 90 per cent A neck dissection is done on the ipsilateral side whether or not nodes are palpable If nodes appear on the contralateral side, a neck dissection is performed later

Fistulas occasionally occur, but when present are usually small and spontaneous closure is obtained if a pressure dressing is applied Wound infection, skin slough, and postoperative hemorrhage may occur as with other surgery, and the incidence is no greater

Reconstitution of swallowing is related with re-establishment of the laryngeal airway Voice quality is hoarse when the arytenoid is removed but is of excellent tone when it is not removed Large pharyngeal operative defects should be skin grafted even though convalescence is slightly prolonged over smaller pharyngeal defects closed without skin grafts When such defects are small, there is little difference in deglutitive function with or without grafts, but with larger defects, functional results are better with skin graft compared to those without grafts

It is admitted that these observations represent only a short term follow-up In time, with a better accepted classification scheme, an accurate appraisal of the primary lesion and a long term analysis of homogenous sampling of these cases, the selection of patients suitable for this operation will be better delineated, however, we believe it worth while to present this surgical approach in these cases for three reasons *1* the biological behavior of these tumors in terms of five-year

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THE ABNORMALLY OPEN EUSTACHIAN TUBE

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The Eustachian tube occasionally acquires an abnormal patency that results in its lumen remaining open for considerable periods of time, or even continuously This abnormal patency produces a clinical entity, the symptoms of which are more disturbing to the patient than is an occluded Eustachian tube This condition has been discussed only sporadically in the literature of this century, however, it is not rare and should be kept in mind when confronted by a patient who complains of a low-pitched roaring noise in the ear or of a "stopped-up" ear It apparently occurs more often than is generally recognized but may be frequently overlooked

The purpose of this paper is to review the literature and to discuss briefly the author's experience with 31 patients seen with this entity during the past seven years I shall call attention to an etiologic factor that has not heretofore been mentioned in the literature and to an additional method of treatment in the persistent cases

HISTORY

The history of this condition has been reviewed in detail by Perlman[1] (1939) in an excellent, comprehensive paper He gives credit to Jago, an otologist who worked with Toynbee, as being the first to describe this clinical entity in 1867 Jago himself had symptoms over a period of 14 years, which he correctly interpreted as being due to a continuously open Eustachian tube He believed this to be a rare phenomenon Since 1890, textbooks on otology have made only occasional mention of this condition A review of the literature back to 1929 reveals only five papers on this subject in the American literature and one by Zollner[2] (1936) in German

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REVIEW OF ANATOMY AND PHYSIOLOGY

The normally functioning Eustachian tube remains closed in the shape of a vertical slit in its membrano-cartilaginous portion, except during the act of swallowing or yawning.

The normal closure is probably maintained by two forces one of them passive and the other active. It has been generally accepted that passive closure is maintained by the pressure of surrounding soft tissue structures, and especially by a thick pad of fat which surrounds the lateral wall of the cartilaginous tube, the fat layer of Rudinger.

In 1955, Guild[3] made a study of the elastic connective tissues in the walls of the human Eustachian tube, originally described by Reitmann[4] and by Citelli[5] half a century earlier. He found the elastic tissue to be quite dense over the "cap" of the tubal cartilage. The mucosal layers of elastic tissue were thicker and more dense in the lateral, or membranous wall of the tube than in the medial, or cartilaginous wall, while the reverse was the case with the submucosal elastic tissue. Guild stated "There is evidence to indicate that the rapid closure of the tube which normally follows any opening of its lumen during swallowing or yawning, may be a rebound action of the elastic tissue of the 'cap' of the cartilage and of the other abundant elastic tissue of the region, which has been stretched by the muscular contraction that opened the tube." Hence closure of the tube and maintenance of its closure are probably also the results of an active force.

The normal Eustachian tube opens momentarily during the act of swallowing or yawning, as the result of the action of two muscles, the tensor veli palatini and the levator veli palatini.

ETIOLOGY

Bezold[6] ascribed the cause of the continuously open Eustachian tube to emaciation with loss of the normal supporting pad of fat around the Eustachian tube. He stated that this occurred in advanced tuberculosis and other serious chronic diseases.

I have encountered six cases of abnormally open Eustachian tubes in young pregnant females who were otherwise in good health Pregnancy has been the most frequent single known cause or associated condition in my experience with 31 patients Fat metabolism is apparently increased during pregnancy Davis[7] states that "alterations in fat metabolism as the result of pregnancy are suggested by high plasma lipid values and by the ease with which ketonuria and elevated ketonuria occur" He states further that "the hyperlipemia of pregnancy consists mostly of the neutral fat fraction, which is nearly doubled"

Contraction of scar tissue in the fossa of Rosenmuller was thought to be the etiologic factor in some of the cases reported by Pitman,[8] Shambaugh,[9] and Simonton[10] Pitman believed that hypertrophied tonsils could exert pressure upward on the cartilage of the tube to cause the pharyngeal orifice to remain open

Shambaugh[9] and Simonton[10] noted atrophy of the mucosa of the nasopharynx after irradiation for carcinoma in one case each, and Shambaugh ascribed simple atrophy of the mucosa of the nose and nasopharynx as the cause in another patient

Perlman[1] reported three cases following retrogasserian neurectomy for trigeminal neuralgia He attributed this to injury to the motor root of the Vth cranial nerve with paralysis of the tensor veli palatini muscle Perlman was of the opinion that the tensor veli palatini muscle is a factor in keeping the Eustachian tube normally closed, and that we need to reexamine the concepts pertaining to the function of all muscles acting on the Eustachian tube

One of Simonton's[10] cases followed polio-encephalitis with facial paralysis on the side of the involved ear

In the majority of cases of abnormally open Eustachian tubes, the etiology is obscure, as it was in 20 of those in my series

SYMPTOMS

The symptoms of an abnormally patent Eustachian tube are produced by the free passage of air and sound through the open tube from the pharynx into the middle ear

The majority of patients complain of a roaring or blowing sound in the ear, synchronous with their breathing The blowing sound is louder on expiration through the nose This is their most disturbing symptom The patient's own voice echoes or reverberates in his ear

These symptoms are usually intermittent or recurrent but may be constant They are made worse by exercise Perlman[1] noted that the resistance of the Eustachian tube was reduced by as much as 50 per cent after exercise The symptoms are temporarily relieved by short forceful inspirations, by inflating the Eustachian tube, by holding the head down forward on the chest or between the knees, and by lying prone Complete relief of the symptoms usually accompanies a cold The hearing is not impaired as a result of an abnormally open Eustachian tube

DIAGNOSIS

The diagnosis is usually suggested by the history The patient may mislead the examiner temporarily by stating that his ear feels "stopped-up", however, the normal appearance of the drum, its unusually good mobility, and the absence of hearing loss should soon dispel the possibility of an obstructed Eustachian tube The important question to be asked is, "Do you hear the sound of your own breathing in your ear?"

The diagnosis can easily be confirmed by listening with a diagnostic tube in the patient's ear When the Eustachian tube is open, the patient's breath sounds will be heard as an intermittent blowing or hollow roaring sound, similar to that produced by blowing across the mouth of an empty bottle His voice may be heard through the tube as a rough hollow sound

The drum will be found to be unusually mobile on massage with the pneumatic otoscope Hartmann[11] (1879) was the first to report observing a few patients in whom he could see the drum moving in and out, synchronous with respiration This phenomenon has been observed in a few instances by others when the drum was thin and atrophic, or was the site of a thinly healed perforation

Further evidence of abnormal patency may be obtained by measuring the quantitative resistance of the Eustachian tube with the aid of a sphygmomanometer Politzer's[12] method was to observe for outward movement of the drum while the patient practiced the Valsalva maneuver against a nasal tip attached to the mercury manometer, having an assistant note the level of the mercury column at the instant the drum moved outward Perlman modified this test by listening through a diagnostic tube held in the patient's ear, using a low-pitched "lub" sound as the end-point When the tube was abnormally open, the "lub" sound was heard at pressures varying from 2 to 12 mm of mercury He concluded that when the pressure required to overcome the resistance of the tube is less than 20 mm of mercury, the tube can be considered to be abnormally patent

Although the patient is not aware of any hearing loss, the audiogram may show a slight loss for low tones up through 500 cps, because of the masking effect of his breath sounds passing through the open Eustachian tube

TREATMENT

Non-surgical

Various methods of treatment for the abnormally open Eustachian tube have been tried since Jago's time (1867), but most of them were abandoned as unsuccessful These have been discussed in an interesting article by Moore and Miller[13]

The one procedure that has met with the widest acceptance and success is that advocated by Bezold and Siebenmann,[14] consisting of the repeated insufflation of a mixture of salicylic acid and boric acid powder (1 4) through a Eustachian catheter This has been used by Shambaugh,[9] Perlman,[1] Moore and Miller,[13] and by Simonton [10] I have used this same method of treatment with varying degrees of success There have been no reports of stenosis of the Eustachian tube following this treatment

Shambaugh[9] advocated irrigation of the nose and nasopharynx with normal saline solution for patients with atrophic mucosa

Those patients whose abnormal tubal patency is secondary to weight loss should, of course, be encouraged to regain weight to restore the fatty layer around the tube

The cases of open Eustachian tube occurring during pregnancy usually clear up spontaneously after termination of the pregnancy Only one of the six such cases I encountered failed to do so

Surgical

Adhesions in the fossa of Rosenmuller have been divided surgically by Moore and Miller[13] and by Simonton[10] with no lasting benefit

Zollner[2] infiltrated paraffin around the Eustachian orifice

Simonton[10] attempted unsuccessfully to reduce the size of the Eustachian orifice by various plastic procedures Two of his patients developed complete stenosis of the tube with resultant serous otitis media He concluded that these plastic procedures had no merit and should be abandoned

Polyethylene tubes have, of course, been used frequently in the treatment of chronic serous otitis media since their use was advocated by Armstrong [15] I used an indwelling polyethylene tube in the one persistent case of continuously open Eustachian tube following pregnancy with complete relief of symptoms and recommend it as a simple method of treating this troublesome condition, particularly if it fails to respond to insufflations of salicylic-boric acid powder The indwelling tube serves as a "pass-valve" for the expiratory air currents and also eliminates the autophony

DISCUSSION OF CASES.

I have encountered a total of 31 patients with symptoms compatible with a diagnosis of abnormally open Eustachian tube since 1952, a period of seven years. Most of these patients had symptoms intermittently, rather than continuously, but all complained of "hearing their breathing" in one or both ears at one time or another. Twenty-four patients had unilateral involvement, while the symptoms were bilateral in seven.

The breath sounds could be heard distinctly through the diagnostic tube at the time the patient was seen in the office in only ten of the total.

This condition seems to occur more frequently in females, there being 21 females and ten males in this group.

The audiogram revealed a slight loss for low tones in six young patients with normal hearing (masking effect of breath sounds).

The etiology was not evident in 20 patients. Five patients had lost weight, two of these having chronic heart disease and one having active pulmonary tuberculosis. Peculiarly enough, six of the cases of open Eustachian tube occurred in pregnant women, the onset of symptoms being in the first trimester in two, in the second trimester, in three, in the third trimester, in one. I have not seen any other reference to pregnancy as a possible etiologic factor in the literature.

The salicylic-boric acid powder (1:4) tubal insufflation was used on nine patients, with relief of symptoms for periods varying from 24 hours to eight months in those on whom follow-up observations were available. The treatment was usually repeated at one- or two-week intervals as long as the symptoms persisted. Because their symptoms are so distressing, these patients are very faithful in returning for appointments. The maximum number of insufflations in any one patient was seven. No incidence of stenosis of the tube was experienced.

One patient was treated successfully with an indwelling polyethylene tube after two insufflations of salicylic-boric acid powder failed to relieve symptoms

CONCLUSIONS

Abnormal patency of the Eustachian tube apparently occurs more frequently than is generally recognized A total of 31 patients with symptoms of this clinical entity was seen by the author over a period of seven years

It apparently occurs more frequently in females than in males

Although the etiology is obscure in the majority of patients, loss of weight, adhesions in the fossa of Rosenmuller, and mucosal atrophy following irradiation have been mentioned by others as causative factors

Pregnancy may also be an etiologic factor as a result of loss of fatty tissue around the tube due to increased fat metabolism

The insufflation of salicylic-boric acid powder is a safe method of treatment

The use of an indwelling polyethylene tube is advocated as a simple method of relieving symptoms, particularly in those patients who do not respond to insufflation of salicylic-boric acid powder

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DISCUSSION OF CASES

I have encountered a total of 31 patients with symptoms compatible with a diagnosis of abnormally open Eustachian tube since 1952, a period of seven years Most of these patients had symptoms intermittently, rather than continuously, but all complained of "hearing their breathing" in one or both ears at one time or another Twenty-four patients had unilateral involvement, while the symptoms were bilateral in seven

The breath sounds could be heard distinctly through the diagnostic tube at the time the patient was seen in the office in only ten of the total

This condition seems to occur more frequently in females, there being 21 females and ten males in this group

The audiogram revealed a slight loss for low tones in six young patients with normal hearing (masking effect of breath sounds)

The etiology was not evident in 20 patients Five patients had lost weight, two of these having chronic heart disease and one having active pulmonary tuberculosis Peculiarly enough, six of the cases of open Eustachian tube occurred in pregnant women, the onset of symptoms being in the first trimester in two, in the second trimester, in three, in the third trimester, in one I have not seen any other reference to pregnancy as a possible etiologic factor in the literature

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14 Medical Arts Square

THE MOUNT SINAI HOSPITAL POSTGRADUATE COURSE

In order not to conflict with the International Congress of Otolaryngology to take place in Paris in July, 1961, the annual intensive postgraduate course in Rhinoplasty, Reconstructive Surgery of the Nasal Septum and Otoplasty will be given January 7-20, 1961, by Dr. Irving B Goldman and staff at the Mount Sinai Hospital, New York, in affiliation with Columbia University

Candidates for the course should apply to Registrar for Postgraduate Medical Instruction, The Mount Sinai Hospital, Fifth Avenue and 100th Street, New York 29, N Y

CLINICAL STUDIES IN STEREOPHONIC HEARING*†

HARRIET L HASKINS, A M ,
and
WILLIAM G HARDY, Ph D ,

Baltimore, Md

Stereophonic hearing—literally the depth perception of sound—is an old concept Its application to various persons with problems of hearing, via modern eye-glass hearing aids and other ear-level instruments, definitely offers some interesting possibilities This discussion was not designed as a report of controlled investigations, but, rather, as a series of critical observations and comments on clinical findings that have been found interesting and instructive

A wide variety of different problems has been studied, and it is probably significant that the nature of the problem of the particular individual can rarely be generalized in terms of the precise benefits of stereophonic hearing Here, again, one is always working with the complex of a behaving person, not simply with the mechanics of an auditory system

The essence of this kind of inquiry is the fact that we were fortunate in having freely available a reasonable variety of aids in different models, which could be loaned to patients on a temporary basis Thus was made possible the accumulation of much more information than could have been obtained from a formal situation of clinical tests One must consider the patient's problems in terms of value received, for the use of binaural hearing, at best, is an expensive undertaking Our inquiry was the freer, in that the question of immediate possible financial burden was not pertinent

The types of problems studied may be described grossly in five categories first, *the complicated neural problems*,

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[*] Read at the meeting of the American Speech and Hearing Association New York N Y Nov 18 1959

[†] From the Johns Hopkins University Medical Institutions

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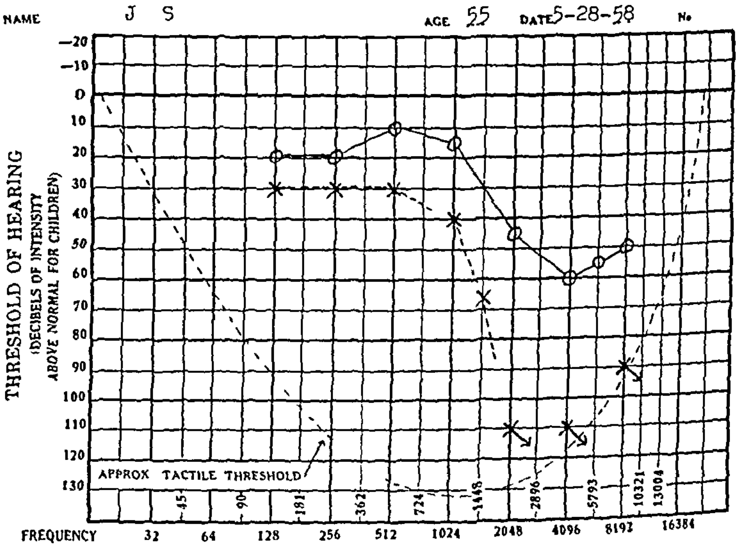
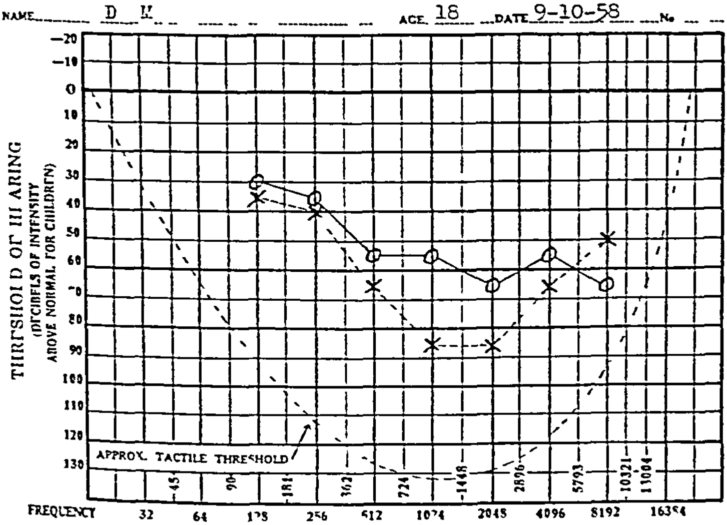
which had been studied with great care and found to have far better peripheral than central auditory function All these persons were most anxious to hear better, and had been consistently unsuccessful in the use of conventional monaural hearing aids More than half the group found it impossible to use binaural hearing Approximately one-fourth found the stereophonic effect helpful in controlled situations where the signal-to-noise ratio was not troublesome Second, problems which present an imbalance between the ears, with a mild loss on one side, and a moderate-to-severe impairment on the other In all these cases, amplification in the poorer ear which established a working balance between the two ears was effective (see Fig 1) In a few cases, where mild-gain



	UNAIDED		GLASSES—LEFT EAR	
S R T	Sound field	10 db	Binaural sound field	2 db
	Headphone right	10 db	Sound field, right	10 db
	Headphone left	40 db	Sound field, left	6 db
Disc	Sound field	100 %	Binaural sound field (In quiet and noise)	100 %
	Headphone right	96 %		
	Headphone left	92 %		

Fig 1

amplification in the better ear could be tolerated, the general effect was clearly improved by the use of appropriate amplification in each ear It is probably significant that most of these persons were seriously involved in the need to communicate in complex group situations—board meetings, problem-solving groups, and the like—and were highly motivated to achieve optimal results in difficult and important communicative situations Third, moderate-to-severe bilateral neural impairment, with fair-to-good discrimination Of this group all the persons studied have enjoyed binaural hearing after a reasonable period of adjustment and after an opportunity to learn and adapt in typical daily experiences Indeed, this is the group which is most critical in terms of the generally effective use of binaural aided hearing (see Fig 2)



	UNAIDED		GLASSES—RIGHT EAR	
S P T	Sound field	62 db	Binaural sound field	24 db
	Headphone right	60 db	Sound field, right	30 db
	Headphone left	68 db		
Disc	Sound field	88 %	Binaural sound field	90 %
	Headphone right	88 %	Sound field, right	84 %
	Headphone left	64 %		

Fig 2

By and large, routine clinical tests are not effective measures of the improvement of function, in fact, some refused to specify subjective better-ear hearing, and insisted that they were much more comfortable and satisfied with the use of both ears It is probably significant that these were persons who obviously improved in self-monitoring—they knew when their voices were well-projected and clear, and guided themselves accordingly It is worth reporting, that, without exception, the families of these persons reported significantly better communicative function in all respects. Fourth, those with moderate-to-severe, bilateral, mixed-type impairment All of these cases studied with binaural hearing, profited sufficiently to choose it They enjoyed the quality, and, like others who enjoy the effect, they report that events in sound are more natural, not so "thin," with "both sides of the head alive" In general, they localize better and hear with more assurance, and with obvious relief from communicative pressures In most instances, they perform better in the presence of background noise than they have ever been able to do with monaural hearing Finally, those with a moderate-to-severe, bilateral, conductive type impairment Because of the expense of binaural aids and the relative effectiveness of monaural hearing for their needs, the number of these persons studied is small Those who have purchased binaural aids, however, are so satisfied with the results of the stereophonic effect that they do not want to return to monaural aids Two of these persons have used binaural microphones with a monaural receiver, and have been pleased with the quasi-stereophonic results

It is worth emphasis, perhaps, that in some instances the test-variables, with and without binaural aids, were so slight that no quantifiable significance could be derived, in other instances, the variations between monaural and binaural hearing were astonishingly great In either extreme, there were no clear indicators of cause and effect It is clear that localization, *per se*, is by no means the factor of greatest importance, it is rather a matter of more information received and a sensation of awareness and security in listening which comes from the ready perception of depth-in-sound, which these persons report They are more relaxed, more assured, and more effective communicators These are important subjective relationships which do not readily lend themselves to formal testing and to statistical manipulations, we do not have adequate measures of these aspects of psychoacoustics

On the other side of the coin, there are some negative aspects of binaural hearing Some persons are troubled by the complexity of this auditory experience, although they may enjoy the sensation in a quiet situation The mechanics of proper adjustment are vitally important, and there are some who simply cannot master the necessary relationships and adjustments Third, for obvious reasons, many persons cannot afford the expense of binaural hearing

What we have learned thus far suggests that we must explore much further with children who have complicated problems of hearing Some of the experiences to date have proved fruitful, and should be pursued Children with dysacusis who use binaural aids have been observed to monitor their speech better, to be more alert to a variety of sounds of moderate intensity and to understand more speech and language Some children with a moderately severe hypoacusis have been able to function markedly better in general communicative situations, much like the adults of group three.

A few generalizations can be derived from these experiences *1* the psychic aspects of psychoacoustics become even more important, if possible, in working with stereophonic hearing than with monaural aids, *2* some of the traditional tools of audiologic measurement are not usually suitable indicators in making a judgment about performance, *3* the individual involved is his own best index—with some experience, he readily recognizes when and how the stereophonic effect is beneficial and expresses himself freely, *4* the selection of binaural hearing aids is a highly individual thing, and cannot be generalized as a matter of course from the evidence of a routine battery of tests, and *5* much work must be devoted to the development of better indicators of success in terms of stereophonic hearing, and especially of ability to handle informational material in the presence of competing messages As matters presently stand, it is imperative to give serious consideration to binaural hearing as a possible aid to every hard-of-hearing person's problem

POSTGRADUATE COURSE IN ALLERGY

A continuous course of two weeks' duration is being offered by the Departments of Allergy and Applied Immunology of the Temple University Medical Center and the Graduate School of Medicine of the University of Pennsylvania Sessions will be held daily at the Temple University Medical Center from 9 00 A M to 5 00 P M from February 2 to March 10, 1961 Tuition fee—$175 00 Enrollment limited Dr Louis Tuft is course director with Drs George Blumstein and Merle M Miller as associate directors

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The course is designed for physicians desirous of extending their knowledge of allergy It could serve as an introductory course for those about to enter the field or as a review course for practicing allergists

For brochure and application forms write to Dr George Blumstein, c/o Temple Medical Center, Philadelphia 40, Pa

PITFALLS IN TYMPANOPLASTY*

BRUCE PROCTOR, M.D.,

Detroit, Mich.

The purpose of this paper is to review critically the experiences and results of three years (1957-1959) in tympanoplasty. A total of 273 cases had adequate follow-up to form a basis for consideration. Many fine papers have been published on techniques and some on the results of this reconstructive surgery. Careful analysis of the results with particular emphasis on the causes of failures are as yet scantily reported. Such reports should soon be forthcoming in greater numbers once experience is gained in a large series of cases by a large number of otologists.

In a general way the following methods were used in the present series. Local anesthesia reinforced with preoperative drugs and intravenous applications during surgery was employed in nearly all cases. Exceptions were children and nervous adults. The predominant route was via an endaural incision which was extended to a Heerman's incision when necessary.

Tympanoplastic surgery must then be planned in a step by step manner. The following routine for this procedure has been followed by a number of surgeons.[3]

1. The mastoid and antrum control.
2. Perilabyrinthine control.
3. The ossicular chain control.
4. The epitympanum control.
5. The oval window control.
6. The round window control.

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*Presented at the annual Spring Otolaryngological Conference, The University of Michigan Medical Center, April 21, 1960.

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7. The tympanic sinus control
8. The mesotympanum control
9. The hypotympanum control
10. The tube control
11. The drum membrane control.

Eradication of infection is the primary objective in tympanoplasty Reconstructive surgery for restoration or preservation of hearing is of secondary importance Complete and thorough removal of all accessible diseased mastoid cells should be done if more than a simple meatoantrotomy is performed

The perilabyrinthine cells must be meticulously cleaned under microscopic control If there is caries of the labyrinthine capsule reconstructive surgery should be abandoned

Once the mastoid antrum and perilabyrinthine areas are controlled one proceeds to exploration of the middle ear The objective now will be to inspect the contents of the middle ear and to determine the nature and extent of the pathology The question then is, what is reversible and what is non-reversible pathology Upon this question hinges the definitive operative procedure to use

The posterior bony meatal wall can be removed until the bridge is flat and wide Before separating the drum from the bridge the outer layer of the drum remnant is carefully stripped off if one is to repair a perforation This maneuver is difficult once the tension of the drum is lost The drum is next dissected off from its posterior attachment Usually the incudo-stapedial articulation and both windows can be inspected If pathology (granulations, cholesteatoma, adhesions) is evident on the ossicular chain then it becomes necessary to remove a part or all of the bridge until all pathology is exposed If disease is evident in the attic then the upper bridge and antrum must be removed until the full extent of the pathology is uncovered

If the ossicles are covered by granulations or cholesteatoma, bound by adhesions or hyalin plaques or if they are carious then the incus, head and neck of the malleus are removed If the pathological mucosa is removed and the denuded ossicles are preserved they will probably be bound down by adhesions in the healing process Only occasionally can one employ interpositioning of ossicles or use prosthetic ossicle replacements to maintain a functioning ossicular chain and hope for some success

The next step is the control of the oval window If the chain is to be preserved, gentle palpation of the ossicles is carried out while watching for movements of the round window membrane If polypoid mucosa is present in the round window niche a small portion may have to be carefully removed before the round window membrane becomes evident If too much mucosa is removed a subsequent fibrosis may cause immobility of this membrane If the oval window niche contains excessive granulations, cholesteatoma or adhesions the chances for a normal functioning window are definitely diminished It is also inadvisable to place the drum or a graft onto a stapes surrounded by pathology Extreme caution must be exercised in removal of pathology from the windows lest the inner ear be opened into an infected field If only the footplate of the stapes remains and it is fixed it should not be mobilized as a primary feature unless the ear is free of suppuration The drum or graft should then be placed on the promontory and the footplate left open to the radical cavity Later, after the ear has healed completely and all suppuration controlled, one can resort to fenestration

After completing the inspection and manipulation at the windows, careful and thorough inspection of the sinus tympanicus is performed This pocket may extend posteriorly and medially to the descending portion of the facial nerve for a considerable distance Great care must be taken in removing the pathology completely from this region

In the mesotympanum granulations may be encountered on the promontory These may be carefully removed with sharp dissection If the cavum minor technique is to be used, the processus cochleaformis, the anterior spine and the pyramidal eminence are removed with curette or burr

Granulations in the hypotympanum are removed by sharp dissection taking precautions to preserve as much mucosa as possible so as to maintain an air column from the tube to the round window membrane If the mucosa is deficient it must be replaced with a mucosal graft

Tubal stenosis presents a major problem If a passage can be made through into the nasopharynx, an indwelling polyethylene tube can be inserted temporarily in an attempt to re-establish patency

TABLE I

Results in Tympanoplasty

Type	I	II-A	II-B	III-A	III-B	IV-A	IV-B	V	Total
No Cases	24	27	42	34	40	27	64	15	273
Cholesteatoma Found at Surgery	0	1	19	12	22	12	48	11	125
Perforations	8	12	3	14	7	16	8	5	73
Recurrent Cholesteatomas	1	4	2	5	0	2	2	2	18
Wet Ears	0	2	2	3	3	1	1	1	13

Whenever feasible pedicle and rotation flaps were used to bridge defects in the drum head or to seal off the round window niche from the external ear Most defects of the drum head were bridged with free full thickness skin grafts taken from behind the ear If mucosa in the tympanum was deficient, the defect was covered with a free mucosal graft taken from the lower lip

ANALYSIS OF RESULTS IN TYMPANOPLASTY

In Table I the 273 cases studied are tabulated as to the number of each type of tympanoplasty If most of the drum can be preserved the cases were classified in the "B" group If most of the drum had been destroyed by disease, the case was classified in the "A" group

Perforations developed in 73 (28 per cent) of the cases In the "A" group the perforation occurred in 42 of 88 cases

(47 per cent) In the "B" group the perforations occurred in only 18 of 146 cases (12 per cent) Some of these perforations were delayed and first appeared in the second and third year after tympanoplasty, apparently as a result of trophic changes Others developed as a result of the development of either a recurrent cholesteatoma or a graft cholesteatoma There were some that perforated because they did not adhere to the entire bed or were blown or pulled off

Recurrent cholesteatomas recurred or developed from the graft in 18 cases In 11 instances they developed in the 88 "A" grafts In four instances they developed in the 146 "B" graft cases It appears then that the larger grafts are more prone to develop recurrent or graft cholesteatomas and are more likely to perforate In seven of these 18 cases the cholesteatomas were successfully marsupialized as an office procedure Six were extensive in their recurrence and at operation were converted into radical cavities In three a new graft was placed onto the stapes, and in two the new graft was placed on the promontory.

Two revisions were performed because of persistent suppuration from the perilabyrinthine area—one was converted into a radical mastoidectomy, and the other had a graft replaced onto the promontory Four revisions were done because of perforations in the graft, in two a new graft was placed on the promontory, in one the new graft was placed on the stapes, and in the fourth, the ear was radicalized Finally, a revision was done because of a shift of the graft A new graft was placed onto the stapes Thus far a total of 25 revisions was performed

Cholesteatomas may appear as a late complication of tympanoplasty[1] Some develop from hair follicles which have been cut across in thinning down the free grafts At first these appear as small epithelial cysts in an otherwise intact graft If they are opened early and evacuated the graft will usually be preserved If they develop on the tympanic side the whole graft must be removed and replaced with a fresh one When only a small free graft is required the skin in the depth of the external meatus may be used as a free graft processus cochleaformis, the anterior spine and the pyramidal eminence are removed with curette or burr

Granulations in the hypotympanum are removed by sharp dissection taking precautions to preserve as much mucosa as possible so as to maintain an air column from the tube to the round window membrane If the mucosa is deficient it must be replaced with a mucosal graft

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In the present series, 60 per cent of the cases showed improvement of hearing In the II-B group less than half of the cases showed improvement in hearing This would seem to indicate that too often minor pathology about the ossicles was irreversible and the ossicular chain was not functioning properly

TABLE III

Hearing Results in Tympanoplasty

Type	I (24)	II-A (27)	II-B (42)	III-A (34)	III-B (40)	IV-A (27)	IV-B (64)	V (15)
Average Preoperative Hearing in db	30 9	38 1	35 0	46 2	43 0	50 5	49 0	55 9
Average Gain Postoperative	8 3	6 1	1 1	6 0	10 2	3 1	6 3	0 3
Average Postoperative Hearing	22 6	32 0	33 9	40 2	32 8	47 4	42 7	55 6
Reached 30 db Level	21	14	22	13	21	6	16	4
Reached 15 db Level	8	5	8	2	4	0	1	0
Air Bone Gap Closed 30 db or More	22	22	26	19	29	13	43	5
Air Bone Gap Closed 15 db or More	14	12	13	9	12	1	13	1

Hearing improved by 10 db or more in 37 3 per cent of all cases It remained in the range of plus 10 to minus 10 db in 45 4 per cent of all cases, and it was worsened in 15 per cent of cases by 10 db or more This worsening of hearing was again predominant in the "A" group

In Table III the average preoperative hearing is listed for the various types and beneath that is the average postoperative hearing Cases in the III-B group showed the highest average gain (10 2 db) The results are slightly better than in the II-B group and slightly worse than in the II-A group

It is thin and devoid of appendages which may be the cause of graft cholesteatomas In the present series of tympanoplasties such a graft was used on 12 occasions In none of these did a cholesteatoma develop One of these grafts, however, did not remain viable and had to be replaced The technique of ear drum implantation is extremely important in this surgery and is best described in a paper by Zollner[10]

Of the 273 tympanoplasties, cholesteatomas were found in 125 (45 per cent) at the initial surgery In the "A" group, 25 cases (28 per cent) had initial cholesteatomas In the "B" group, 89 cases (61 per cent) had cholesteatomas The significance of this difference in these two groups is that cholesteatomas tend to be limited by mucosal folds and ligaments to the attic and antrum, thus sparing the greater portion of the drum head

TABLE II

Results in Tympanoplasty

Type	I	II-A	II-B	III-A	III-B	IV-A	IV-B	V	Total
No Cases	24	27	42	34	40	27	64	15	273
Hearing Better	18	18	16	19	28	17	39	9	64 60%
Hearing Worse	5	8	18	9	7	10	20	6	83 30 4%
Hearing Better by 10 db or More	10	10	8	13	20	12	26	3	102 37 3%
Hearing at +10 to 10 db	14	12	24	12	16	9	29	8	124 45 4%
Hearing Worse by 10 db or More	0	4	7	7	3	7	9	4	41 15%

Of the 273 tympanoplasties only 13 had persistently wet and discharging ears, excluding, of course, the perforations and recurrent cholesteatomas This may attest to the value of complete exenteration of mastoid and perilabyrinthine air cells

The results of tympanoplasty have been recorded in various ways by different investigators If absolute hearing levels

15 db levels are also listed (see Table III). A total of 117 cases (42.9 per cent) reached the social level of hearing. Of 180 cases in the Types III, IV, and V tympanoplasty, 60 (33.3 per cent) reached the social level of hearing. This is good evidence of the value of tympanoplasty over the previously executed radical mastoidectomy which would have been done in these cases, furthermore, the average postoperative hearing in Types III, IV and V is about 10 db greater than the average hearing result following the classical radical mastoidectomy.

TABLE IV

Significance of Oval Window Pathology in Types III and IV tympanoplasty

Type	Window Pathology (Adhesions Granulations)	Fixed Stapes		Normal Windows		
		Not Mobilized	Mobilized		Graft Perforated	Graft Intact
III A	18 Cases −0.2 db	0	0	13 Cases +14.4 db	4 Cases +5.5 db	9 Cases +20.6 db
III B	11 Cases +4.6 db	5 Cases −1.0 db	0	20 Cases +17.8 db	4 Cases −0.5 db	16 Cases +22.4 db
IV A	13 Cases +1.8 db	3 Cases −0.6 db	3 Cases +7.6 db	8 Cases +7.5 db	4 Cases No Change	4 Cases +15.0 db
IV B	21 Cases −0.7 db	2 Cases +3.5 db	6 Cases +8.0 db	31 Cases +9.8 db	4 Cases +10.5 db	27 Cases +9.7 db

If the results are tabulated on the basis of air-bone gap closure (Table III), we get a much more favorable impression of this type of surgery. A total of 179 cases (65.5 per cent) reached a level of closure of the air-bone gap by 30 db. The air-bone gap was closed by 15 db in 75 cases (27.5 per cent). Many authors report their results on the basis of air-bone gap closure. The results in the present series approach the results reported by Wullstein[9] and others, when this method was used.

The significance of oval window pathology was examined in Types III and IV tympanoplasty (see Table IV). The results were revealing. If adhesions, granulations or cholesteatoma involved the oval window the results were uniformly poor in the 63 cases where this pathology was observed. If hearing is to be improved fenestration will probably be of benefit providing the labyrinth responds normally to the caloric test

If there are granulations, thickened mucosa, caries, adhesions or cholesteatoma involving the ossicular chain, it is probably better to remove the malleus and incus and put the drum head onto the stapes head, making sure that raw surface is presented on the inner surface of the drum head which is to be packed onto the raw surface of the stapes head

There was very little gain (1 1 db) in the average II-B case This poor showing would indicate that too often the ossicular chain was considered to have reversible pathology when it really did not, and healed in a rigid state, or else the pathology progressed

The long term hearing results in the Type IV cases are relatively poor My initial reports[6,7] on the functional results in tympanoplasty indicated better hearing but many of the cases slowly progressed to lose some of the initial gain The incidence of perforation of the graft was highest in the IV-A group (see Table I), and this probably accounts for the poor showing There appears to be another factor in this delayed hearing loss The oval window niche is open to the outer ear and is no longer protected by the drum head There is a thickening and drying of the epithelium in the niche which will tend to render the footplate less mobile

The poorest results were obtained in the Type V group Only 15 cases were done by this method In three cases the membranous labyrinth appeared pathological upon opening the horizontal canal, and in others pathology in the round window niche was extensive To achieve a successful fenestration (Type V) the following conditions must be fulfilled·

1 Good cochlear function *2* Normal caloric response, (hot and cold) as well as normal rotation tests *3* Previous formation of a small middle ear *4* Complete healing and epithelialization of the cavity, otherwise, fenestration may result in further loss of hearing Miodonski[5] fenestrated 12 cases of otitis but only three showed moderate hearing improvement He did fenestrate 14 cases of adhesive otitis media with fairly good results

The number of cases that reached the absolute 30 db and

15 db levels are also listed (see Table III). A total of 117 cases (42.9 per cent) reached the social level of hearing. Of 180 cases in the Types III, IV, and V tympanoplasty, 60 (33.3 per cent) reached the social level of hearing. This is good evidence of the value of tympanoplasty over the previously executed radical mastoidectomy which would have been done in these cases; furthermore, the average postoperative hearing in Types III, IV and V is about 10 db greater than the average hearing result following the classical radical mastoidectomy.

TABLE IV

Significance of Oval Window Pathology in Types III and IV tympanoplasty

Type	Window Pathology (Adhesions Granulations)	Fixed Stapes		Normal Windows		
		Not Mobilized	Mobilized		Graft Perforated	Graft Intact
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III B	11 Cases +4.6 db	5 Cases +1.0 db	0	20 Cases +17.8 db	4 Cases −0.5 db	16 Cases +22.4 db
IV A	13 Cases +1.8 db	3 Cases −0.6 db	3 Cases +7.6 db	8 Cases +7.5 db	4 Cases No Change	4 Cases +15.0 db
IV B	21 Cases −0.7 db	2 Cases +3.5 db	6 Cases +8.0 db	31 Cases +9.8 db	4 Cases +10.5 db	27 Cases +9.7 db

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The significance of oval window pathology was examined in Types III and IV tympanoplasty (see Table IV). The results were revealing. If adhesions, granulations or cholesteatoma involved the oval window the results were uniformly poor in the 63 cases where this pathology was observed. If hearing is to be improved fenestration will probably be of benefit providing the labyrinth responds normally to the caloric test and the round window membrane is functioning When the stapes was found fixed and was not mobilized the hearing status was not improved by tympanoplasty as would be expected These ten cases might again be improved by fenestration

In nine cases a fixed stapes was mobilized either initially or as a secondary procedure If done initially the ear must be absolutely free of suppuration for many months The nine cases showed a significant gain as a result of mobilization Removal of the footplate and replacement with a suitable graft may be done in the future after proper research in this type of situation

When the windows were normal the results were strikingly good in Types III and IV tympanoplasty and especially so when the graft did not perforate Nine cases of Type III-A gained on the average 20.6 db, 16 cases of Type III-B gained 22 4 db, four cases of Type IV-A gained 15 0 db and 27 cases of Type IV-B gained 9 7 db

DISCUSSION

Tympanoplastic surgery is still in its formative stage To obtain any degree of success and avoid the many pitfalls this surgery will require mastery of every aspect of otological surgery Preoperative clinical investigation will require careful otoscopic examination, complete audiometric assessment, sound probing and patch testing where feasible, tests for Eustachian tube patency and radiological examination of the temporal bone

Contraindications for this surgery must be sought for They include

1 Intracranial complications

2 Tubal infection of the middle ear in early childhood

3 Nasopharyngeal and sinus suppuration (these must be eradicated as a preliminary procedure)

4 Age over 60

5 Chronic infection in constitutional diseases—allergic states, tuberculosis, etc

6 Extensive osteitic changes in the labyrinthine capsule

7 Occluded Eustachian tube

8 Extensive mucosal deficiency in the tympanum

9 Poor bone conduction, *i e*, more than 30 db loss

Tympanoplastic surgery should not be unduly delayed Protracted inflammatory processes cause progressive destruction of normal tissues in the middle ear cleft The younger the patient and the more recent the infection, the better the results in reconstructive surgery

If hearing is good in the presence of chronic inflammations, do not explore the ear unnecessarily The results with the conservative radical mastoidectomy have in the past been very good If a conservative radical procedure turns out later to be disappointing a second stage operation can be done The two-stage operation should also be kept in mind when severe inflammatory changes are encountered and one cannot decide with certainty as to what is reversible and what is not reversible

The fact that this surgery is primarily done for control of infection should be impressed on the patient, furthermore, the patient should be told that hearing may be made worse as a result of the healing process The patient should not be led to expect a hearing gain, for if it is not forthcoming there will be a profound disappointment, even though progressive infection has been eliminated from that ear

We might emphasize at this point that the grafts must be handled carefully Once in place they should be carefully packed—not too tightly for fear of ischemia, or too loosely so that it moves from its intended position The packing on the graft should not be disturbed for two weeks Once the packing is removed the graft must not be touched with swabs or instruments for several months The blood supply is precarious for some time The patient must be warned against nose blowing and sneezing Inflations had better not be done for six to eight weeks for fear of detaching a portion of the graft An inflation to prevent adhesions will be just as apt to detach the periphery of the graft.

The mucosa on the walls of the middle ear and on the ossicles must not be touched or probed Suction tips must not be applied to normal mucosa Bone chips and blood clots must be removed completely and with care

Revision operations reported by Goto[3] in 65 cases showed that failures were due to edematous swelling of the mucosa around the ossicles (seven cases), granulations about the stapes (12), granulations in the niches, hypotympanum or sinus tympanicus (15), perilabyrinthine or mastoid infection (11), dry perforations of the graft (11), separation of stapes from the drum (3), postoperative labyrinthitis (2), oval window covered with the graft (2) adhesions ossicles (2)

Jongkees[4] reoperated 29 failures in a series of 200 tympanoplasties In eight of these cases the ossicles had been preserved In some cases the middle ear and attic had not been cleaned out enough, and in others the attic had been closed by a skin flap and granulations or cholesteatoma had formed again In one case the ossicles had been dislocated He concluded that it was unwise to leave diseased ossicles and that it seemed better not to close the epitympanum because leaving it open did not deteriorate hearing and it facilitated the cleaning of this important region If the attic is closed with a flap, cholesteatoma or granulations may grow undetected in the space thus formed

In revising nine cases with the graft or drum placed on the stapes, Jongkees found that in some, the stapes had been fractured He suggested that the stapes be cleaned from a posterior to anterior direction to avoid a fracture After cleaning the stapes the stapedius tendon should be cut In one case the stapes had been destroyed by granulation Poor results in this group were also due to non-adherence of too thick a graft to the stapes or to adherence of the graft to the promontory

In revising 11 cases with the graft or drum placed on the promontory, the oval window was found to be closed by tough connective tissue in three cases and in the remaining eight cases the graft had been placed over the footplate and either had not healed to the footplate, or thick scar tissue had filled the niche

Many of the cases, with hearing which became worse following tympanoplasty, had such hearing losses on the basis of a postoperative neural loss In the present series 30 cases in Types III, IV and V had 10 db or more of hearing loss as a result of this surgery In 18 of these the loss was predominantly of the neural type and in all of these excessive granulations had been removed from the windows In such instances, it is not unusual to find only a membranous oval window, and undoubtedly manipulations stirred up a postoperative serous labyrinthitis One must conclude then that the removal of granulations from the window niches may be a dangerous procedure It would perhaps be better to leave such granulations alone and let them regress spontaneously in the postoperation period If labyrinthine function is preserved some of these cases may later be fenestrated

Schwetz,[8] in a study of 156 tympanoplasty cases, reported that 52 showed a severe inner ear lesion postoperatively Twenty-two of these represented a marked postoperative hearing loss as a result of tympanoplasty

Temporary facial paresis occurred twice in this series of 273 tympanoplasties One cleared within a week and the second required six weeks Both cases were thought to be due to heat from the high speed burr Bukhman[2] reported paresis of the facial nerve in 27 of 233 tympanoplasties It was immediate in ten of his cases due to removal of granulations from the region of the horizontal portion of the fallopian canal (four cases), as a result of lowering the facial canal wall and piercing of the nerve sheath by small splinters or by hemorrhage into the canal In the delayed paresis (17 cases), Bukhman considered the following etiological factors perineural inflammation, diffusion of toxins through dehiscences in the canal wall, injury of vessels supplying the facial nerve, and lastly, hemorrhage

CONCLUSIONS

1 Tympanoplasty requires a routine discipline to be fully *effective* Routine manipulations in the following regions and in the order given are advised *1* mastoid and antrum, *2* perilabyrinthine areas, *3* ossicular chain, *4* epitympanum, *5* oval window, *6* round window, *7* tympanic sinus, *8* mesotympanum, *9* hypotympanum, *10* Eustachian tube and *11* the drum membrane

2 All mastoid and perilabyrinthine cells should be removed unless an antrotomy only is done

3 If the bridge is to be preserved it should be as flat and as wide as possible

4 If the ossicles have a pathological mucosa it is advisable to remove the incus and the head of the malleus

5 Manipulations in the middle ear must be very gentle to avoid traumatizing normal mucosa

6 If the stapes is surrounded by pathological tissues it is better to place the drum head onto the promontory

7 Extreme caution must be exercised in removal of pathology from the window niches Serous labyrinthitis may develop and cause a neural loss in hearing

8 The larger the graft the higher the incidence of perforation and recurrent cholesteatoma

9 Cholesteatomata are confined to the attic and antrum, for long periods by the normal mucosal folds and ligaments in the middle ear

10 Hearing results are best reported on the basis of air-bone gap closure

11 Delayed hearing loss in Type IV tympanoplasty may in time be due to stiffening of the annular ligament after direct exposure to the external ear

12 If the footplate becomes fixed in the oval window, one can overcome this with fenestration, mobilization of the footplate, or even removal of the footplate and insertion of a graft over the oval window.

13 Excellent results in Types III and IV tympanoplasty were obtained when the window niches were normal and the grafts did not perforate

14 The patient should be made to understand that this surgery is performed in order to a control infection, b prevent further hearing loss, c attempt to obtain improved hearing by reconstructive techniques At the same time it should be emphasized that hearing is very often relatively unchanged and may even become worse (15 per cent)

15 Failures due to recurrent cholesteatoma, perforations of the graft and failures to obtain a satisfactory hearing gain may respond to exploration and repeat grafting

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1229 David Whitney Building

MINIATURE AIR DRILL FOR MICRO-TYMPANIC PROCEDURES *

ROBIN P MICHELSON, M D ,

Redwood City, Calif

The instruments and drills, particularly cutting instruments, available at the present time for micro-tympanic procedures, such as the stapes mobilization operation, have gone through an evolutionary period in which the size has been decreased to a point where their usefulness is greatly increased In devising the miniature drill, the author had this idea in mind The cutting tool is only eight-one-thousandths of an inch in diameter, and resembles a jeweler's pivot drill It is sheathed and supported almost to its tip by a No 25 gauge hypodermic needle tubing This, in turn, is supported by a still larger, or No 20 gauge hypodermic tubing, which in turn, fits into the tapered nose section of the turbine housing The supporting sleeve which slides into this section can be moved in and out so that the shank of the cutting head that is exposed, may be varied to suit the operator, also, the length of the entire drill can be varied somewhat by putting in longer or shorter cutters The cutters are made of stainless steel music wire which is readily available in most hardware stores The sharpening of the point was done under microscope, utilizing a fine high speed grinding wheel First two flats were ground on each side of the wire, and these were then ground at the tip to a point The point was then sharpened by "relieving" the oblique or slanting surfaces It was found that this tool could be used either as a drilling instrument or would cut sideways The tapered section of the turbine housing is approximately two inches long while the turbine housing containing the air motor is approximately seven-tenths of an inch long, and three-tenths of an inch in diameter The handle is three inches long and is set into the turbine housing

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*From the Department of Otolaryngology Division of Surgery Stanford University School of Medicine

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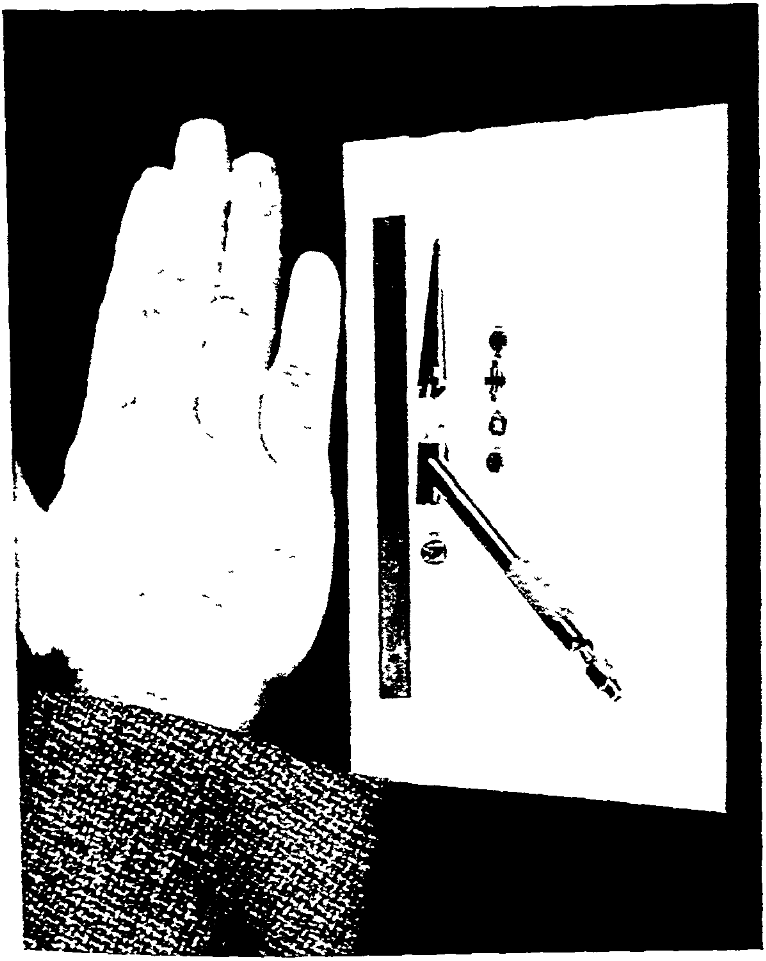

Fig 1 Instrument before assembly showing air motor parts

at a 45-degree angle, and is itself three inches long The air is fed in through a connection to the hypodermic needle hub at the rear of the handle, and the spent air is also exhausted out the rear of the handle

Speeds of the device can be varied from a point where the cutter is just barely turning over, to approximately 225,000 R P M at 40 pounds of pressure This upper speed limit is only estimated, and at present no tests of its speed have been

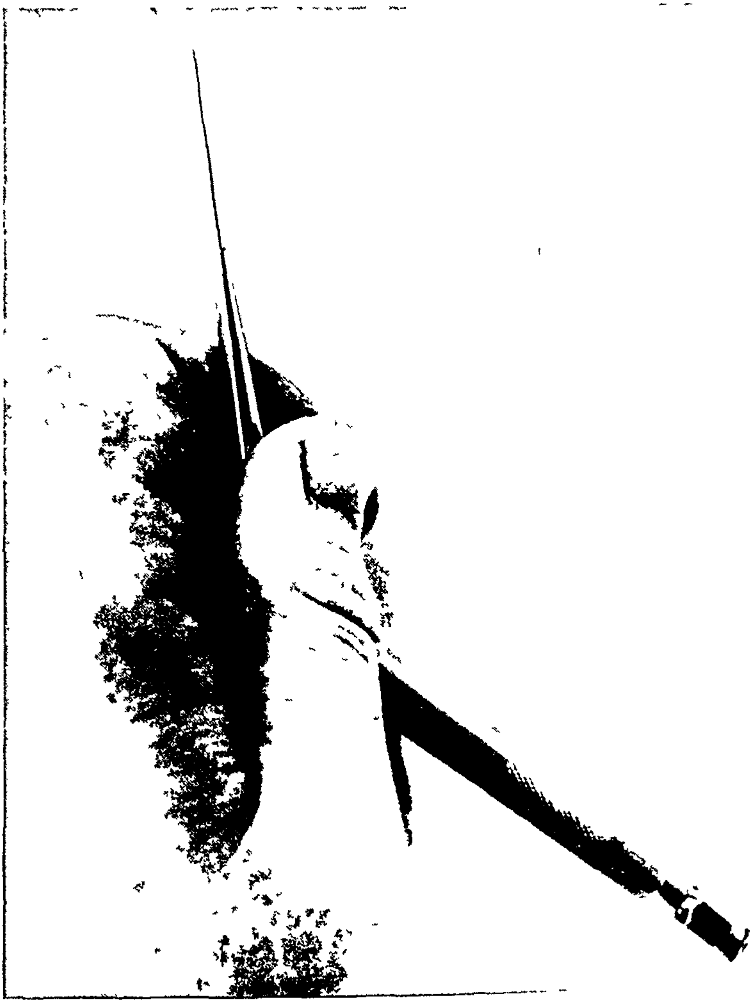

Fig 2 Another view of the assembled instrument

done Noise and vibration have been almost entirely eliminated, and one cannot tell whether or not the turbine is running unless it is actually held to the ear

The noise has been eliminated by the use of an axial flow turbine in which the air is used to push the blades In the usual Pelton wheel-type of turbine, the blades in rotation tend to chop the air-stream, thus a distinct whine or siren effect is produced This has been eliminated in this device, since the noise would definitely be objectionable in ear surgery

Sterilization may be accomplished by boiling in oil followed by a rinse in absolute alcohol and ether, followed by lubrication with a light special oil In practice, the author has soaked his instrument in absolute alcohol and then followed this up with ether, and then a few drops of oil applied to the ball bearings at either end of the turbine before putting the instrument into operation The miniature ball bearings in this instrument jam rather easily so that extreme care must be used in allowing particles of dust or dirt to enter the turbine housing while it is in use For this reason, a five micron filter and suitable control valve with gauge must be used

The drill may be stopped and started and its speed controlled with a small pressure control valve in the plastic air-line This can be incorporated into a foot-controlled switch or valve or the operator may hold it in his other hand The entire drill weighs one ounce

In two cases that he has operated upon, the author has found that it has been possible to cut a small groove around the footplate without disturbing the crura It has been necessary, however, to cut the stapedius tendon in order to accomplish this, thus, it is possible to remove the entire footplate without fracturing it or entering the labyrinth In cadaver material, it was found that it was possible to drill a hole through the neck of the stapes without disturbing it, or dislocating the footplate from the oval window

This drill is manufactured by the Storz Instrument Co, St Louis, Mo See their advertisement on page 5 of this issue

39 Birch Street

ABSTRACT OF THE PROCEEDINGS OF THE SCIENTIFIC SESSIONS EIGHTY-FIRST ANNUAL MEETING OF THE AMERICAN LARYNGOLOGICAL ASSOCIATION

Miami Beach, Fla, March 18-19, 1960

WM. J MCNALLY, M D.,
President

SAMUEL SALINGER, M D ,
Abstract Editor

PHYSIOLOGY OF OLFACTION AND GUSTATION

LLOYD M BEIDLER, M D , (By Invitation)

Receptors for the olfactory, visual and auditory senses are well protected, but since the gustatory sense must be accessible to ingested food, it is provided with another protective mechanism, namely the ability to regenerate taste buds following any trauma to the nerve mechanism The taste cells reproduce each other through mitotic division, also, the innervation via the chorda tympani and the glossopharyngeal protects taste function after injury to any one nerve bundle Dr Beidler demonstrated the results of electrophysical investigations in animals on the mechanism of taste reception These delineate the areas sensitive to various taste qualities While some fibers respond chiefly to one taste quality, all of the fibers are capable of responding to a lesser degree to the other qualities

The olfactory function differs greatly from the gustatory in that it can differentiate thousands of odors at concentrations as much as a million times more dilute than those normally used in gustation The olfactory receptors are bipolar neurons and not differentiated epithelial cells, and their dendritic processes number into the millions as against less than 1,000 taste receptors The functional significance of this large number of receptors is not known, but it may be related to odor quality discrimination

Dr Beidler described the experiments on animals, whereby the responses to stimulation by odors were recorded through an oscilloscope and a Sanborn recorder. It was shown that the trigeminal nerve participates in odor detection at various levels although to a different degree from the olfactory nerve

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ELECTRON MICROSCOPY OF THE OLFACTORY AND GUSTATORY PATHWAYS

A J DE LORENZO, Ph D , M D , (By Invitation)

Examination of the olfactory mucosa and the papillae foliata of the rabbit under electron microscopy reveals some marked differences in their receptor mechanism Since no definite structural variation could be demonstrated in the olfactory receptors, discrimination of odors can be attributed only to the varying reactions of the receptors to molecules of varying structure The fine structure of the receptor cells, their supporting cells and the arrangement of the ciliary processes are all beautifully seen through electron microscopy

Examination of the taste buds by this same method reveals receptor cells in various stages of development thus demonstrating the rapid turnover as new cells replace the old This suggests that new nerve fibers are constantly regenerating and that specificity of function is determined by the axon to cell contiguity at any particular time

DISCUSSION

Dr Arthur Proetz asked whether Dr Beidler could say a word about the nature of what he calls in his last slide an adaptation and which he (Proetz) used to denote as fractional fatigue, namely a specific and rapidly progressing insensitivity to certain odors, as for instance tobacco smoke in a room, which can be rapidly revived and which has no effect on certain contiguous odors, these remaining as sharp as ever Is this a matter of fatigue involving individual receptors among the millions, or is it a function of the turnover, or what is the explanation? This is a phenomenon not encountered anywhere else in the body with the possible exception of the eye The explanation of this phenomenon might lead to a great deal of valuable information as to the nature of the olfactory process

Dr Beidler, in reply, stated that adaptation is usually defined as the declining response under constant stimulation In the eye most of it is in the sense organ In olfaction, under constant stimulation, if recorded directly from the receptor, there is no adaptation at low concentration of odors, however, at high concentrations adaptation is complete On the other hand if you take the same odor, stimuli, same concentrations and study the olfactory bulb, you find the adaptation does occur even at low concentrations, so we have adaptation in two places one at the receptor at high concentrations immediately, and the other at all concentrations in the olfactory bulb, thus it would appear that adaptation is a property of all neural systems except possibly the peripheral nerve fiber itself

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CINEFLUOROGRAPHY IN THE PRE- AND POSTOPERATIVE MANAGEMENT OF LARYNGEAL CANCER

JOHN A KIRCHNER, M D, (By Invitation)

Dr Kirchner cited several cases which illustrated the value of cinefluorography in the diagnosis and management of carcinoma of the larynx. This procedure supplements routine examination and laminagrams by more effectively delineating the extent of masses and the mobility of muscles This may be very important in the decision between surgery and irradiation In the postoperative follow-up causes of dysphagia were determined as due to bunching of the pharyngeal muscles or to the formation of a false pocket as shown in cases cited

DISCUSSION

Dr Joseph Ogura has employed this procedure as an intermediary step between the indirect and the direct examination and considers it a valuable adjunct in diagnosis, as pointed out by the essayist The only objection to it is the time and cost involved

He has been asked what type of Dionosil he has used and replied that the oily solution is less likely to cause granuloma formation

Dr Corgill requested an opinion from Dr Kirchner in relation to a recent case of his where in the course of a very radical neck dissection, the left vagus nerve was removed Since then the patient has been regurgitating soft foods and fluids through the nose This would happen at intervals He would like to know whether this was due to malfunction of the cricopharyngeus and whether resection of this muscle might not relieve the condition

Dr Kirchner replied that in his opinion the condition was not due to spasm of the muscle and, therefore, a resection of it would not be the answer He has seen regurgitation in patients whose vagus nerve was not damaged and in cases where there was no stricture The picture is, therefore, very confusing, and he personally cannot supply a definite answer

THE ANATOMY AND PHYSIOLOGY OF THE LARYNX AND EAR OF THE BAT

Alvin Novick, M D , (By Invitation)

Manuscript Not Available

LARYNGEAL MANIFESTATIONS OF SYSTEMIC DISEASE

Walter H Maloney, M D

Dr Maloney called attention to several systemic disorders which result in laryngeal manifestations Hypo-metabolic states even in the presence of normal PBI may be the cause of hoarseness due to edema Thyroid replacement at the cell level through tri-iodothyronine has proven very effective

Granulomatous lesions should suggest not only T B but also various micotic conditions such as histoplasmosis, blastomycosis and actinomycosis Diagnosis may depend on a thorough history as to environment, working conditions, travel, etc

Collagen diseases such as rheumatoid arthritis, dermatomyositis, lupus, polyarteritis nodosa and scleroderma may be associated with histologic evidence of connective tissue changes in the larynx When the diagnosis is established proper steroid therapy should bring about a restoration to normal function The pitfalls of this form of therapy are pointed out along with the precautions that should be taken

DISCUSSION

Dr Francis W Davison likes to use the term "mucosal behavior" in describing laryngeal symptoms when a neoplasm has been ruled out, particularly in cases where systemic disorders are the cause He, also, favors the use of thyroid therapy when clinically indicated, despite the lack of confirming laboratory findings, although there are times when the patient should be referred to an endocrinologist

Dr Maloney acknowledged his indebtedness to Dr Davison, when he worked with him, for calling his attention to these problems He emphasized the point that we laryngologists should know more about the action of the steroids and their application in suitable cases without having to consult with an endocrinologist on every occasion

CLINICAL TESTING VERSUS ELECTROMYOGRAPHY

DAVID W BREWER, M D,
F FAABORG ANDERSON, M D,
and
F BERTRAM BRIESS

Faaborg Anderson's method of obtaining electromyographic determinations of all of the intrinsic muscles of the larynx was thoroughly tested out by Brewer and Briess The technique of the application of the electrodes into the individual muscles and readings obtained at rest and under various states of vocalization, resulted in accurate determinations of the activity of the muscles and deviations from the normal whether hypo- or hyperfunction

This procedure is very valuable in substantiating the rationale of voice therapy and explains the results obtained, thus a new diagnostic method is added to the armamentarium of the laryngologist and the voice therapist The laryngologist determines the status of the vocal apparatus by the history and examination, the faults are demonstrated by the therapist in his clinical voice testing, who then directs the therapy Their results are confirmed by the electromyographic readings

Dr Faaborg Anderson demonstrated the recordings at various levels showing the close relationship between the clinical voice testing and the electromyographic activity of the muscles This is dramatically revealed when applied to cases where disturbances in speech production and phonation were tested prior to and subsequent to therapy thus substantiating the value of electromyography as an adjunct in the diagnosis and therapy

Mr Briess whose work in the field of voice therapy has been previously reported, supplemented the demonstration by pointing out the close relationship between the two methods as illustrated in the slides and recordings of typical cases His diagnosis of hypo- or hyperfunction which he could determine as originating in this or that muscle or group of muscles could be confirmed by this new objective method, the two being parallel to a remarkable degree

DISCUSSION

Dr Joseph P Atkins expressed his enthusiasm over the procedures described which he is convinced may be means of solving many problems relating to laryngeal paralyses Dr Brewer thanked Dr Atkins for his comment and called attention to Dr Faaborg Anderson s first publication which initiated this procedure and described various types of laryngeal paralysis, a very interesting piece of investigation

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THE USE OF REGIONAL FLAP IN HEAD AND NECK SURGERY

JOHN J CONLEY, M D

Regional flap for the immediate coverage of defects following surgery of the neck offers the great advantage of shortened convalescence as well as adequate protection for the major arteries

It is indicated in cases where the vitality of the bed is impaired through prior irradiation, in cases where the malignancy has involved the covering tissues as well as the mucosal surfaces and in instances where the skin is used for the formation of the cervical esophagus

The flaps are derived from the anterior chest, the nape of the neck or the scalp depending upon the location of the neck defect and the sex of the patient

Large flaps are raised immediately following the definitive surgery and sutured into the defect The donor area is covered with a thick split free skin graft Dr Conley showed a number of slides illustrating a variety of flaps tailored to the particular needs of the individual case

DISCUSSION

Dr John M Converse pointed out the advantages of the procedures described by Dr Conley It obviates the necessity for preliminary planning and preparation of flaps which may not fit exactly the needs of the particular case when the resection has been completed What Dr Conley failed to emphasize was the fact that these flaps must be handled with great care and delicacy which is something acquired only after long experience Since the flaps must receive some blood supply from the recipient area as soon as possible it is important that the coaptation be perfect and without tension

Dr Julius McCall mentioned his experience in four cases where a spontaneous rupture of the carotid artery complicated surgery after extensive irradiation As a result he now follows Dr Conley's suggestions and plans a prompt coverage with an adjacent flap in every case where irradiation has preceded neck surgery

Dr Conley recalled having heard Dr McCall mention the disastrous hemorrhage cases last year and is pleased to note that he has adopted the procedure described in this presentation

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POSTERIOR CHOANAL ATRESIA

DANIEL C BAKER, M D ,
JULES G WALTNER, M D , (By Invitation),
and
WILLIAM NOVICK, M D , (By Invitation)

Fifteen cases of choanal atresia are reported, of which five were bilateral Ten were bony and five were membranous The youngest patient operated upon was 22 months In two cases the bilateral obstruction was membranous and was operated upon via the intranasal route

The best results were obtained through the transpalatine approach To prevent postoperative closure a piece of cleared

X-ray film was introduced in a vertical plane extending from the tip of the nose to the choana This was found to be more satisfactory than a polyethylene tube

DISCUSSION

Dr Lyman G Richards suggested that in adults having a unilateral atresia it is possible to avoid the tedious and disagreeable palatal approach by removing a generous portion of the septum so as to permit drainage from the obstructed side to find its way into the opposite side This will relieve the constant accumulation of mucous in the obstructed side and permit breathing through both nares via the septal opening into the unobstructed side

Dr Arthur J Cracoraner called attention to the necessity of preserving the flap to cover the denuded areas and to make the bony opening large enough by removing as much of the vomer as possible, also his incision is not so close to the teeth as shown, because it is extremely difficult to push a large palatal flap backwards in a small child

He has used the polyethylene tube not so much with idea of maintaining patency as for the purpose of keeping the flap in place

Dr Herbert H Harris recalled Dr Atkin's mentioning that these children do not breathe well or eat well for about five months What does he do about it? Dr Harris stated that he does a tracheotomy early and postpones the correction of the atresia until the age of two He is convinced that the palatal approach is the best, and he uses a dental burr to thin down the lateral wall and take off the posterior end of the septum In adults he has done these via the transeptal route much as described by Dr Richards Occasionally one has to punch out subsequent scar tissue formation

Dr Shirley Baron has operated upon a patient at only four months and is not entirely convinced that a tracheotomy is necessary He called attention to ballooning the palate as a means of diagnosing bilateral atresia in a child This is seen when the tongue is depressed so that the child cannot exhale

Dr Henry L Williams after hearing Dr Harris' remarks concerning the need for doing early tracheotomy was reminded of a paper written by Dr Gordon New about 40 years ago in which he suggested putting a plastic tube into the mouth and through the palatal opening and fixing it This allows the child to breathe and permits delaying the operation until the child's condition improves

Dr Baker closed the discussion by first commenting on Dr Richard's statement He feels that the palatal approach is easier and more direct and does not involve any more surgery than the transnasal approach As for Dr Harris' comment it is possible that he (Harris) misunderstood him What he said was that the newborn does not learn to breathe through the mouth until five months of age

As for tracheotomy, it is necessary in some cases but not all A membranous atresia can be perforated easily and a tube inserted so the child can breathe until he learns to breathe through his mouth Then definitive surgery can be carried out

He had not heard of the ballooning test but would be on the lookout for it, also he had not heard of New's operation but was very much interested to know about it

FINDINGS REFERABLE TO THE UPPER PART OF THE RESPIRATORY TRACT IN WEGENER'S GRANULOMATOSIS

HENRY A BROWN, M D ,
and
LEWIS B WOOLNER, M D , (By Invitation)

Drs Brown and Woolner gave the histories of six cases of Wegener's granulomatosis with the autopsy findings in four One patient, a girl of 14, survived The case was evidently a mild one

The authors agree with other observers that this syndrome and lethal midline granuloma are one and the same, even though in some instances the involvement of internal organs was not observed Analysis of most cases show that the process begins in the upper respiratory tract and later involves the internal organs such as the lungs, kidneys, liver and spleen The characteristic autopsy findings are those of a generalized periarteritis nodosa Failure to demonstrate this in the biopsies from the nose and throat may be due to incomplete search of the tissues throughout

In none of the cases cited was there any evidence of reticulum cell sarcoma which some observers seem to think is the real cause of the process

DISCUSSION

Dr Henry L Williams stated that he has always maintained that mid line granulomas and Wegener's granulomatosis were one and the same, being varied manifestations of a periarteritis nodosa The nasal lesions may be either primary or secondary and are frequently incited by local infection

Dr Joseph P Atkins has observed that the respiratory tract is not necessarily involved primarily, being secondary to a primary skin lesion

The histologic picture of biopsies from the respiratory tract is too often obscured by secondary infection so that the fundamental nature of the process is not readily determined Lesions from other parts of the body will show a clearer picture

Dr Atkins feels that since the true nature of periarteritis nodosa is not understood we had better retain the names of the various clinical manifestations until the pathogenesis of the disease is definitely determined

Dr Van Alyea asked the essayist why he stresses the importance of a history of previous upper respiratory infection when the incidence of this disease is so minimal considering the vast number of cases of respiratory infection He recalled an article by Dr Otto Saphir, a pathologist in Chicago, who found cases of periarteritis nodosa apparently resulting from the administration of sulfonamides Has Dr Brown ever noted such a connection in any of his cases?

Dr Brown replied to Dr Van Alyea s query by stating that no one can say why Wagener's granulomatosis should develop in so few of the thousands of respiratory infections, but the implication may be that the respiratory infection (and this may apply also to the taking of sulfa drugs) lowers the local resistance and favors the development of the more serious disease It is to be noted that more thorough histologic study of biopsies and post mortem specimens may bring to light the true nature of the disorder

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THE CEREBRAL CORTEX AND HEARING

LAMAR ROBERTS, M D , (By Invitation)

An attempt to evaluate the cerebral cortical function in relation to hearing was made through animal experiments as well as observation in the human The animal experiments in cats and monkeys consisted of evoked potentials, cortical stimulation and ablation, and in man by stimulation of the exposed cortex under local anesthesia and observations following diseases and excisions It appears that frequency discrimination is not found in the cortex but in the medial geniculum whereas sound localization and pattern discrimination are affected by cortical lesions which are more difficult than simple frequency discriminations Although some of the techniques used in the laboratory can be carried over to the human, further experiments on men are necessary to analyze auditory stimuli during cortical stimulation under local anesthesia Not only are the known auditory centers represented but it appears that the temporal and insular areas also participate in this function Theories of hearing must also take into consideration the types of defects seen in both central and peripheral lesions Further studies are necessary to delineate more accurately the relationship between the two

No discussion

SOME CLINICAL ASPECTS OF APHASIA

J R BROWN, M D, (By Invitation)

Dr Brown defined aphasia as a primary disorder of the reception, manipulation and expression of language "Primary" excludes defects secondary to deafness, motor loss of speech mechanism, intellectual defects and emotional disorders

It is generally agreed that the speech centers in right-handed persons is in the left hemisphere whereas in left-handed persons a small proportion may be in the right hemisphere.

As for location, the evidence points to the posterior portion of the temporal lobe, the Broca center and the junction of the parieto-occipital lobes In very few cases has the lesion been found in the frontal lobe or adjacent portion of the temporal lobe

The various levels of speaking are listed as follows the automatic, the socially reactive, the ability to utter the desired specific word, the ability to convey ideas accurately and discuss concrete matters and the ability to communicate abstract ideas clearly The more severe the aphasia the lower the level of speaking in the order described above

Basic defects in aphasia are blocks and distortions which the essayist describes in detail

Prognosis depends on the nature of the lesion A condition which is stabilized within three months may permit more definite forecasting as to future improvement and form the basis for corrective speech therapy

No discussion

THE PAROTID GLAND IN MIKULICZ'S DISEASE AND SJOGREN'S SYNDROME

WILLIAM G HEMENWAY, M D, (By Invitation)

Four cases of Mikulicz's disease and six cases of Sjogren's syndrome were presented Pathologically the involvement of the parotid gland appears to be the same in both conditions The difference between the two lies in the fact that the glandular involvement in Sjogren's syndrome is only one aspect of a constitutional affection which includes ocular, pharyngeal and arthritic lesions The syndrome is also found predominantly in females, whereas the Mikulicz disease is found in both sexes

The pathologic findings are destruction of the acini and proliferation of lymphoid cells which block the ducts Sialograms show a picture of sialectasis The term "chronic lymphoepithelial sialadenitis" seems preferable as a descriptive term in both conditions

No discussion

THE SPHENOIDAL SINUS IN ITS RELATIONSHIP TO THE SELLA TURCICA

GUNNAR HAMMAR, M D , (By Invitation)

Dr Hammar presented a study of the relationship of the sphenoid sinus to the sella turcica based on planagraphs in a series of 105 hypophysectomies and 120 consecutive postmortems These pictures indicate three types of sinuses, namely, conchal, sella and pre-sella The conchal type is small, does not reach into the body of the sphenoid and is separated from the sella by cancellous bone about 10 mm thick

In the pre-sellar type the cavity does not extend beyond a plane perpendicular to the sphenoidal plane through the tuberculum sella, a line easily located in the radiogram The anterior wall of the sella does not bulge into the cavity In the sellar type the anterior wall and floor of the sella bulge into the cavity from which it is separated by no more than 1 mm of bone This type is found in both sides in 59 per cent of the cases Mixed pre-sellar and sellar types occur in 27 per cent of cases Thus a sellar type is seen at least on one side in 86 per cent of all cases

Dr Hammar then described the variations in position of the intersinus septum and other septums that may be found.

All of this data is pertinent since hypophyseal surgery is being employed in so many cases of mammary carcinoma

Dr Hammar recommends planagraphs taken in both the frontal and axial planes

TRANS-ANTRO SPHENOIDAL HYPOPHYSECTOMY

CARL-AXEL HAMBERGER, M D, (By Invitation)

Dr Hamberger described in detail the technique of transantral hypophysectomy with appropriate illustrations He considers this approach the least dangerous, offering at the same time a wide exposure The operation is divided into two procedures first, the sinus operation, second, the hypophyseal operation The first stage comprises the sublabial exposure of the antrum, removal of its medial wall, exenteration of the ethmoids and wide opening of the sphenoids with removal of the intersinus septum

Dr Hamberger then described the approach to the hypophysis, showing the layers encountered, the venous blood supply which should be avoided, the capsule which is opened and the gland which is shelled out, severing it at the stalk Some cerebrospinal discharge follows for a short time but soon subsides The cavity is packed with muscle tissue and gauze which is left *in situ* for six to nine days

Hormonal therapy is instituted first by Cortisone dectate 100 mg I M, and later by mouth Postoperative polyuria is usually moderate

The author and his associates have done 150 of these operations There have been three cases of meningitis as a result

Definite statements as to the final results cannot be made at present since so much depends on the selection of cases

In young women where oophorectomy has been effective good results from hypophysectomy may be expected in about 80 per cent

In all women, where primary hypophysectomy is done, an objective remission will follow in about 30 per cent

The operation was done in 30 cases of diabetes with retinal changes All became insulin sensitive, and the tendency to acidosis disappeared Progression of the retinopathy ceased, however, operation must be performed before any severe proliferation of the vitreous has appeared

DISCUSSION

Dr Geoffrey Bateman acknowledged his indebtedness to Dr Hamberger for the privilege of seeing him do this operation He himself, has been doing a similar operation in which the nasal septum is resected first, however, the ethmoids are exposed through an orbital incision affording a direct approach to the sphenoid which is already located by the pack from the septal operation The nasal mucosa can readily be pushed out of the way thus affording enough space for the operating microscope which has proved very helpful in delineating the various structures

Dr W Likely Simpson agreed that both the transantral and the orbital approach can afford adequate exposure He would suggest infracting the posterior part of the septum for additional space

ARYTENOIDECTOMY IN CHILDREN

ROBERT E PRIEST, M D ,
HAROLD S ULVESTAD, M D ,
FRANK VAN DE WATER, M D ,
and
ROBERT J RICHARDSON, M D , (By Invitation)

Three cases of bilateral abductor paralysis of the vocal cords in children were presented Two of these were due to poliomyelitis and the third from unknown cause The ages varied from two years to 16 years In all three cases the patients had worn tracheotomy tubes for some time and were able to discard them after the arytenoidectomy The technique employed was a modification of Woodman's

DISCUSSION

Dr Kenneth C Johnston would prefer to postpone surgery in these children at least until they had reached the age of five or six They have seen about 40 to 50 cases of which only 12 were bilateral Those occurring in newborn or seen in early infancy were subjected only to tracheotomy Of the unilateral cases, most of them had sufficient motility to permit adequate ventilation In two of these cases tracheotomy was necessary

Dr Johnston pointed out that bilateral paralysis in the newborn frequently disappears In two cases tracheotomy was performed, and subsequently it was seen that the cords had regained complete mobility

Dr Edgar P Cardwell reported a case of a young man with bilateral paralysis, both cords in the cadaver position, the result of polio Later he developed insufficiency, became dyspneic and required a tracheotomy Both cords were then in complete adduction It would appear that the patient recovered the use of his adductors but not the abductors The question now is, will the condition improve without an arytenoidectomy?

Dr Edwin P Broyles raised the question as to whether bilateral paralysis due to polio is the result of vagus involvement or only the recurrent laryngeal The question is important, because the role of the superior laryngeal nerve must be taken into consideration

Dr Joel J Pressman has found the operation readily performed in children because their arytenoids are proportionately larger than those of the adult

Dr Priest closed the discussion He did mention the case of a little girl with a bilateral paralysis who was operated upon but in whom subsequent laryngoscopy failed to show any regeneration As to Dr Cardwell's question one would have to wait quite a long time before it would be apparent that recovery of the abductors would or would not take place

Regarding the question whether the entire vagus was involved or just the recurrent laryngeal, he was unable to answer since these were not his cases, however, he believed the entire vagus was involved, at least on one side

Dr Pressman's comment was very interesting and may well explain the good results obtained

EARLY INVASION OF THE THYROID GLAND IN CARCINOMA OF THE GLOTTIS

RICARDO TAPIA ACUNA, M D

Attention is directed to the possibility of aberrant thyroid tissue in the interior of the larynx as observed by Dr Acuna in at least two cases The thyroid gland may become involved in carcinoma of the glottis without much gross evidence of disease, however, it has been found that in these cases there is an abnormal enlargement of the thyroid cartilage In such cases it is advisable to do a laryngectomy and at the same time a partial thyroidectomy A paramedian incision is recommended with lateral flaps affording good exposure for the neck dissection The details of the technique were given in full

DISCUSSION

Dr Leroy A Schall was happy to see that Dr Tapia Acuna was doing a wide field operation, something which he, himself had advocated for many years

Dr Herbert H Harris during the past eight years has been removing at least one lobe of the thyroid particularly in low pyriform sinus lesions and certain glottic lesions He had not seen a metastasis to the thyroid until last year when a case of a cordal lesion treated by irradiation developed a rapidly growing carcinoma of the thyroid

He feels that resection of the thyroid in most cases can do no harm

THE ROLE OF RADIATION THERAPY IN CARCINOMA OF THE LARYNX

JOSEPH L GOLDMAN, M D ,
and
SIDNEY M SILVERSTONE, M D

An evaluation of the results of radiation therapy in 312 cases observed at Mt Sinai hospital during a period of 35 years brings out some interesting conclusions Carcinomas limited to the vocal cords and suitable for partial laryngectomy were amenable to radiation therapy yielding 88 per cent five-year cures Similar cases suitable for total laryngectomy when so treated resulted in 63 per cent survival A 56 per cent survival rate was obtained in the anterior group of the extrinsic larynx A 32 per cent survival rate was obtained in cases of the postero-lateral group and 14 per cent in the extensive involvement of the external larynx

These statistics are compared with those from surgery alone The techniques and dosage of the radiation therapy employed were given along with recital of such factors as modalities and postradiation edema Improvement has been noted since adopting cobalt 60 teletherapy

Combined radiation therapy and surgery has not proven successful

Dr John Conley wondered why Dr Goldman failed to compare his statistics with those of other irradiation specialists as Lederman of London, McLess of the Curie Institute, Delgado of U S, Martin of Memorial and Martin of Texas Their average survival rates in hypopharynx cases average from 6 to 12 per cent

Surgery after massive irradiation unless performed early yields poor results because of damage to the circulation, vice versa the same applies to irradiation following surgery

Dr Conley called attention to blood analysis indicating a high percentage of cancer cells in pheripheral veins in cases of cancer of the tongue He would suggest that Dr Goldman's pilot project be expanded to include the investigation of cancer cells in the blood The local findings may indicate a cure whereas the blood examination may show a canceremia

Dr F Johnson Putney has not been able to achieve the results by irradiation claimed by Dr Goldman The allover figures show far better results from surgery than irradiation although small superficial cord lesions are amenable to irradiation In reviewing statistics one should take the absolute figure rather than the determinate figure When we say that patients are dead of other causes we cannot be certain that they do not still have a cancer unless a complete autopsy is done In such cases the patient is dead so far as statistics are concerned and only the survivors should be counted Dr Putney agrees that irradiation is of some value in extensive lesions where surgery is contraindicated

The question of resectability of a tumor after irradiation poses a problem because post irradiation edema obscures the picture

Dr Jerome A Hilger stressed the importance of close cooperation between the therapist and the laryngologist This is occasionally lacking in some centers The point brought up by Dr Conley concerning canceremia points to the possibility of eradicating the spread by means of chemotherapy He approves of Dr Goldman's thesis that if the result is not favorable following irradiation, one should lose no time in proceeding to radical surgery

Dr Thomas C Galloway who for a long time harbored a prejudice against irradiation therapy, is now willing to admit it has value if used by the therapist in conjunction with the laryngologist He would also call attention to the fact that irradiation itself may cause cancer as evidenced by 121 cases of cancer of the thyroid and 36 cases of post cricoid cancer following irradiation therapy One must also take into consideration the age of the patient A young individual may develop the secondary carcinoma at a late period since the latency of post irradiation carcinoma may be from five to ten years

Dr Joel J Pressman has not been too impressed with the value of statistics, recalling an analysis by Negus of replies to a questionnaire wherein the results were classified with reference to the Broder's system

At that time this classification was considered important Today we hear less and less of it in connection with indications for therapy The number of frozen sections done in a given case will vary at different institutions, thus the indications and results will be different

Dr Pressman was impressed by Dr Conley's statement relating to the presence of cancer cells in the blood If this is true, why don't we find metastases in the liver more often? Much more information must be obtained concerning the dissemination of cancer cells in the circulation before we can assess its importance in the final outcome

Dr Goldman replying to the various comments of the discussors admitted that the subject was a big one with various ramifications Dr Conley asked why the statistics did not conform to those from other centers It is possible that those whom he cited did not have the same type of setup as prevailed at Dr Goldman's institution Certainly much of his results have been duplicated by Schall and Cunning and so reported by them The secret is in proper selection of cases and close continuous supervision by both the laryngologist and the roentgenologist

Dr Goldman called Dr Putney's attention to a report from Temple University some years ago in which intrinsic lesions suitable for laryngectomy yielded equally as well to radiation therapy

He agreed with Dr Conley and Dr Pressman that canceremia is a problem dealing with the reaction of the host and is a subject for further study One piece of work from Hungary demonstrated destruction of these cells in the lymph nodes His own studies have demonstrated cells in the tissue planes as well as in the nodes These areas can be sealed off by preliminary irradiation before surgery

As to statistics the actual true result is somewhere between the determinate survival rate and the absolute survival In his figures the patients called indeterminate, whom they eliminated, had as many as three or four years of cure, yet these cases were discarded from the reckoning He did agree with Dr Putney that for a basis of comparison between various centers a common base line should be agreed upon

He has never encountered a case of carcinoma caused by irradiation

In the next ten years they plan on treating all advanced lesions by combined therapy just as Dr Kramer did however, certain elderly individuals are too infirm to submit to more than palliative radiation

Concerning the use of Broder's Classification, their cases were studied and results analyzed by this standard, and they could find no relationship in the results as determined by Dr Harris and Dr Klemferer, their pathologist

EXTRAMEDULLARY PLASMOCYTOMA OF THE UPPER AIR PASSAGES

JULIUS W McCALL, M D

Five cases of plasmocytoma are reported, three involving the sinuses and adjacent areas, one the mandible; one, the larynx

Microscopically the cellular make-up of the tumors is identical with that of multiple myeloma It may be that the latter represents a progression of plasmocytoma

Since these tumors are of lymphatic origin they are radio sensitive and, therefore, irradiation therapy is to be employed except in cases where the tumor is pedunculated and can be readily excised *in toto*

In the author's series, two cases treated surgically are alive The larynx case survived seven years without recurrence until just recently

One case involving multiple bony and soft tissue lesions succumbed within two-and-a-half years after diagnosis

DISCUSSION

Dr Clyde A Heatley has found that the location of the tumor has much to do with the therapy and the prognosis In the pharynx and larynx they are usually pedunculated and favorable for excision, in the nose and the antrum they are more malignant and invasive Extensive resection is not advisable, because it frequently leads to systemic spread of the myeloma While the results of irradiation are not all that could be desired, it is still preferable to resection

Dr Robert E Priest mentioned a case which he has had under observation since 1946 This man has had plasma cell tumors in his nose, nasopharynx, oropharynx, hypopharynx and larynx which have been removed He still gets recurrences in his larynx necessitating removal of masses to help his breathing His tonsils were removed in 1936 and kept by the patient all these years Dr Heatley intends to have them sectioned, suspecting that they were plasma cell tumors which they resembled in the gross

Dr Edwin N Broyles stated that he had seen a case in Baltimore ten years ago very similar to the one shown in Dr McCall's movie It happened to be a bloody tumor in the trachea which was removed endoscopically The pathologic diagnosis was angiosarcoma The patient has remained well for the past ten years, and Dr Broyles is still suspicious that it was originally a plasmocytoma

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THE

LARYNGOSCOPE.

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REVIEW OF THE AVAILABLE LITERATURE ON THE LARYNX FOR 1959

FRANCIS E LEJEUNE, SR , M D ,
WARREN STASSI, M D ,
and
FRANCIS E LEJEUNE, JR , M D *

ANATOMY

Interest manifested in the laryngeal appendix by Broyles[1] was stimulated by the fact that on examining several larynges immediately after laryngectomy, he was impressed with the large size of the laryngeal appendix or saccule The sizes of 50 fresh larynges removed at necropsy were carefully studied Twice as many large saccules were found in white persons as were found in Negroes The male saccule, as would be expected, was larger than the female's The inside of the saccule is lined with ciliated columnar epithelium and one of its functions is thought to be provision of adequate mucus over the vocal cords for lubricating purposes

Pressman and associates[2] describe interesting experimental work on the larynx by injections of radio-isotopes They proved that injection of dye or istotopes into one side of the larynx extended only to the median line and did not cross over to the opposite side The lower border of extension was the inferior border of the cricoid This finding is significant because it proves that there is no free communication of lymphatics between the larynx and the trachea Isolation of one

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*From the Department of Otolaryngology Ochsner Clinic New Orleans

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Other good studies, however, have demonstrated cases of good esophageal speech despite absence of this structure

The esophagus forms the main air reservoir, and intrathoracic pressure transmitted into the esophagus provides the force to propel the air through the pseudoglottis and cause speech The direct inhalation method of obtaining air seems far superior to the swallowing method

Timcke, von Leden and Moore[6] conducted experimental studies on vibration of the vocal cords They concluded that the anatomic configuration of the vocal cords plays a significant role in the production of the vibratory pattern and that changes in air pressure, both static and dynamic, exert considerable influence on the vibratory cycle Periodic changes of the vibratory pattern occur during voice production, and laughter and other explosive phenomena require more forceful efforts on the part of the larynx

Sonesson[7] presents a preliminary report on a method for studying the vibratory movements of the vocal cords which has not been used previously The subglottic space is illuminated by means of a laryngodiaphanoscope lamp, and the light passing through the glottis is intercepted by a plexiglas rod introduced through the mouth The rod conducts the light to a multiplier phototube, whereupon the recording takes place by means of an oscilloscope As a result of vibratory movements of the vocal cords the flow of light through the glottis changes, and a sinusoidal trace is recorded The apparatus is called glottograph, and the traces obtained are called glottograms The work of standardizing the method and of collecting and processing more extensive material is in progress

Furstenberg[8] presents a highly scientific study giving experimental evidence that laryngeal movements can be obtained by stimulating regions of the cortex other than the primary motor areas These experiments, conducted on monkeys, are carefully described The conclusion reached indicates that laryngeal movements can be obtained from many areas of the cortex in addition to the primary motor field Some of the additional motor areas are in fields where the connections half of the larynx from the other, corresponds to the known behavior of many cancers of the larynx in which enormous tumors occupy one side of the larynx without involvement of the other Pressman and co-workers state that sufficient radio-isotopes remain within specific areas of the larynx to be considered of therapeutic value In reading this article one cannot fail to be impressed with the clinical implications of this study

Man's desire to simulate a human larynx is responsible for the interesting experimental work by Campbell and Murtagh[3] in which double reeds were used in conjunction with special tubing These authors believe that the actual subglottic shape of the thyroarytenoid muscle may be the primary determinant of frequency of vibration of the vocal cords

PHYSIOLOGY

Hilding[4] presents an interesting study on the ciliary beat in the larynx and trachea in which tracheas from freshly killed cows and calves were used His studies indicate that the plane in which the tracheal cilia beat is generally parallel with the long axis of the trachea The epithelium on the margin of the cords is squamous, whereas that in the interarytenoid region is ciliated Practically all mucus normally drained from the lower tract passes out between the arytenoids There is, however, a slowing down of the flow of the mucus on reaching the level of the vocal cords, particularly at the anterior portion, and occasionally, the blanket of mucus is retarded in the interarytenoid area Hilding wonders what relation, if any, this pattern of flow has to the distribution of disease in the respiratory tract Carcinoma occurs most frequently at the margin of the cords and in their anterior half, whereas tuberculous lesions occur characteristically in the interarytenoid area His theory and speculation are interesting to study

Thomas[5] carefully reviews the literature comprising our knowledge of production of esophageal speech These various studies present a fairly consistent picture of esophageal speech, crediting the cricopharyngeal fold as the pseudoglottis changes possibly occurring in the mast cell count per unit volume in certain regions of the larynx Mast cells were found in the larynx of all embryos having a crown-rump length of 5 4 cm and a 1 per cent increase in the length of the embryo was followed by a 3 per cent increase in mast cell count

Ruedi[12] presents a complete and enlightening thesis on the histology of the larynx, the types of mucosa and the part they play in the development of malignant disease He has been able to change the type of mucosa by trauma, drugs and medication

The second part of the thesis compares the clonic to the tonic theory of voice production Ruedi adheres to the tonic theory and is convincing in his arguments giving excellent reasons and illustrations

To be properly appreciated this article should be read and reread rather than summarized

PHOTOGRAPHY

Wieland[13] describes a method of improved photography of the larynx in which he uses an endoscope with a mirror reflex viewing device attached to a Robot camera More light is transmitted through improved optics The camera is used much in the same manner as indirect laryngoscopy Unfortunately, the photographic reproductions were blank, and the type of photography presented could not be evaluated

DIAGNOSIS

Laryngography is a useful, simple radiographic diagnostic procedure for evaluating abnormalities of the pharynx and larynx, according to Borden and Berger [14] After topical application of a local anesthetic, coating of the laryngeal mucosa is obtained by a smooth spread of 5 cc. of a radiopaque medium (Dionosil oily) dripped over the back of the tongue Four views of the larynx are taken immediately. The results obtained are thought to be diagnostically helpful

According to Golden,[15] roentgenography of the larynx should suggest that they have to do with emotional responses Furstenberg further states that in the course of normal behavior the additional motor areas of the larynx, like those of other types of movements, serve as centers for automatic associated movements They dress up and give personality, as it were, to the voluntary motor responses

Fuller, Fozzard and Wright[9] conducted radiographic experiments proving the sphincteric action of the cricopharyngeus muscle This was done by intranasal introduction of calibrated tubes to a point just below the cricopharyngeus and taking repeated roentgenograms

Laryngectomy, and the consequent respiration by means of the trachea, abolishes the vertical type of physiologic respiratory resistances that frequently exist at the level of the superior air passages Brunetti[10] presents his observations of the respiratory dynamics in laryngectomized patients He investigated the pneumotacograms of eight adult patients before and after laryngectomy After total laryngectomy the physiologic resistances of the upper respiratory tract from the larynx upward were eliminated Brunetti observed a constant increase in the flow rate during inspiration and expiration, an altered or diminished ratio between expiration and inspiration, and appearance of a post-inspiratory pause It was also observed that the shape of the pneumotacograms in every patient after laryngectomy had been modified, which indicates less economical function than found in normal persons

HISTOLOGY

Grahne[11] presents a good review of the literature and description of mast cells, their distribution, chemistry and function He concluded that a high cell count is a sign of high functional capacity of the connective tissue In his investigative work, which was conducted on 30 human embryos, the ventricular band, the vocal cords and the laryngeal ventricles were designated for intensive study Difficulty in staining the cells was encountered but overcome, however, specimens could not be obtained from all regions in the seven smallest embryos The purpose of this investigation was to study

This was found to be true by Gregg and Shreves,[18] who describe a penetrating wound of the larynx in which a stone fragment punctured the skin, cricothyroid membrane and lower portion of the thyroid cartilage on the right side, lodging in the airway Hemoptysis, mild subcutaneous emphysema and hoarseness were the only symptoms The patient subsequently coughed up the fragment, and a speedy and uneventful recovery followed Gregg and Shreves discuss the various complications of penetrating wounds and urge prompt action in such injuries

Holinger and Johnston[19] present a most informative and practical description of laryngeal trauma and its complications Various causes of laryngeal trauma are discussed, automobile accidents being responsible for the majority of multiple laryngeal injuries An abnormal or absent voice, or dyspnea, developing after injury is fairly indicative of some type of laryngeal trauma In all such cases examination, including palpation of the larynx, is indicated Therapy must be guided by the extent of the injury Immediate repair within a day or two after the injury is most desirable if fractured cartilage is to be replaced in normal position and the airway reestablished It is usually necessary to insert a splint in the lumen of the larynx to maintain fractured cartilages in the desired position, and emphasis is placed on low tracheotomy In multiple injuries in which there is possible fracture of the larynx and the dyspnea is relieved by tracheotomy, too frequently after this procedure the larynx is either forgotten or neglected while attention is directed to other injuries

Camps[20] relates an interesting case in which a man was murdered by a blow on the larynx with the side of the hand producing a vertical fracture of the thyroid cartilage The body was found hanging from a staircase, and the death was considered suicide Because of interesting developments the body was exhumed two years later and by conducting experiments it was proved that hanging could not have been the cause of death A vertical fracture of the larynx can be caused by a blow on the anterior portion of the larynx The assailant was found, tried and convicted be done by lateral stereoscopic projections and by body section films in the anteroposterior projection Planigrams should be taken with and without phonation Roentgenography may give information not obtainable otherwise, as in malignant cases with extension into the pyriform sinus or esophagus or even subglottic extension that would not show up in indirect laryngoscopy

Because he was dissatisfied with the usual techniques used for direct laryngoscopy Prohovnik[16] resorted to a Haslinger laryngoscope with the patient in the sitting position, employing the posterior approach as used originally in Europe Prohovnik reports superior results by having the patient sit on a low stool with head extended backwards The laryngologist stands behind the patient and after adequate cocainization, the laryngoscope is introduced and the larynx clearly visualized

FOREIGN BODY

Brandenburg[17] states that foreign bodies can find their way into the air passage of a person of any age A 30-year-old Mexican, during a drinking spree, apparently had become unconscious and upon regaining consciousness noted that his partial denture was missing and he was unable to talk On mirror laryngoscopy, firmly impacted in the laryngeal orifice could be seen the missing denture, which was removed without difficulty

INJURIES

Penetrating wounds of the larynx are commonly observed during war time, when many persons are exposed to fragments of high velocity missiles These types of injuries are associated with high mortality and morbidity rates because of alteration of respiratory function and the potential sequelae During peace time these injuries usually receive more prompt attention, and as a result, the survival rate should be higher and the morbidity rate should be lower than during war time velops with inspiration, closely resembling bilateral abductor paralysis Bowing of the vocal cords on inspiration differentiates crico-arytenoid arthritis from bilateral abductor paralysis

Acute laryngitis may be a serious condition in small children whose larynges are so narrow that moderate edema of the mucosa can close the airway According to Coffin,[24] the outcome of laryngitis in children is often unpredictable as the disease varies in severity from the adult type with mild hoarseness to the swiftly fatal forms, such as laryngotracheobronchitis and epiglottitis Stridor and dyspnea are warning signs, and the desirability of tracheotomy must be kept in mind Coffin reports several cases Corticoids apparently relieved dyspnea and partial respiratory obstruction in all of his patients In addition to antibiotics, oxygen and mist, prednisone or hydrocortisone parenterally was given to curtail edema and exudation and to open the airway Improvement was rapid, and the excellent outcome suggests the more frequent use of Cortisone derivatives when acute inflammatory edema threatens a child's larynx

Copeman, Elkin and Pearce[25] report a case of rheumatoid arthritis of the crico-arytenoid joint in a woman 38 years old Because of bilateral fixation of the cords in the median line tracheotomy became necessary In spite of all types of therapy the arthritis gradually involved all joints, and eventually the patient succumbed to bronchopneumonia At post-mortem examination the crico-arytenoid joints appeared to be fixed and immovable The histologic picture is well described and bears out the general opinion that in arthritis of this joint the arytenoid is fixed, whereas in bilateral abductor paralysis the arytenoids can be retracted or abducted on pressure

Polisar[26] presents an excellent discussion of rheumatoid arthritic involvement of the crico-arytenoid joint The anatomy and physiology of this diarthrodial joint are thoroughly reviewed The differential diagnosis of bilateral abductor paralysis and fixation of the crico-arytenoid joint is discussed, and Polisar correctly states that in paralysis the cords are flaccid and easily abducted passively, whereas in

DISEASES

Vocal rest is beneficial in cases of acute and chronic inflammation of the vocal cords, certain cases of specific infections such as tuberculosis, ulcerations, small tumors and postoperative laryngeal conditions Myerson[21] is of the opinion that vocal rest for practically all other conditions occurring in the larynx is of little or no good After deposit of fibroblasts and formation of fibrous tissue, little or no results will be obtained by vocal rest The presence of fibrous tissue on the vocal cords bespeaks permanency Myerson discusses contact ulcers and vocal nodules and thinks the latter is best removed by indirect laryngoscopy, although many will disagree with him He states that application of voice rest has not been too well defined in the past Comprehension of the tissue changes that occur before and after removal of vocal cord tumors will furnish a better understanding of application of this form of therapy

Peach and Zaiman[22] conducted a study of 122 cases of laryngotracheobronchitis in Toronto, where this condition is a common cause for hospitalization each Winter The largest number occurred in the age group of 12 to 23 months One-third of these required tracheotomy Clinical features are well discussed, and each child was treated in an atmosphere of 100 per cent humidity and given antibiotics freely That only one death occurred in their series attests to the efficacy of the therapy instituted

Arthritis of the crico-arytenoid joint is most commonly associated with generalized arthritis Montgomery[23] reports two such cases in which he was able to obtain postmortem serial sections of the crico-arytenoid joint showing arthritic changes He also discusses the usual symptoms and states that a positive sign of the disease is pain elicited by pressure against the anterior aspect of the thyroid cartilage or by compression of the superior cornua of the thyroid cartilage The acute stage of the disease is characterized by considerable hyperemia and swelling of the arytenoids, and manipulation of the arytenoids is painful In the chronic stage bilateral fixation in the position of adduction occurs, and stridor de-which contains 10 per cent urea peroxide, in the treatment of diseases of the larynx No specific form of laryngitis that would be benefited by use of this product is mentioned, and it is hard to conceive how it could be used except as a nebulizer

In a most excellent and instructive paper Davison[31] decribes six conditions responsible for acute laryngeal obstruction in children These are acute obstructive supraglottic laryngitis, supraglottic allergic edema, laryngeal diphtheria, subglottic allergic edema, acute laryngotracheobronchitis and foreign bodies Each of these conditions and its specific treatment is thoroughly discussed Davison stresses the fact that fatalities can be prevented if the correct diagnosis is made early and proper therapy is instituted In many of these cases tracheotomy must be considered, and it is wise not to procrastinate in the dyspneic patient Oxygen may be given as a temporary measure, but it is never a substitute for a needed tracheotomy

According to Blattner,[32] various forms of upper respiratory ailments, particularly croup, have been shown to be due to viruses Recently, a viral strain identified as Type II hemadsorption has been shown to be the etiologic agent in many cases of childhood croup In addition, this viral strain has been shown to be related to the Asian flu strain Blattner believes that confirmation of these observations will lead to inclusion of this strain in any vaccine against viruses of the upper respiratory tract

Bjorkenheim[33] describes two cases of laryngitis of pregnancy Both patients were completely aphonic In one, because of toxemia of pregnancy the patient was so dyspneic that she could not be fully recumbent The second patient had simple laryngitis of pregnancy In both patients the hoarseness disappeared soon after delivery Bjorkenheim contends that laryngitis of pregnancy is not basically an inflammatory disease even though an inflammatory type exudate may be present Careful management of delivery is essential since dyspnea may already be present and closure of the glottis in bearing down may be impossible fixation the vocal process and arytenoid eminence are immobile In reviewing numerous articles on this subject Polisar states that one cannot help but marvel at the clinical acumen of our forebears This is one of the most interesting articles on this subject that has come to the attention of the reviewers

Infection with Candida (Monilia) albicans has become a source of greater concern now than in the past According to Lees,[27] lesions due to it have become more in evidence since introduction of antibiotics Described as the most mutable and treacherous of fungous diseases, moniliasis is widely distributed throughout the world The broad spectrum antibiotics are known to upset the balance of the bacterial flora in the mouth and gastrointestinal tract. As a result oral, gastrointestinal and vaginal moniliasis are now being seen more frequently

Cracovaner[28] reminds us that hyperkeratosis of the larynx is an important and perplexing subject to the laryngologist It is a precancerous lesion and must be treated as a potentially malignant growth An excellent description of these lesions is presented Cracovaner treats small localized lesions by stripping the cords under direct laryngoscopy Lesions that recur on one vocal cord are removed by laryngofissure and lesions recurring on both cords are treated by radiation If this latter is unsuccessful, laryngectomy is performed Extensive involvement of the larynx by a hyperkeratotic lesion is treated by laryngectomy

Lupus erythematosus with involvement of the larynx is extremely rare Scarpelli and associates[29] report such a case in a 21-year-old Negro who had been treated for one year He was apparently progressing satisfactorily, when a sudden explosive onset of laryngeal edema developed and proved fatal Microscopic evidence in the larynx of fibrinoid degeneration of collagen and of hematoxylin bodies that were Feulgen positive was assumed to be diagnostic of lupus

Harkins[30] advocates use of an improved hydrogen peroxide, are applied in order to avoid pinching or crushing the recurrent nerve or one of its branches Eternal vigilance is a necessity if one is to be successful in avoiding trauma to the inferior laryngeal nerves in thyroid surgery

Unilateral paralysis of the vocal cord is usually caused by neoplastic disease, including the larynx, pharynx, bronchus, esophagus and thyroid glands as well as metastatic lesions of the mediastinum When such paralysis is associated with a mass in the neck, tumors of the thyroid are frequent offenders Seldom, however, does one find an aneurysm of the inferior thyroid artery as the cause of paralysis of one vocal cord Doumanian and associates[37] report such a case, and on clinical examination the cause was undetermined Operative treatment not only established the diagnosis but also corrected the condition In a study of 633 cases of such paralysis, Doumanian and associates found that cardiovascular disease was the second most important factor in the etiology of paralysis of the vocal cord, other causes of paralysis in these cases were mitral stenosis, aneurysm of the aorta, cardiomegaly and congenital heart disease

In a discussion of the significance of paralysis of recurrent laryngeal nerves Vinson[38] calls attention to inflammatory conditions affecting the recurrent nerves Such a paralysis due to inflammation can well be compared to Bell's palsy, which often subsides spontaneously Vinson reports 14 cases of paralysis of one vocal cord due to inflammatory reactions In seven patients the voice and normal function of the larynx were completely restored There is no specific treatment for this kind of paralysis but secondary traumatic laryngitis may be avoided by requesting patients to refrain from forcing their voices

Mollison[39] reports eight cases of idiopathic recurrent laryngeal paralysis seen over a period of years No attempt is made to give a definite diagnosis as to the cause of the paralysis The symptoms and clinical observations are described

STRIDOR

Congenital laryngeal stridor resulting from hypermobility

PARALYSIS

Williams[34] investigated 100 thyroidectomies for benign, nonrecurrent goiters Preoperative indirect laryngoscopy was performed by a laryngologist, and there was no evidence of a nerve lesion Immediately after the operation the larynx was examined and in none of the 100 patients was there any impairment of motion of the vocal cords Four or five days later the cords were examined by indirect laryngoscopy and seven of the 100 patients had paralysis of one cord Five of the seven patients made a complete recovery and two had permanent paralysis Williams was certain that the nerve had not been divided, crushed or traumatized because of its behavior at the end of the operation Of the possible causes of early postoperative paralysis Williams believes that edema of the nerve causing compression of its fibers is the most likely The various aspects of the problem of dissecting out the recurrent nerve so as to visualize this structure properly are discussed No definite advice to the general surgeon can be offered except that preoperative examination of the larynx in all potential candidates for thyroidectomy should be routine

Fateen[35] conducted experiments on the recurrent laryngeal nerve in dogs in order to establish the extent and permanency of injury from crushing this nerve Over a long period 80 dogs were anesthetized, and the recurrent laryngeal nerve was exposed and crushed with a Kocher forceps After recovery the barking was hoarse and remained hoarse for about three months, after which it resumed its normal ringing character in every case Fateen concluded that nothing should be done until three months after thyroidectomy, since during this time there is hope of spontaneous recovery Crushing of the phrenic nerve produced no significant change in respiration Numerous recent articles on this subject are quoted including some advocating attempts at anastomosing the hypoglossal and the distal end of the recurrent nerve

Finochietto[36] makes an ardent plea for better knowledge of the anatomy of the thyroid region and more careful surgical technique in order to avoid trauma to the recurrent laryngeal nerve Bleeding vessels should be clearly seen before forceps should never be considered a trivial condition Occasionally, tracheotomy becomes necessary as a life-saving measure Stenosis in the region of the larynx or a growth may produce stridor Immature cartilagenous structures of the larynx, such as the epiglottis and the arytenoids, are frequently responsible for stridor in infants

STENOSIS

Laryngologists are familiar with the problem of chronic stenosis of the larynx and upper trachea Use of acrylic materials in the larynx, as described by Norris,[44] helps to solve the troublesome problem of inflammatory reaction caused by intralaryngeal rubber molds Despite frequent changing of rubber molds, ulcerations and granulating tissue were often observed Acrylic materials do not irritate, and are nonporous, easily shaped and comfortable for months Norris gives a good description of the acrylic resins, which constitute one class of plastics The object of core-molding may be to prevent development of chronic stenosis in cases of recent trauma or fractures of the laryngeal cartilages In addition to this, maintenance of dilatation in chronic stenosis is most important The various types of molds are illustrated in this article With the increasing number of laryngeal injuries from automobile accidents, the acrylic mold will play a more important part in rehabilitation of the fractured and stenosed larynx

Cranmer[45] summarizes his excellent article on strictures of the larynx by stating that laryngeal injuries are assuming greater importance primarily because of automobile accidents with their high morbidity and mortality rates That these injuries do not occur more commonly is due mainly to the protected position of the larynx The many types of open and closed injuries vary widely in severity Cranmer emphasizes the fact that early treatment should be directed toward maintaining the airway with tracheotomy, prevention of stenosis, internal splinting and careful repair of lacerations Later cicatricial stenosis may indicate reconstructive surgery and rehabilitation is not complete without speech therapy for persistent hoarseness of the epiglottis may be corrected by a simple operative procedure as described by Ochs.[10] With a Davis Crow mouth gag in place, the epiglottis is exposed, and the mucosa on the inferior two-thirds of the lingual surface of the epiglottis is removed in conjunction with a corresponding amount of mucosa from the base of the tongue These raw surfaces will adhere so that the epiglottis is held in a more erect position, and the stridor is corrected This surgical procedure was performed on several occasions years ago by one of the reviewers using the suspension laryngoscope to visualize the epiglottis with excellent results

Smith[11] reports two cases of severe lumbar meningomyelocele, which at one or more times had been seen as emergencies because of laryngeal stridor One of these patients died of hydrocephalus, the post-mortem examination revealing no abnormality of the larynx The question of possible "coning" of the medulla with dysfunction of the vagi is raised as a cause for this Smith warns that tracheotomy should not be employed in such cases, but he does not say what form of treatment should be employed to relieve the obstructed airway in such cases

Schutt and Robb[12] report the clinical and necropsy observations on an infant with intermittent stridor which appeared in the third week of life and persisted until death in the sixth week An anomalous left pulmonary artery, coursing posterior to the trachea, between it and the esophagus, together with the upper left pulmonary vein and aorta crossing anterior to the trachea in normal fashion, formed a vascular ring around the trachea Progressive obstruction with pulmonary emphysema followed Only five similar cases have previously been reported, three of which were successfully corrected by operation The embryologic considerations are also discussed in this interesting report

Pereira[13] presents a good discussion of stridor The condition is considered under two large divisions, acute and chronic Eight subheads are discussed under acute stridor and only four are classified as chronic Laryngeal stridor is seen more frequently in the infant, and when present, it

accident, before the cartilages have become fixed in their new positions and before development of scar tissue The most effective treatment of chronic laryngeal stenosis is preventive tracheotomy rather than intubation, low rather than high tracheotomy, removal of feeding tubes if possible, and therapy of acute laryngeal abscesses, tuberculosis and crushing injuries of the larynx This article should be read by all who practice laryngology

ANESTHESIA

Steiner[49] describes a new type of laryngeal spray utilizing the sprinkler system with ten small holes in the distal end of the cannula The patient is first made to gargle with an analgesic solution and then a laryngoscope is used to visualize the larynx The new laryngeal syringe, consisting of a cannula 20 cm long with a slight-anterior curve at the distal end, is introduced into the larynx This laryngeal spray is intended to produce thorough anesthesia of the larynx, thereby facilitating introduction of intubation tubes for anesthesia

Proctor and Edgerton[50] describe management of a case of traumatic occlusion of the larynx secondary to endotracheal anesthesia Endotracheal anesthetization performed on two occasions three years apart was followed by progressive, increasing hoarseness and dyspnea Mirror examination showed the larynx to be filled with a granulomatous mass After tracheotomy a large skin flap was turned back across the midline of the neck, and the laryngeal cartilages were explored The thyroid cartilage was split in the median line, and the granulomatous tissue was removed from the interior of the larynx A split thickness graft was fitted about a stint of polyvinyl alcohol sponge shaped to fit the glottic aperture This was held in the denuded portion of the larynx by a No 28 stainless steel wire suture passed through the thyroid cartilage and skin The thyroid cartilage was closed by appropriate sutures, and excellent final results were obtained Good illustrations accompany this excellent paper

Gabuya and Orkin[51] describe a new laryngoscope with a curved blade to facilitate introduction of an intubation tube

Cicatricial stenosis of the larynx resulting from a feeding tube is rather rare Schwartz, Devine and Erich[16] found only five of 142 cases of laryngeal stenosis seen from 1940 through 1958 to be due to feeding tubes Management of laryngeal stenosis depends on the degree of respiratory and vocal impairment and the general condition and age of the patient Minor degrees of stenosis may be overcome by dilation Severe degrees of stenosis or completely stenosed larynges can be successfully corrected only by surgical reconstruction consisting of excision of scar tissue, lining the larynx with a free split-thickness skin graft, and wearing an obturator to prevent contraction and subsequent closure of the stoma Such treatment is long and drawn out, requiring much patience on the part of the laryngologist as well as the patient

Rapid progress has been made in the surgical treatment of chronic laryngeal stenosis since Schmiegelow described his method in 1929 His operation with modifications has given excellent results Smathers[17] believes that many patients continue to wear a tracheotomy tube because of cicatricial stenosis of the larynx This can be corrected by proper treatment, which consists of removal or control of the infectious process before reconstruction is attempted Smathers emphasizes the necessity of low tracheotomy and states that reconstruction of the laryngeal lumen is a three-stage procedure requiring six to 12 months for completion Through a laryngostomy approach the stenotic area is removed and a skin graft inserted over foam rubber Ten days later the rubber stent is removed, and an acrylic obturator molded to the interior of the larynx is inserted into the larynx and allowed to remain there from six to nine months, after which the laryngeal stoma is closed and decannulated if the procedure has been successful

Holinger and Johnston[18] present an excellent study of 163 cases of chronic laryngeal stenosis treated during the past ten years It now seems that trauma is rapidly replacing acute laryngeal disease as the commonest cause of chronic laryngeal stenosis Management of these various problems is discussed Emphasis is placed on the importance of performing reconstructive procedures within the first few days of the

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Gabuva and Orkin[51] describe a new laryngoscope with a curved blade to facilitate introduction of an intubation tube for induction of endotracheal anesthesia Advantages are that the new laryngoscope may be introduced more easily into small mouths, preferably on the right side, better visualization of the larynx, better protection of the upper teeth, and improved use of binocular vision General views of this new laryngoscope are shown

Campkin[52] states that contact ulceration and pedunculated laryngeal lesions may occur in the pathogenesis of post-intubational granuloma, an uncommon complication of endotracheal intubation He believes that the incidence of this form of granuloma is increasing because of the frequent use of endotracheal intubation in recent years Post-intubational granuloma is more common after thyroidectomy probably because of hyperextension of the head and the placing of a sand bag or pillow under the shoulders In this position the endotracheal tube will make pressure on the vocal process

Schwarzbart[53] reports eight cases of intubation granuloma He believes that the incidence of this condition can be decreased by careful preoperative screening of surgical patients for catarrhal, allergic and inflammatory conditions with postponement of operation when possible if any of these conditions exist Postoperative physical rest and silence are recommended together with systematic laryngoscopic examination to prevent progression or development of these lesions

SURGERY

Lillie and Devine [4] describe the indications and their technique for the laryngofissure operation, which does not differ from the generally accepted procedure In brief, it consists of suspension laryngoscopy and biopsy An intratracheal tube is inserted through the mouth, and the suspension laryngoscope is removed The neck is prepared and draped, and tracheotomy is performed The intratracheal tube is removed, and a cuffed tube is introduced through the tracheal stoma It is a bit confusing to note, in another paragraph, a statement that Lillie and Devine always perform the tracheotomy with use of a local anesthetic The usual midline incision is described, and the thyroid cartilage is split with an oscillating saw Retraction of the wings of the thyroid laterally brings into view the lesion on the vocal cord Dissection is carried out, and the mass is removed *en bloc* Bleeding is controlled by ligation and electrocoagulation The tracheotomy tube is removed on the fourth or fifth day The incidence of cures obtained by others ranges from 75 per cent to 92 per cent, but the results obtained by Lillie and Devine are not given

Schall[illegible] describes an extralaryngeal approach for certain benign lesions of the larynx by means of an incision at the upper margin of the thyroid alae on the involved side By dissection the tumor is exposed and usually extirpated without entering the interior of the larynx This simple, practical operation is briefly described and well illustrated It is best adapted for slowly growing, submucosal, supraglottic and intraglottic new growths and cysts of the larynx

In an excellent comprehensive article Reed, Mueller and Snow[illegible] present a detailed report of 200 cases of radical neck dissection between 1948 and 1958 Their method of study and analysis of the primary site and grade of the lesions and the classification of the nodes found in the neck leave little to be desired For those interested in the study of neck dissections this article is highly recommended

Conley[illegible] presents methods of primary closure of the larger laryngeal wounds created in performing partial laryngectomies, with illustrations His description of the technique of flap movement of mucous membrane is such that to the uninitiated it sounds easy Conley claims the resultant voice and airway show much improvement after use of a mucous-membrane flap in the rehabilitation of the partial laryngectomy wound

LARYNGOCELE

DeRosario and co-workers[illegible] state that laryngocele, a rare and interesting swelling of the neck, arises from the ventricle of Morgagni, where an outpouching frequently occurs in the roof of the ventricle It is usually caused by repeated elevation of intralaryngeal pressure, which is transmitted to the sacculus, and which may dilate to form a laryngocele As the sacculus dilates, it is usually contained by the thyrohyoid membrane, however, if it penetrates this membrane, it then becomes known as an external laryngocele and is located in the anterior triangle of the neck Such a case is reported

BENIGN TUMORS

Scleroma is an endemic disease prevalent in Eastern Europe, Russia, South America and certain far eastern islands It is a specific granuloma affecting mainly the nose, although the pharynx, larynx, trachea and bronchi may be affected Massoud and Awwad[59] present a clinical review of 84 cases of scleroma treated between 1951 and 1956 Eighteen patients had laryngeal involvement, in all of whom the subglottic region was affected When granulomatous infiltration involved the interior of the larynx, deformity of the structures resulted, and concentric narrowing of the subglottic airway and upper part of the trachea occurred Known antibiotics have no effect on scleroma Roentgenotherapy is of some benefit

In an editorial[60] it is stated that benign tumors of the vocal cords frequently present confusing diagnostic problems, in fact, the clinical picture cannot always be reconciled diagnostically with the pathologist's interpretation of the extirpated tissue This is particularly true for polypoid laryngitis since there is no acceptable histopathologic picture characteristic of laryngeal polyps It is thought that these polyps originate in the subepithelial space of Reinke and are able to assume various histologic appearances There is general concurrence that trauma of vocal abuse and the irritation from smoking are the common inciting factors producing polypoid laryngitis Accurate surgical removal of the polypoid tissue followed by pathologic examination for confirmation of the clinical diagnosis is always desirable

Chondromas are among the rare connective tissue tumors of the larynx, 80 per cent of which are found in men Schiff and Bender[61] report such a case occurring in a housewife who had a tumor removed from the larynx 28 years before They were unable to obtain the original histologic diagnosis A laryngofissure approach was used to remove the chondroma from the right side of the larynx after which the patient made an uneventful recovery

Walter[62] reports a case of chondroma of the larynx In his review of the literature he found 96 cases An outstanding characteristic of this tumor is failure to metastasize, but frequent local recurrence results from inadequate removal

Arnvig[63] reports apparent regression of advanced papilloma of the larynx after use of hydrocortisate spray three times daily in a seven-year-old girl During nine months, the tumor had not recurred Simultaneously, with this treatment, however, she had had repeated simple excision of the papilloma, application of podophylline solution, roentgenotherapy and tracheotomy, so that recovery cannot definitely be attributed to the hydrocortisate spray If this simple method of treatment proves to be effective in other cases, it will be a most valuable addition to the therapeutic armamentarium of the laryngologist

Moffitt[64] successfully used wart vaccine of bovine origin in the treatment of multiple papillomatosis of the larynx Dramatic cessation of regrowth occurred in one case and decided improvement was noted in three others Since papilloma of the larynx has resisted all forms of treatment, it seems worth-while to use this vaccine in additional cases to determine further its value

Nunez[65] presents a review of a large number of articles written on the treatment of multiple papillomatosis of the larynx He quotes these various authors and comments on the therapy employed by each He is forced to the conclusion that there is no one treatment for laryngeal papillomatosis and that many of these cases clear up at the age of puberty He advocates pinching of the papilloma under direct laryngoscopy with as little damage to the normal mucosa and to the vocal cords as possible

Baker informs us that pseudosarcoma is the term introduced by Lane to indicate large polypoid connective tissue masses associated with squamous carcinoma of the mouth, fauces and larynx Occasionally, these masses assume such prominence as to dominate the clinical picture completely The lesion has the gross appearance of a sarcoma, and when examined with the microscope, resembles a highly anaplastic growth with giant neoplastic cells The pathology is thoroughly discussed Baker reports three cases of pseudosarcoma of the larynx, which is a nonmalignant connective tissue tumor, usually associated with squamous cell carcinoma

Harper[67] states that fatty tumors of the pharynx and larynx are uncommon and are probably one of the rarest forms of benign tumors encountered in this region He found 30 such cases in the literature and added one of his own This was a lipoma attached to the posterior portion of the larynx, it was smooth and pale yellow It had caused choking, coughing and laryngeal spasm Its surgical removal presented no difficulty

Nimbkar, Gadre and Desai[68] report a case of neurofibroma of the larynx and review the literature on this subject In this location neurofibroma is considered rare These tumors have no tendency to invasion but tend to destroy the structures in the vicinity by direct pressure The chief symptoms are hoarseness and eventually dyspnea Treatment is surgical excision either intralaryngeally or by an external approach, and as a rule, the diagnosis is made postoperatively by the pathologist

Bailey[69] states that during the last two years a series of plasma cell granulomas have been detected in a small farming area around Plymouth, England The lesion consists of ulceration or proliferation of tissue within the mouth, pharynx and larynx and resembles no other condition It is extremely painful and is resistant to any form of local treatment except Cortisone derivatives The one case involving the larynx required not only tracheotomy, but also a transhyoid approach to remove the granulomatous lesions, which have not recurred

Laryngeal cysts may be congenital, retention, degenerative, lymph, blood or implantation cysts Retention cysts are by far the most common type of cyst found in the larynx and most frequently occur in the glosso-epiglottic recess El Mofty[70] reports a large, trilobed, laryngeal cyst obliterating view of the glottis and occupying the right aryepiglottic fold, the ventricular fold and the pyriform fossa The cyst was successfully removed through an incision along the border of the sternocleidomastoid muscle

Amyloidosis is the deposition into tissues of a substance called amyloid because of its resemblance to starch Many theories have been developed in an attempt to determine the etiology of amyloidosis Although the condition is rare, Holinger, Johnston and Delgado[71] report seven cases seen by them The clinical picture and pathology are well described The diagnosis is always made by microscopic examination of the tissue The treatment advocated is surgical removal endoscopically, which was successful in all but one of their cases

Smith[72] reports a case of a true benign striated muscle tumor of the larynx Only two other cases have been recorded since Ewing's classical description in 1919 According to Smith, this condition is distinct from the granular cell myoblastoma of the larynx, and the granular cell probably represents a degenerative lesion of muscle rather than an embryonic precursor of striated muscle fibers

MALIGNANT TUMORS

Virtama and Harma[73] reviewed 724 cases of laryngeal carcinoma from the standpoint of influence of institution of adequate treatment They believe that delay in institution of adequate treatment is the most significant factor in the mortality rate of laryngeal carcinoma Among the 724 patients, delay in consulting a physician was responsible for 50 per cent of the total sample and the next most significant factor was failure of the general practitioner to recognize symptoms

Postoperative management of carcinoma of the larynx entails not only the immediate period while the patient is in the hospital, but also the five-year period after operation during which the patient should be observed and treated for associated conditions, according to Callahan,[74] who reports the case of a patient emotionally disturbed after operation on the larynx These emotional disturbances must be recognized and managed with the greatest of care

Fisher and Miller[75] reviewed 48 cases of carcinoma *in situ* They point out that this type of lesion continues to present a problem to the laryngologist It is still not possible to describe a characteristic clinical picture obtained by direct or indirect laryngoscopy that would lead to a diagnosis of carcinoma *in situ*, however, this lesion can be recognized in three distinct histopathologic types the basal, the squamous and the bowenoid Leukoplakia was the most frequent pathologic change observed on the cords The therapy of this condition is fully discussed

Asbury[76] reports an unusual case of sarcoma of the larynx in a woman 36 years old Nine years previously she had had a tumor in the right knee which eventually was diagnosed as chondrosarcoma The same diagnosis was established in the larynx, and this was thought to be metastatic, however, because this was the only manifestation, the question arose as to the possibility of this lesion's being primary Sarcomas constitute only 1 per cent of malignant lesions of the larynx

Pietrantoni, Felisati and Finzi[77] studied the frequency of laryngoceles and carcinoma of the larynx and the relationship of these two types of lesions The records of more than 1,000 patients who had laryngectomies were examined, in 857, complete records with tomographs were available for study The incidence of laryngocele and carcinoma of the larynx occurring simultaneously was 6 18 per cent It was concluded that there is no definite relationship between these two types of lesions and that other factors are responsible for the production of laryngoceles

Clarke[78] reports successful use, for a long period, of a prosthesis to close a large esophageal fistula in a man, 71 years old, with extensive intrinsic carcinoma of the larynx He was given a heavy course of irradiation before total laryngectomy Postoperatively, a large pharyngo-esophageal fistula developed, which persisted in spite of repeated plastic operations After six months of tube feeding, the portex tube as described by Shaw and Ormerod in 1957, was inserted into the pharynx and allowed to remain there for seven months During this time food could be taken orally, and secretions would not spill out into the tracheal stoma Tissue reaction necessitated changing of the tube every three months after this The shape of the tube, a cylinder with its upper end flared and a fusiform dilatation just below the flare, renders it self-retaining, and apparently, it is well tolerated by the patient This seems to be a simple, practical method of temporarily solving a debilitating complication of laryngeal surgery

Foroughi and Rosedale[79] report the case of a 64-year-old woman with intrinsic carcinoma of the larynx removed by laryngofissure The patient was asymptomatic for 11 years before recurrence necessitated laryngectomy A concise review of the age and sex incidence of this disease as well as its usual histologic types and location are included

Sarma[80] makes the astounding statement that cancer of the larynx forms approximately 33 per cent of all tumors of the body seen in Assam He also states that cancer of the larynx forms 50 per cent of cancers of the upper alimentary tract No reason is offered to explain this tremendous incidence of this particular lesion Sarma frankly states that the population is ignorant of the symptoms of cancer of the larynx and implies that the doctors in Assam are not much better informed, hence, there is much delay in establishing a diagnosis It is unfortunate that so little of ear, nose and throat diseases is taught undergraduates in Assam

Storer and Goodsitt[81] remind us that carcinoma of the larynx is primarily a disease of males They report a case of a highly malignant oat-like-cell carcinoma occurring in the larynx of a woman aged 25 years The patient received Roentgen radiation to the neck for tuberculous adenitis Storer and Goodsitt question whether this therapy may have played an important part in the development of carcinoma of the larynx in such a young woman

Nelson[82] reviews, discusses and reports the newer methods of treatment of cancer of the laryngopharynx A number of cases are reported, each one had such extensive lesions that only major surgical procedures developed in the last few years could offer these patients any hope Laryngectomy, single or bilateral block dissection of the neck and pharyngotomy and pharyngectomy offer these patients an excellent chance of cure

Feinmesser and Gay[83] report an unusual case of carcinoma of the larynx In January, 1952, a biopsy specimen from the right vocal cord was reported as anaplastic carcinoma The patient refused operative or radiologic treatment Two years later a painless swelling about the size of an egg developed on the right side of the neck Indirect and direct laryngoscopy failed to show evidence of a lesion in the larynx The tumor was removed surgically, the pathologic report was anaplastic carcinoma involving a lymph gland Undoubtedly, this was metastatic carcinoma of the vocal cord, which now showed no evidence of malignant growth Apparently, the cordal carcinoma was removed completely at the time of the biopsy Radical block dissection of the neck was done, and five years later the patient showed no sign of recurrence It is now seven years since the original biopsy

Surgical treatment of cancer of the post-cricoid region which involves the entire circumference of the mouth of the esophagus is difficult for the patient as well as for the surgeon Since 1926 Sercer[84] tried various methods of management of such lesions without success If the tumor has not invaded the larynx, Sercer advocates lateral hypopharyngoscopy with complete removal of the malignant lesion followed by severing of the trachea and creation of a permanent tracheal stoma The larynx is then slightly displaced backward, and the lower portion of the larynx and trachea are sutured to the upper portion of the esophagus and the upper portion of the larynx to the hypopharynx Interference with the blood and nerve supply is assiduously avoided, and a feeding tube is passed between the vocal cords into the esophagus and stomach Rapid convalescence occurred in the three cases reported These patients speak with the larynx and swallow through the same organ Little comment is made on the quality of the voice

O'Keefe[85] reminds us that early in the care of the patient with laryngeal cancer, we are confronted with the decision to operate and must select a technique appropriate to the task Such a selection is based on inclusive appraisal of the patient, including his tumor, and it should have as its primary intention, the best chance of cure O'Keefe discusses the various types of laryngeal cancer and their criteria of operability The intent of this presentation is to help clarify the issue of conservative vs radical in the surgical treatment of laryngeal cancer

In an interesting and comprehensive paper Norris[86] discusses the causes of failure in surgical treatment of malignant tumors of the larynx This study covers 209 cases of partial laryngectomy, 181 cases of laryngectomy and 64 cases of laryngectomy with simultaneous block dissection Each of these groups is considered separately, and appropriate comments are offered The changing surgical trends for carcinoma of the larynx is discussed, and the fronto-lateral operation is advocated in the endolaryngeal cases with involvement of the anterior commissure The indications for radical neck surgery in conjunction with laryngectomy are increasing and statistics show less recurrence when this form of surgical treatment is followed This is a most interesting presentation which should be read in its entirety to be appreciated

McGavran, Spjut, and Ogura[87] present a critical analysis of the success and failure of laryngofissure in the treatment of laryngeal carcinoma Laryngofissure as the only definitive therapeutic procedure was considered a success if the patient was alive five years or more without evidence of persistent carcinoma or if the patient died within five years but had no evidence of carcinoma at necropsy The operation was considered a failure if subsequent irradiation or surgical therapy was employed, carcinoma recurred, or the patient could not be traced or died without necropsy Much depends upon proper evaluation of the existing lesion Seventy-four per cent of well differentiated cancers were treated successfully whereas only 35 per cent of the poorly differentiated cancers were controlled by laryngofissure The presence of tumor at the excisional margins and invasion of intrinsic muscles contribute to failures. Much emphasis is placed on periodic observation for indefinite periods.

It is surprising to read that the authors conclude that "Because the survival rates of patients with intrinsic laryngeal carcinomas treated by irradiation are reported as good as, if not better, than those treated by laryngofissure, and because the functional result in terms of voice is better after irradiation therapy, it would seem that irradiation should supplant laryngofissure as the therapy of choice."

Riegler[88] analyzes statistics in the treatment of laryngeal carcinoma at the Roswell Park Memorial Institute, Buffalo, New York, from 1935 to 1955. Before 1951 most laryngeal malignant tumors were treated by radiation, and after 1951 surgical removal was more extensively employed. The results for cordal, intrinsic and extrinsic laryngeal malignant tumors were strikingly better with surgical treatment than with radiation.

Work[89] reports his surgical experience with 22 patients treated initially with Roentgen rays for primary carcinoma of the larynx. All had received adequate roentgenotherapy and surgical intervention was necessary because the carcinoma had not been arrested. Work describes the groups into which these cases were placed when seen and the type of surgery performed in each group. All needed wide field laryngectomies, and in addition several needed radical neck surgery. The time interval ranged from two months after irradiation to 12 years. No surgical technical difficulties were encountered when operated upon within a year after radiation, however, surgery of the neck, on patients who have had irradiation 12 months or more for cancer of the larynx, is fraught with many difficulties. This is an excellent article worth reading in its entirety.

In an interesting article appearing in the Cancer Bulletin[90] cancer of the larynx is discussed at length. It is stated that the optimal chance for preservation of life and the voice of the patient with laryngeal cancer is afforded if the surgeon and the radiotherapist work together as a team to determine the treatment of choice When successful, radiotherapy offers the advantage of preservation of the voice whereas surgical intervention offers a higher percentage of cures The complexity of the laryngeal structures and the nature of the different types of cancer in these structures make necessary separate analysis of the therapeutic problem in each case

In a discussion of cancer of the larynx Mill[91] divides the larynx into supra-glottic, glottic and sub-glottic regions He emphasizes the significance of persistent hoarseness and the value of early examination Teleradium bomb and the cobalt beam are discussed as one form of treatment and laryngofissure and laryngectomy as the other form The necessity on occasions of radical neck dissection is mentioned Mill does sound a good warning which should receive attention if after tracheotomy laryngectomy is performed, recurrence of the growth near the tracheal stoma is not at all rare, this implies the possible transmission of cancer cells by tracheal secretion to a new raw area This is food for thought

The purpose of a paper presented by Wood[92] is to describe the techniques and the early results of treatment of carcinoma of the larynx by supervoltage radiation The average laryngologist will be interested in knowing that it is claimed that supervoltage radiation causes less cutaneous reaction Seventy-two patients with carcinoma of the larynx were treated Thirty of these were considered to have stage I with full mobility of the cords, no nodes and disease limited to the mucous membrane, 87 per cent survived one year and 73 per cent two years There were 39 cases in stages II and III and the one- and two-year survival rates were 77 5 per cent and 64 5 per cent, respectively The reviewers are of the opinion that the results obtained in stage I were such that no other patient should have been subjected to such treatment when the survival rate the second year was 73 per cent Other methods offer patients with stage I disease a much greater survival rate

Nachlas[93] describes cobalt 60 teletherapy as essentially a form of super voltage roentgen-ray therapy which is, for all practical purposes, identical to two to three million volt roentgen-ray generators When first results of cobalt 60 were published, the general trend of opinion was highly favorable, however, recent reports, according to Nachlas, indicate cobalt 60 therapy is not proving as efficient in the number of five-year cures as was first hoped Nachlas sounds a warning to surgeons With conventional Roentgen-ray the skin assumes a typical burned appearance, but with cobalt 60 the skin appears normal in spite of skin devitalization and will lead the surgeon into serious complications he might have avoided Judging from the findings in the series of cases herein presented and a review of the literature, Nachlas concludes that surgical removal is still the treatment of choice for carcinoma of the laryngopharynx

Padovan[94] describes use of artificial radioactive elements in 195 cases of malignant tumors of the larynx Radioactive cobalt pellets, strontium tablets or cesium sticks were introduced into the lumen of the larynx by means of laryngofissure or endolaryngeal catheter, to deliver doses of irradiation to the tumor as high as 15,000 r, several times higher than could be accomplished by external irradiation This method is reported to produce less apparent effect upon the cartilage of the larynx and skin of the neck and to be simpler, shorter, more easily tolerated and much cheaper than conventional methods of irradiation Although the question of whether the same results might be achieved by giving postoperative irradiation, or no irradiation at all, is not actually answered, use of isotopes will almost certainly attract the further interest and attention of laryngologists

Dillion[95] points out the importance of close cooperation between the surgeon and radiation therapist in the effective treatment of laryngeal tumors In a discussion of carcinoma of the larynx he seems to favor radiation therapy for many types of lesions His discussion of the surgical indications leaves much to be desired Cordectomy, partial laryngectomy for anterior lesions and total laryngectomy are discussed, but laryngofissure and thyrotomy are not mentioned The three major techniques employed in radiation therapy of the larynx are described in detail The poor description of surgical techniques leads one to believe that the author is a radiologist

In a review of the results of treatment in six major treatment centers, as well as 58 cases of his own, Cantril[96] reports that there have been improvements in the techniques of radiation therapy in the past two decades. Chief among these are careful clinical observation during the course of treatment to determine the amount of fractionation of the planned dose, and strict limitation of the size of the field commensurate with the extent of the disease. He believed that more collaborative selection of patients for limited surgical treatment, laryngectomy or irradiation would result in higher rates of cure with preservation of the larynx. He concludes that treatment is least successful in advanced endolaryngeal cancer with cartilaginous invasion, immobility and edema of mucosa. These cases, as well as lesions at the anterior commissure involving thyroid cartilage, and all frank subglottic cancers, he believes, are best treated by laryngectomy. He advocates roentgenotherapy for cancer involving the epiglottis.

A prediction by the pioneer radiotherapist, Henri Coutard, that between one-third and one-fourth of cancers of the larynx can be made to disappear through irradiation seems to be substantiated by the survival of approximately one-third of the total number of patients treated in whom subsequent laryngectomy was not necessary. Another one-sixth of the total number survived with subsequent laryngectomy. This paper requires careful reading to avoid misinterpretation of wording at times but is of interest to all who treat laryngeal carcinomas.

Perrone[97] describes his method of combatting the annoying and serious problem encountered in the development of tenaceous secretion with actual crust formation in the tracheobronchial tree after laryngectomy. The apprehension of the patient during the episode of dyspnea, due to obstruction in the tracheobronchial tree by the secretion and crusts, leads to consternation with mental depression, and often despondency. In the series surveyed by Perrone crusts and scabs developed in the trachea of only 2.75 per cent. Particularly was this true during the winter months. In such cases Alevaire with a nebulizer softened and dissolved these crusts by spraying this solution into the tracheobronchial tree.

Norris[98] states that deformities resulting from total laryngectomy and from operations which include laryngectomy depend in large part on the extent of the surgical removal Persistent pharyngeal fistula, stenosis of tracheal stoma and stenosis of upper trachea and keloids are the most common deformities encountered These and many other conditions occurring postoperatively are well discussed

Szunyogh[99] describes the shoulder disability after radical neck dissection in which the spinal accessory nerve is necessarily severed This results in palsy of the upper half of the trapezius muscle with consequent "shoulder drop," and eventual painful periarthritis of the shoulder joint This discouraging sequence of events can be relieved only temporarily by use of sedatives, analgesics, local heat, and elevation in a sling Shoulder exercises seem to be useful in slowing down the onset of periarthritis, but this effect is always temporary In spite of this disability, Szunyogh does not advocate preservation of the nerve for successful surgical treatment of cancer

Nursing care of the laryngectomized patient presents specific mechanical and psychologic problems[100] Apprehension about loss of the voice is undoubtedly intensified by the necessity for innovations that are strange and, therefore, alarming to the patient, such as the laryngectomy tube, nasogastric feedings and the actual realization of inability to talk Preoperative instructions are most important to the family and patient, and this article in the Cancer Bulletin[100] states that although the necessary procedures for postlaryngectomy care of the patient are simple, they must be performed with care and skill Frequent cleansing of the laryngectomy tube and suction of thick mucus from the tracheobronchial tube are important for the comfort of the patient If cyanosis develops, immediate removal and changing of the laryngectomy tube is advisable, as crust and thick gelatinous mucoid material will frequently block the distal end of the tube The inner cannula should be cleansed frequently during the first few days Teaching the patient to care for his own needs restores his self-reliance, and when sufficient time has elapsed and normal feeding is resumed, the patient should be prepared psychologically for rehabilitation speech therapy

MacGregor[101] devised a new appliance whereby laryngectomized patients in whom esophageal voices fail to develop may be helped to produce a voice by artificial means It consists of an electrically vibrated diaphragm built into an upper dental prosthesis thereby carrying the source of the sound directly into the mouth The advantage of the oral vibrator is that all sound is generated where it can be used for voice production, resulting in highly intelligible speech The device is relatively inconspicuous Unfortunately, MacGregor's description is meager and the appliance is not illustrated This new apparatus will be of much interest to all laryngologists who have the welfare of their patients at heart

In an analysis of the psychologic factors determining the success or failure of the rehabilitation program of the laryngectomized patient Stoll[102] is of the firm opinion that the personal equation is all important He cites the extrovert's eagerness to learn esophageal speech quickly and to show how well he can speak, whereas the introvert has much difficulty not only with speech production but also with readjusting emotionally to loss of the voice box

In a discussion on rehabilitation after laryngectomy Moses[103] thinks the ideal treatment for these patients will be through the joined forces of the surgeons and medical voice therapists who are able to do more than just teach the patient to belch esthetically He believes that new methods must be developed for therapy and control by gaining deeper insight into the patient's problems and by careful observation and diagnosis Moses does not think that a laryngectomized patient is the proper type of individual to teach voice therapy

All laryngologists will concur with Putney[104] that carcinoma of the larynx should be completely removed without regard for the subsequent establishment of a voice A voice can be developed whether the larynx is removed with no contiguous tissue or with the surrounding structures In order to determine what factors, if any, had a bearing upon failure to develop a voice from an anatomic or physiologic standpoint after laryngectomy, Putney reviewed 440 cases of patients operated upon at Jefferson Hospital No underlying anatomic or physiologic factor was found to account for lack of voice, and patients with extensive surgical removal including most of the pharynx and portions of the tongue as well as the larynx were able to talk expertly The type of cervical incision, the method of pharyngeal closure, or the amount of adjacent cervical tissue excised had no appreciable effect upon establishment of a voice Putney concludes that the primary object in the surgical treatment of carcinoma of the larynx is complete eradication of the malignant tumor, regardless of the sacrifice of adjacent tissue

Conley[105] presents a modification and development of the technique described in 1958 for vocal rehabilitation of the laryngectomized patient Instead of the mucosal tunnel fashioned from the anterior wall of the cervical esophagus, an autogenous graft of the great saphenous vein, 8 cm in length, is used to connect the skin of the neck just above the tracheal stoma with the lower portion of the cervical esophagus A special laryngectomy tube then connects the tracheal airway to the venous tract

In primary implantation of the vein graft a No 8 catheter or plastic tube threaded through its lumen and then inserted into a specially adapted trochar simplifies threading through the fascia between the trachea and esophagus In secondary implantation perforation is made through a fenestrated short esophageal speculum The most troublesome complication of the procedure is closing of the vein, due to neglect by the patient and low grade infection

In a discussion of the technical aspects of speech aids for post-laryngectomized patients Barney[106] first presents the desired objectives from the viewpoint of the patient Since surgical introduction of a transducer into the pharynx is impractical, the only two possible alternatives are to conduct the sound into the mouth through a tube, or by means of a vibrating driver applied to the outside of the throat Hope is expressed that with the development of electronics new aids for laryngectomized patients will be forthcoming

In an attempt at voice rehabilitation Pressman[107] proposes for subtotal laryngectomies, a technique whereby it is possible to remove almost the entire larynx and yet retain a normal airway Three principles are necessary to accomplish this *1* to create a lining, *2* to provide a lumen, and *3* to lend rigidity The first of these is accomplished by using the external perichondrium of the thyroid for a lining, and a proper sized obturator creates and maintains a lumen and transplantation of thyroid alae from a cartilage bank Pressman reports encouraging results with maximum voice rehabilitation after 18 months

Briess[108] describes the specific therapeutic approach used for restoration of normal dynamic equilibrium of the intrinsic and cricothyroid muscles which control the functions of the vocal cords This therapy is considered in four phases and individual muscle function is discussed thoroughly Psychogenic factors usually intensify existing muscle imbalance This and other voice problems are discussed in detail To those interested in voice production this paper is highly recommended

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COURSE IN ALLERGY

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The course will be followed by a meeting of the Gulf Coast Allergy Study Group on February 4th and 5th

For further information, write to Office of the Dean, University of Texas Post Graduate School of Medicine, 410 Jesse Jones Library Bldg, Houston 25, Tex

PARTIAL PHARYNGECTOMY AND NECK DISSECTION FOR POSTERIOR HYPOPHARYNGEAL CANCER

Immediate Reconstruction with Preservation of Voice*

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Winnipeg, Canada,

and

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Recife, Brazil

INTRODUCTION

Malignancies of the posterior wall of the hypopharynx are uncommon and carry a poor prognosis.[1,6,7,10,17] In the past, surgery was considered only in early cases or in the form of a combined excision of pharynx and larynx with reconstruction of a skin-lined gullet, sometimes requiring multiple procedures.[10,17] This removal of a normal larynx,[13] only because adjacent structures were diseased, has led surgeons to attempt to preserve laryngeal function and at the same time eradicate the tumor by a more conservative approach.[3,4,12,14] We have had success with wide local excision of such tumors, preserving the larynx and reconstructing the pharynx primarily by means of stents, skin grafts and muscle flaps.

HISTORICAL REVIEW

In the Anglo-American literature these lesions have been treated mainly by irradiation.[1,8,21] When they have been

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studied in the breast and colon lesions,[2] and seems to hold true for the larynx and pharynx also

Tumors may present a "pushing" margin or an invasive infiltrating margin when slides are examined under a low magnification The "pushing" tumors so far appear to have a much better prognosis,[14,20] however, this characteristic cannot be distinguished on the usual biopsy specimen

Node metastases in the neck from posterior wall lesions are very frequent[11] (50 per cent according to Moss[8]) In this institution the recommended treatment for lymph node metastases has been surgery, because it is felt that cancerocidal doses of therapy cannot be given adequately to the entire neck field (Powers[16])

SURGICAL CRITERIA

The method proposed is advocated for tumors on the posterior wall of the laryngopharynx with or without involvement of the lateral wall of the hypopharynx, extending from above the cricopharyngeus to the level of the tip of the epiglottis or higher This does not include lesions involving the oropharynx

It is essential that the exact extent of this tumor be established Laryngograms[1] are of valuable assistance in determining the superior and lateral extent but do not help to demonstrate the inferior border when tumor is near the cricopharyngeus A barium swallow does not usually delineate the postcricoid and cricopharyngeal area satisfactorily Only by direct laryngoscopy and esophagoscopy can involvement of the cervical esophagus be ruled out This area must be free of tumor to allow an adequate margin of excision of normal mucosa

Films of the cervical vertebrae are taken to rule out bony involvement The tumor must be *mobile* over the bony posterior wall This must be established at the direct examination Portions of the ligamentum may be excised, but this usually is not required

A radical neck dissection is done on the side of major in-treated surgically the larynx and pharynx have generally been removed[5,13,16,19] Simple excision has been done[7] Irradiation had been used for treatment primarily because of surgical difficulties[6] High dose therapy carries the possible dangers of transverse myelitis, cartilage and bone necrosis[8]

We feel that adequate excision of the tumor and its potential field of spread with primary reconstruction of the pharynx, and preservation of the larynx, is feasible and practical in certain carefully selected cases

Conservation has been described in surgery of the hypopharynx, but the term was usually used in reference to saving mucosa and uninvolved laryngeal tissue to reconstruct the pharynx and esophagus but did not refer to conservation of laryngeal function[7] Total laryngopharyngectomy with or without neck dissection has been the recommended surgical procedure if the tumor is large[5,16,19]

PATHOLOGY

Tumors in the pharynx, especially those of the posterior wall, tend to be multicentric in time and space[20] There may be a large differentiated tumor mass with areas of carcinoma *in situ* adjacent to, but separate from, the main lesion These satellite areas are usually but not always close to the main lesion, hence we feel that a one-and-a-half to two centimeter margin of normal pharynx should be excised with the tumor The remaining pharyngeal mucosa may possibly develop malignancy at a later date even though none is present at surgery, however, we do not feel that prophylactic excision of the total pharynx is warranted The lymphatic drainage of the posterior and lateral pharynx is lateral to the cervical nodes and not toward the larynx[8,21] Involvement of the larynx in advanced cases is due to retrograde spread or local invasion It would, therefore, seem that adequate excision of the tumor with a good margin of safety would give as good results as total excision of the larynx and pharynx

Recently preliminary work on laryngeal pathology suggests that the characteristics of the advancing margin of the tumor may influence the prognosis This has been more extensively graft and stent are required If the lesion extends significantly on to the lateral hypopharyngeal wall, a scalene musculo-fascial flap from behind the carotid sheath is mobilized to provide a partial bed for the graft and to reconstruct the lateral wall

SURGICAL TECHNIQUE

Patient is prepared and draped while awake and a vertical tracheostomy is done General anesthesia is started and a Y-incision is made with the anterior limb of the Y extended to the midline at the level of the hyoid bone (see Fig 1)

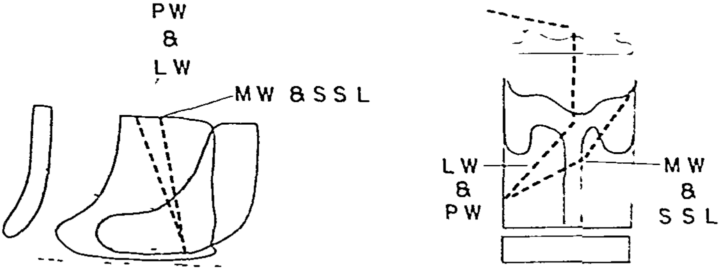

Fig 2 Thyroid cartilage transection variations PW—Posterior wall of hypopharynx LW—Lateral wall of hypopharynx MW—Medial wall of pyriform sinus SSL—Supraglottic subtotal laryngectomy

The customary radical neck dissection is done on the major side and the pedicle left attached from above the hyoid to the lower border of the tumor The hyoid is divided in the midline, and the suprahyoid muscles divided from the cornu Cutting the stylohyoid ligament allows better exposure of the pharynx which is entered through the vallecula on the involved side The tumor is then visualized and inspected but not deliberately palpated in order to avoid unnecessary tumor spread The thyroid cartilage is sectioned and the superior cornu and part of the adjacent ala removed (see Fig 2) A vertical incision is made in the pharynx antero-lateral to the tumor, leaving at least 2 cms margin The tumor is then excised (see Fig 3) Small defects can usually be closed *per primum* Larger defects require a thick split thickness skin graft This is sutured in place with 0000 silk

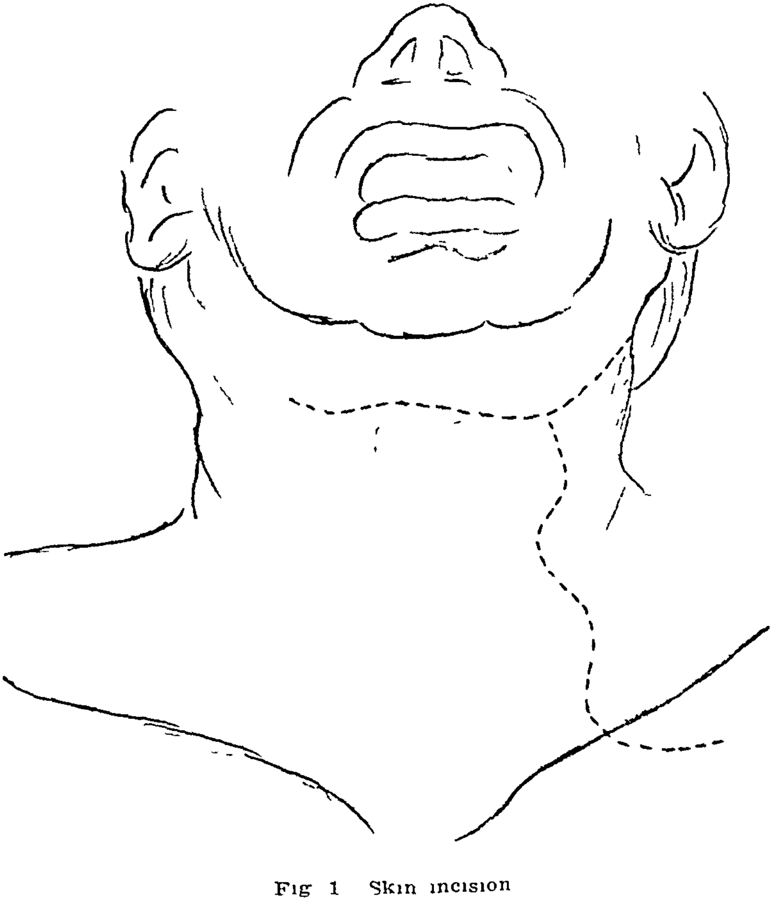

Fig 1 Skin incision

volvement even if nodes are not palpable If these are palpable on one side, this side is dissected even if it be the side of minor involvement If nodes are palpable on both sides a staged bilateral neck dissection is done, or if in one operation, sparing the jugular vein on the least involved side The pedicle is left attached posteriorly from the level of the hyoid to the lower border of the tumor

Lesions under 1 cm in diameter may be excised in continuity with radical neck dissection and the defect closed primarily When dealing with larger lesions a thick split thickness skin wall of the hypopharynx, a graft will be needed laterally as well as posteriorly (see Fig 5) This portion of the graft requires a bed, and this can be formed by rotation anteriorly of a musculo-fascial flap from the scalene group, with preservation of the cervical plexus This flap serves to reconstruct the lateral wall of the pharynx The carotid sheath is retracted laterally, and the anterior scalene muscle is freed above and below the line of the lesion This flap is hinged medially and is rotated anteriorly (see Fig 6) The graft is sutured to the posterior wall and to the muscle flap When the flap is swung into position over the stent, the graft is sutured to the remaining mucosa of the lateral wall of the pyriform sinus and the external perichondrium of the remaining thyroid cartilage The muscles are closed with two layers of interrupted 000 silk A feeding tube is inserted through the stent, and the latter is left in place for at least three weeks Penrose drains are used, and the wound is closed with interrupted subcutaneous and skin sutures of silk

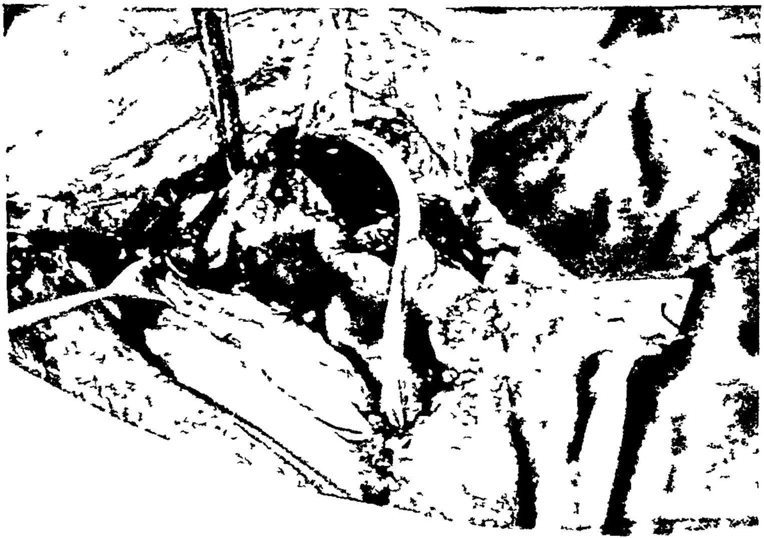

Fig 5 Operation defect Skin graft to posterior of hypopharynx

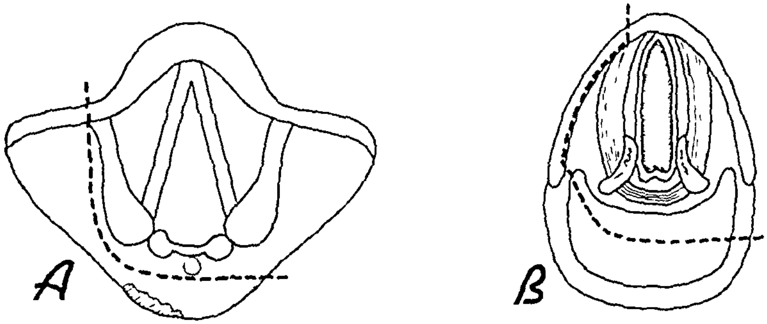

Fig 3 A—Mirror view of area of transection B—Cross section at level of glottis

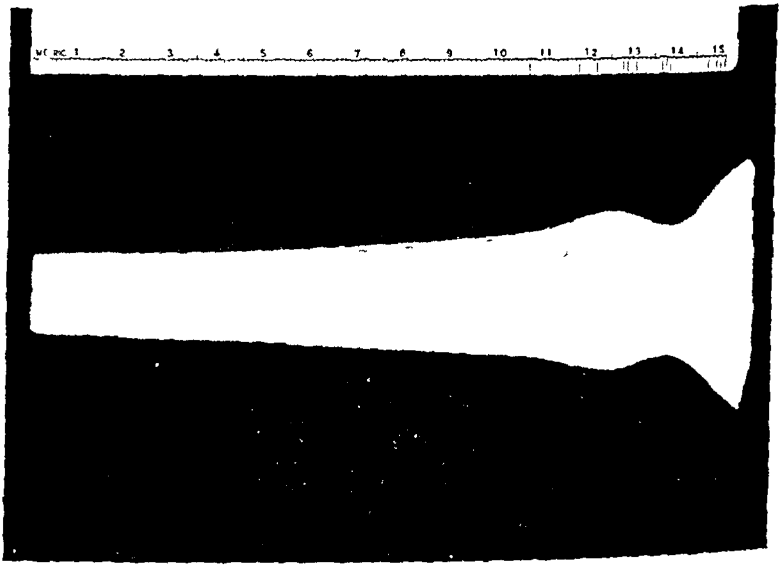

Fig 4 Negus stent

because there is less reaction of the silk to the saliva The sutures slough out spontaneously in several weeks In our experience skin grafts always take very well on the posterior wall A Negus[7] stent is placed, and a feeding tube is inserted through this into the stomach (see Fig 4) The stent remains in place for two to three weeks This is sufficient time, because stenosis is not a problem here

If the excision involves a considerable portion of the lateral

There may be some persistent discomfort with pharyngeal secretions because of the rigidity of the posterior pharynx and the restriction of normal mobility of the larynx The fact that the graft is skin and not mucosa and so is devoid of glands also contributes to the difficulty Preliminary studies of the cineradiography of swallowing of patients who have had partial pharyngectomy suggest that hypertrophy or relative spasm of the cricopharyngeus muscle may contribute to the sometimes awkward handling of the secretions that persist for some time in a few of these patients

In time, the graft appears to change its characteristics and becomes more pink, suggesting a possible metaplasia or reversion to a mucosal type lining, however, no histologic evidence is forthcoming at this time

DISCUSSION OF CASES

Six patients have been operated by this method in the last three years, and all had lesions of the posterior wall of the laryngopharynx Four of them had clinically palpable nodes, and one clinically negative neck had microscopic metastases One patient presented with bilateral clinical cervical metastases, and a staged bilateral neck dissection was performed Skin graft and stent were used in four cases, skin graft alone was used in one patient and primary approximation of the mucosa was accomplished in one instance

The size of the lesion varied from 1x0 5 cms to 3x4 cms Of these, three were "pushing" type tumors and three were infiltrating

The stent was left for three weeks, and the patients were discharged from the hospital by the fourth week, swallowing adequately One patient was lost in the immediate postoperative course from an unexpected cardiac death

The longest survival free of disease has been three years, there has been no local recurrence to date There were no significant pulmonary complications, and the laryngopharyngeal functions have been satisfactory in the remaining five cases (see Fig 7)

A nasopharyngeal catheter is used for continuous suction of saliva for the first few days postoperatively

POSTOPERATIVE REHABILITATION

If no stent has been used, the patient begins to swallow in seven to ten days with a smaller but plugged tracheostomy tube in place Within a few days it is usually possible to remove the feeding tube and the tracheostomy tube as well

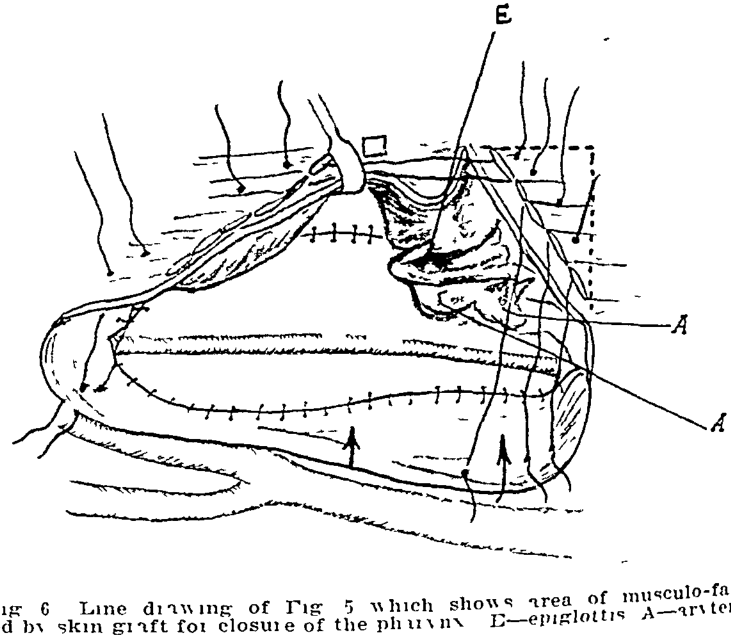

Fig 6 Line drawing of Fig 5 which shows area of musculo-fascial lined by skin graft for closure of the pharynx E—epiglottis A—arytenoid

When a graft and stent are used, approximately three weeks are required for healing and, therefore, swallowing and speaking are delayed due to the presence of the stent

The larynx has not been routinely occluded, however, the pulmonary complications have not been significant with this procedure When healing is completed the stent is removed, and the patient begins to swallow This is difficult at first but soon becomes adequate

SUMMARY

Certain cases of posterior pharyngeal wall tumors may be successfully treated by surgery but without sacrifice of the voice

A neck dissection is performed in continuity with the local excision and the defect in the pharynx is closed primarily, or by the use of skin grafts, muscle flaps and stents

Accurate localization of the tumor is essential A series of six cases is presented and discussed

The short term results in five of six patients extend to three years

CONCLUSION

A composite method of excision and immediate reconstruction is presented for the treatment of posterior pharyngeal wall malignancies allowing preservation of laryngopharyngeal function

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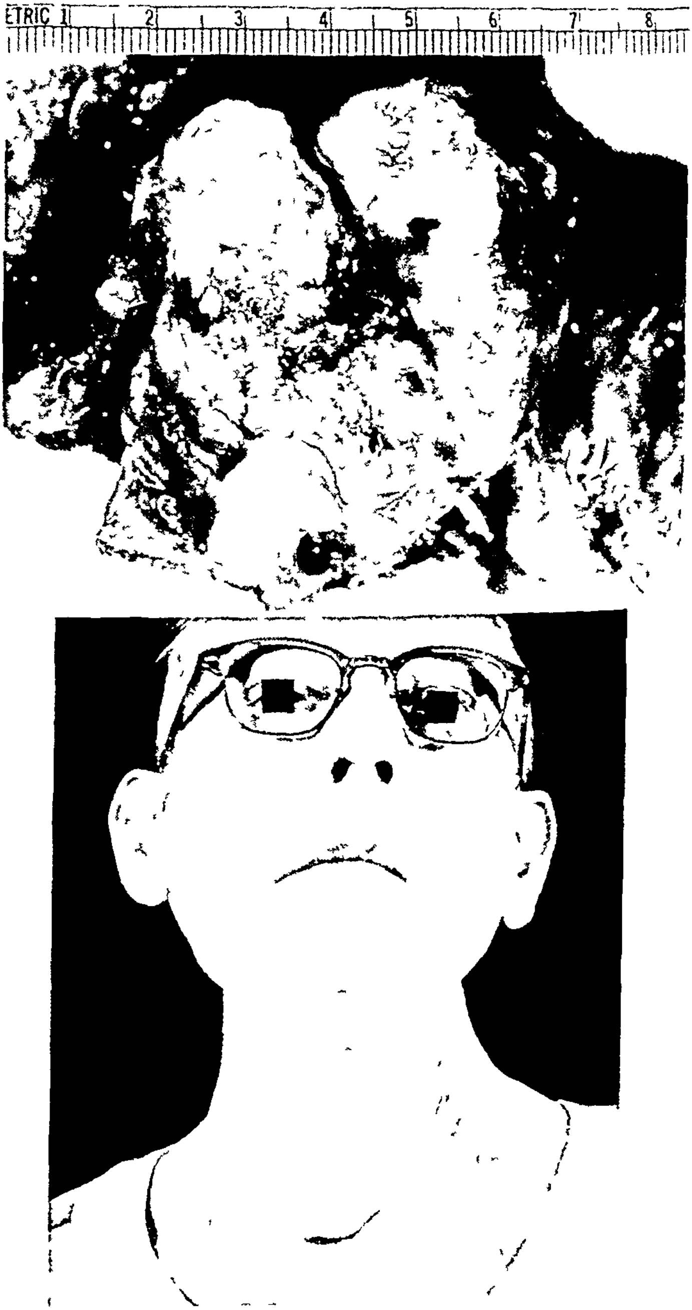

Fig 7 Specimen and postoperative photograph of patient (partial pharyngectomy and bilateral staged neck dissection)

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KAPOSI'S SARCOMA OF THE AURICLE*

R F NAUNTON, M D ,
and
F M STOLLER, M D ,
Chicago, Ill

Kaposi's[1] sarcoma is a disease entity familiar to dermatologists Since there are few recorded instances where the disease has presented itself first in those areas of special concern to the otolaryngologist, a case recently encountered by the authors is reported and discussed in detail

CASE REPORT

A 68 year-old white male cook of Greek birth attended the Ear Nose and Throat Department of the University of Chicago Clinics on February 8 1960 complaining of a slowly growing small blue lump on his right ear It had been present for the last four months and had not been associated with pain or bleeding In 1951 the patient had had similar lumps removed from a different area of the same ear at another hospital

Physical examination of the ears nose and throat revealed no abnormality except for the presence of a firm blue non tender nodule on the anterior helix of the right pinna measuring 5x3x3 mm Family history past history routine blood work including serology, and urinalysis were unremarkable A tentative diagnosis of chondroma or hemangioma was made On February 11, 1960 an excisional biopsy was carried out under local anesthesia The incision healed well and sutures were removed on the fifth day

Pathology

Inspection of the gross surgical specimen which measured 1 4x1 0x0 6 cm suggested that the tumor was well circumscribed The tumor itself was 0 5 cm in diameter felt firm and nodular and appeared to be partly encapsulated The cut surface of the specimen was tan in color smooth and homogeneous in texture

Microscopically the lesion consisted of one large fairly distinct nodular mass and two satellite nodules separated from the main mass by incomplete fibrous tissue trabeculae The lesion which was limited to the subepidermis, was circumscribed and readily distinguishable from the adjacent dermal connective tissue from the slightly thinned out but intact epidermis above, and from the uninvolved cartilage below The tumor consisted of fibroblasts which tended to be plump and only occasionally assumed a marked elongated shape There were occasional mitotic figures and erythrocytes were observed in the interstitial spaces of the tumor and in its numerous thin walled vascular channels Local

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*From the Section of Otolaryngology of the University of Chicago

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ized accumulations of hemosiderin were demonstrated throughout the tumor when sections were stained for iron. These accumulations were most numerous at the periphery of the tumor.

The gross and microscopic picture described led to a pathological diagnosis of Kaposi's sarcoma. This diagnosis prompted a more detailed investigation of this case. The patient's entire skin was scrutinized, but the only additional lesions found were two small areas of blue coloration one on the helix of the left ear, the other on the lower lip, each were about 1 mm^2. Extensive clinical pathological examinations were also made, including studies of liver function, blood nonprotein nitrogen, plasma proteins and alkaline phosphatase, all examinations gave results

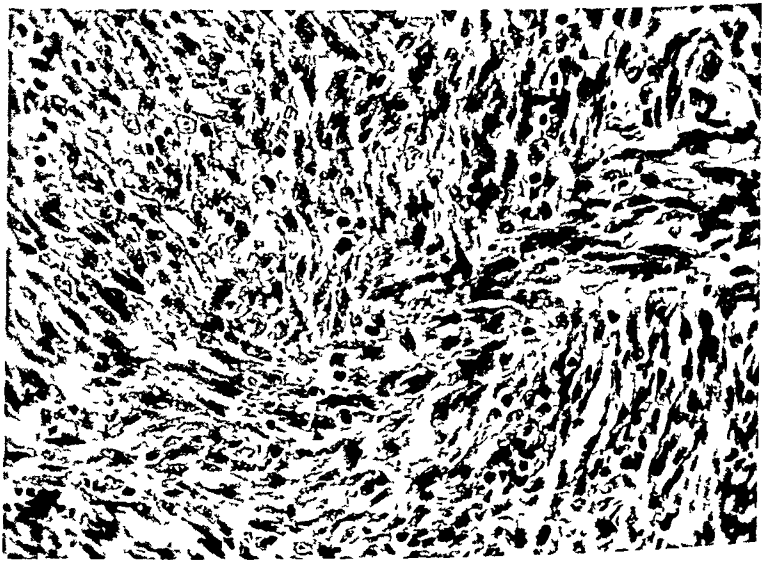

Fig 1. Main tumor and two adjacent smaller tumors separated by fibrous trabeculae. The tumors are limited to the dermis and do not involve epidermis or cartilage. (25X H and E.)

within normal limits. The differential white cell count showed a 10 per cent monocyte and 11 per cent eosinophil count while total red and white cell counts and hemaglobin were within normal limits.

Three small painless tumors had been excised from the medial aspect of the same auricle nine years earlier. Examination of the tissues removed revealed the same pathological features as those found in the recent specimen.

DISCUSSION

The etiology of this disease, first described by Kaposi[1] in 1872, is still a matter of controversy. The tumor has been said, by various authors at various times, to originate from a number of different cell types including fibroblasts,[2] perithelial and endothelial cells, myoneural structures of the glomus[3] and reticulo-endothelial elements.[4] Most investigators agree that the tumor is usually multicentric in origin and may metastasize in its advanced sarcomatous stage. It primarily affects blood and lymph vessels and most commonly arises in the dermis of the skin, changes in the epidermis are secondary. Visceral growths without accompanying cutaneous manifestations are well recognized.[5]

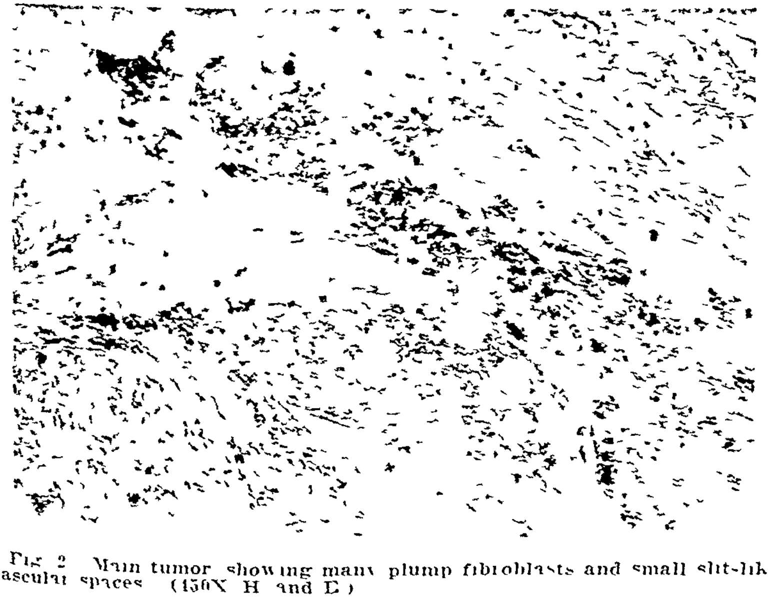

Fig. 2. Main tumor showing many plump fibroblasts and small slit-like vascular spaces. (150X. H. and E.)

Kaposi's sarcoma occurs predominantly in males in the sixth and seventh decades of life,[6] a large percentage of sufferers are of central European stock, and the disease is most common in manual laborers.[7] The tumor usually involves the extremities and is often bilaterally symmetrical. It starts as a bluish, bluish-red, or reddish-brown nodule or plaque and is slowly and intermittently progressive. A number of these plaques may coalesce and reach large proportions. The lesions are usually painless, and ulceration is rare. Edema of the extremities may develop as a result of obstruction to lymph flow In decreasing order of frequency, Kaposi's sarcoma involves the skin, gastro-intestinal tract, external genitalia, lymph nodes, liver, lungs, then almost every other organ of the body.[5]

Blood studies are normal in the majority of cases, however, increases in circulatory monocytes and eosinophils, as found in the authors' case, have been reported[4,5,6] Dorffel[4] explains this finding by theorizing that monocytes are released into the blood stream in increased amounts from the endothelial cells lining the blood vessels of the tumor, these monocytes may or may not derive from the tumor's monocytic perivascular infiltrate

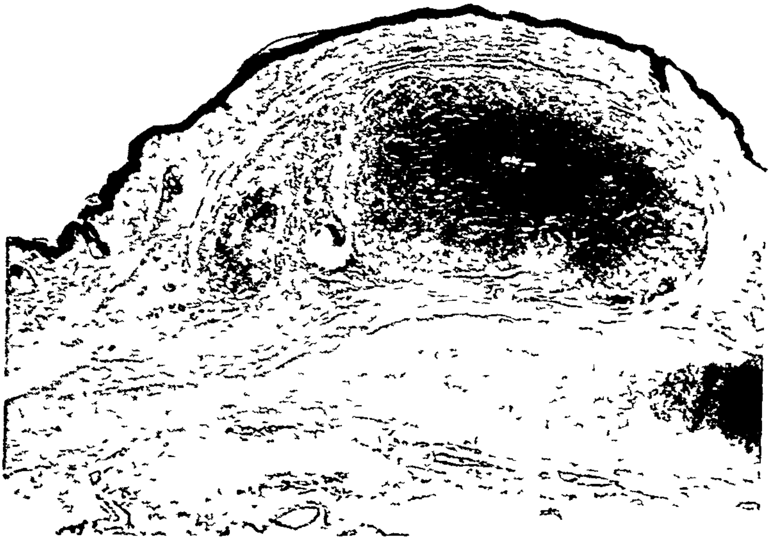

Fig 3 Section from the periphery of the main tumor demonstrating the characteristic abundance of iron pigment (145X Prussian Blue)

Other systemic diseases have been observed in association with Kaposi's sarcoma although no definite relationships have been established These include mycosis fungoides,[9] lymphatic leukemia,[10] lymphosarcoma,[11] Hodgkin's disease[5] and even carcinoma Diabetes mellitus also commonly occurs in patients with Kaposi's sarcoma [12]

Reports of the duration of life, after initial appearance of the tumor, vary from one month[2] to 48 years[13] with average figures of from five to ten years [4, 14]

Dorffel[4] claims that the microscopic development of Kaposi's sarcoma may be divided into five stages The earliest changes are engorgement and dilatation of blood vessels, hemorrhage, and deposition of hemosiderin Then follows perivascular infiltration of monocytes, histocytes and plasma cells, proliferation of blood vessel endothelium occurs later, with the formation of imperfect capillaries In the third stage angiomatous features predominate, and the tumor may resemble a cavernous hemangioma, spindle cells may also proliferate and lead to sarcoma-like appearance In the fourth stage the microscopic appearance is that of an angiosarcoma or fibrosarcoma with numerous cells showing mitotic activity Occasionally a fifth stage, one of involution, is reached, this stage is characterized by vacuolization of nuclei, cellular fragmentation, and fibrosis All developmental stages of this tumor may be found adjacent to one another in a single specimen whether it be from the skin or from a visceral organ [15] Hemosiderin is always present in these tumors, a feature distinguishing them from true fibrosarcoma

Radiation therapy has been considered the treatment of choice since the tumor is radiosensitive [14] Superficial lesions have been treated successfully with low voltage at a dosage rate of 75 r weekly for four to 12 treatments [6] Arsenicals, given orally or intramuscularly, may be helpful [6, 14] In the case presented, the lesion was removed surgically because a pathological tissue diagnosis was sought

It is most unusual for this disease to originate on the auricle, examination of the literature revealing only three such cases [2, 16, 17] Otherwise many of the clinical features commonly found in Kaposi's sarcoma were present in the case presented, *i e*, age, sex, place of birth, type of work, and relative monocytic and eosinophilic leucocytosis The nine year interval between surgical removal and reappearance of an adjacent tumor suggests multicentric origin rather than metastasis flow In decreasing order of frequency, Kaposi's sarcoma involves the skin, gastro-intestinal tract, external genitalia, lymph nodes, liver, lungs, then almost every other organ of the body.[8]

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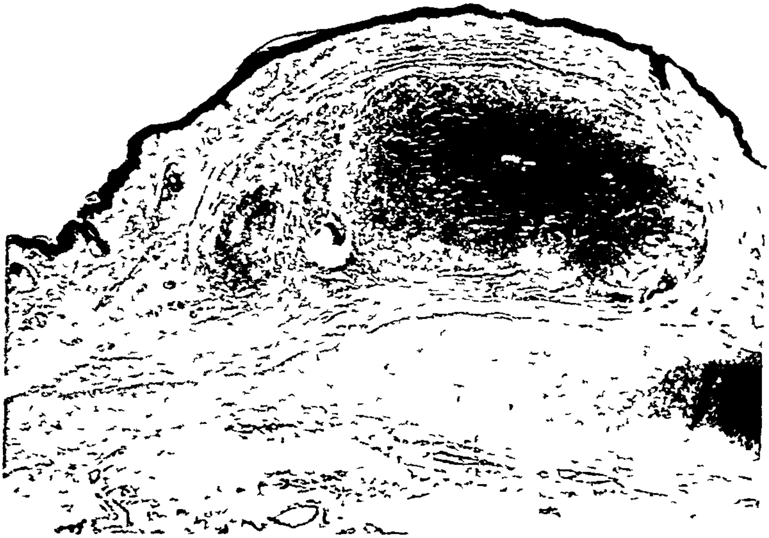

Fig 3 Section from the periphery of the main tumor demonstrating the characteristic abundance of iron pigment (145X Prussian Blue)

into the blood stream in increased amounts from the endothelial cells lining the blood vessels of the tumor, these monocytes may or may not derive from the tumor's monocytic perivascular infiltrate

Other systemic diseases have been observed in association with Kaposi's sarcoma although no definite relationships have been established These include mycosis fungoides,[9] lymphatic leukemia,[10] lymphosarcoma,[11] Hodgkin's disease[7] and even carcinoma Diabetes mellitus also commonly occurs in patients with Kaposi's sarcoma [12]

SPEECH AUDIOMETRY A DISCRIMINATION TEST FOR SPANISH LANGUAGE*

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A test of hearing function may have three main purposes *1* diagnosis and prognosis of ear disease, *2* evaluation of medical, surgical and/or hearing-aid therapy, *3* determination of hearing impairment Pure-tone audiometry is one kind of test for these purposes, but its value is limited, especially in the diagnosis of certain types of deafness, in therapeutic evaluation, and in hearing-aid recommendation

Tests using speech as the auditory stimulus are valuable because *1* they confirm the clinical findings of the pure-tone audiogram, *2* they give diagnostic and prognostic information not given by the pure-tone test, *3* they give a more valid estimate of practical hearing in everyday life[10] In general, if word lists are presented to a listener through earphones or a loudspeaker, the percentage of correct responses increases as intensity increases That intensity at which the subject can repeat correctly 50 per cent of the words given is known as the threshold of intelligibility As the intensity is further increased, the percentage of correct answers will increase proportionally until it reaches the maximum level, called the discrimination score The graph showing the percentage of correct words on the ordinate as a function of intensity on the abscissa is known as the *articulation curve* (see Fig 2)

CLINICAL TESTS

There are two types of auditory tests using speech as a stimulus that are well known and used clinically the first determines the threshold of intelligibility, or the level at which

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SUMMARY

A case of Kaposi's sarcoma is presented in which the only lesion, one of about four months' duration, was found on the auricle Similar lesions had been excised from a different area of the same auricle nine years previously The symptoms, physical signs, histological features and etiology of the disease are discussed in brief

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950 East 59th Street

later as an appendix in his book "Lecciones de Audiometria" in 1949[11] In this work, which I consider a foundation of Latin American audiology, Dr Tato makes a very thorough study of Spanish phonetic composition and concludes that *1* the Spanish language has typically bisyllabic, tetraphonemic words, with a slight predominance of the consonants over the vowels (one vowel to 1 12 consonants), *2* it is very difficult to make a monosyllabic word list in Spanish because of the lack of monosyllabic nouns, *3* a list of trochaic words correlates well with the English spondees as has been demonstrated by oscillographic studies[11]

Based on these conclusions and early work in English on the subject, Dr Tato constructed three types of lists

a Twelve phonetically-balanced lists with 25 trochaic words in each

b Five lists of 15 trochaic, disyllabic words each, not phonetically balanced and used to determine the intelligibility of the consonants relative to the vowels

c Three lists of 50 monosyllabic words each, without phonetic balance, which are "difficult at low levels"

Using his phonetically-balanced lists of bisyllabic words, Dr Tato then determined the articulation curve for Spanish on five listeners at Central Institute for the Deaf Comparing the curve obtained for Spanish disyllabic words with the one obtained for English with monosyllabic words, Dr Tato concludes "In the same circumstances we obtain an equal percentage of English with 10 db less intensity Thus a given person would be more deaf to English than to Spanish"

There are some criticisms to make of Dr Tato's work in the first place, there is no clear specification of the clinical use for the material[9] Second, the articulation curve depends, among other things, on the kind of material used (*i e* the greater the number of syllables in the word, the steeper the curve will be) Thus Dr Tato's conclusions relative to the steepness of the Spanish curve could be due to the different speech material used in the test The articulation curve is the normal subject correctly repeats 50 per cent of the material. Conventionally this intensity level is considered 0 db hearing level for speech. The deviations from this reference yield *hearing level for speech* for different individuals. In many languages the speech material considered best for this threshold test is that which makes the steepest articulation curve and contains items homogeneous with respect to intelligibility. In English, the ideal material consists of two syllables with equal stress on both (spondees). The second type of test, known as the articulation or discrimination test, gives the maximum score of correct words at a high intensity. This test, at least in the United States, has proved to be of great value in differentiating a conductive from a nonconductive hearing loss, besides giving other diagnostic and prognostic information. This paper deals with the discrimination test as it has been constructed in several languages, including Spanish, and presents a new kind of Spanish speech material.

EXISTING SPEECH TESTS

A review of the speech tests in nine languages—English, German, Danish, Swedish, Hebrew, Italian, French, Finnish, and Portuguese—shows that more or less the same criteria are used in the construction of test lists for discrimination.[4]

a. Monosyllabic structure.

b. Equal phonetic composition.

c. Representative distribution of sounds of the given language (phonetically balanced).

d. Equal range and average difficulty.

e. Words of common usage.

In recent years a new criterion has been added in the United States in order to have a more useful hearing test.[, 10]

f. Intelligibility of the speaker.

In Spanish, very little has been added since Tato's work published in *Laryngologica*, Volume I, Number 1, in 1948, and

TABLE I

Percentage of Correct Responses
List No 1

	Per Cent at 20 db	Per Cent at 30 db	Per Cent at 40 db	Per Cent at 50 db	Per Cent at 60 db
Subject No 1	20	24	78	72	90
Subject No 2	12	28	74	90	94
Subject No 3	66	52	80	96	98
Subject No 4	0	4	60	82	80
Subject No 5	8	22	82	92	92
Subject No 6	50	58	74	78	82
Subject No 7	24	60	84	84	88
Subject No 8	4	28	72	84	88
Subject No 9	18	62	70	90	94
Subject No 10	4	56	80	94	90
Subject No 11	22	72	86	88	94
Mean	21	42	76	86	90

TABLE II

Percentage of Correct Responses
List No 2

	Per Cent at 20 db	Per Cent at 30 db	Per Cent at 40 db	Per Cent at 50 db	Per Cent at 60 db
Subject No 1	48	22	80	86	94
Subject No 2	4	20	78	86	100
Subject No 3	36	70	90	96	96
Subject No 4	0	6	54	88	94
Subject No 5	16	40	74	94	96
Subject No 6	14	52	82	80	78
Subject No 7	12	50	80	90	94
Subject No 8	14	38	58	94	92
Subject No 9	14	40	78	88	94
Subject No 10	40	48	78	94	96
Subject No 11	36	80	90	94	96
Mean	21	42	77	90	94

the interest of the listener on the intelligibility of the specific phonetic elements, and since the words are meaningless, they are in no way dependent upon the vocabulary of the listener, *3* it is easy to construct lists of phonetically-balanced nonsense syllables, *4* the Spanish language is essentially phonetic, which premits the subject to transcribe graphically the sounds he hears

The criteria established for a discrimination test were *1* nonsense-syllable list, *2* phonetic composition representaalso influenced by the intelligibility of the speaker. It has been found that by using a speaker with good intelligibility, a steeper curve can be obtained than the one classically known for PB lists.[6]

A third criticism resulted from my experience in the Diagnostic Department of the Instituto Venezolano de la Audicion y el Lenguaje (Venezuelan Institute of Speech and Hearing) in Caracas. Initially we used Dr. Tato's phonetically-balanced lists as a discrimination test, to help in differential diagnosis of deafness. We had somewhat better success when we changed to monosyllabic lists; however, Berruecos, Farra and Fernandez (1952), working with Tato's PB lists found that the threshold of intelligibility differs only 1 db from that found by Dr. Tato in 1949.[9] Although the material we have in Spanish is adequate to determine the threshold for speech, we need new and more difficult material to utilize as a discrimination test.

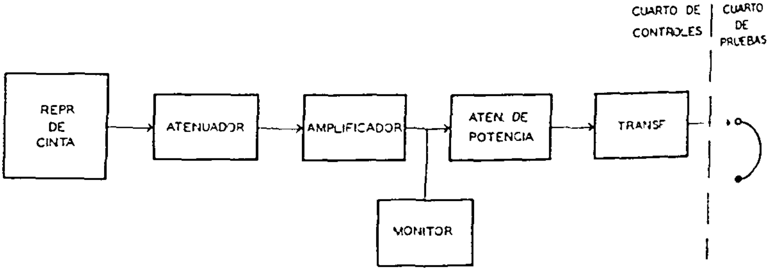

Fig. 1. Block diagram of the equipment used. Note: The speech test was actually administered to each subject in the Soundproof Room, Lester N. Hofheimer Audiology Laboratory, Department of Otolaryngology, Washington University School of Medicine, St. Louis.

A DISCRIMINATION TEST FOR SPANISH LANGUAGE

In May, 1959, we experimented with the use of nonsense-syllable lists for this purpose. We used nonsense syllables because *1* our interest in the measurement of a person's ability to hear spoken language does not require that we investigate the meaning of words[5]; *2* in the study of intelligibility, the nonsense syllable is ideal material because it focuses sonant pattern was used (example, "urp"). The phonetic composition among the lists was maintained the same.

Subjects

Eleven Spanish-speaking subjects, representing seven Latin American nationalities, were used. They varied in age from 16 to 48 years. Their hearing levels, tested on a Beltone Audiometer Model 15A, were between −10 and −5 db, relative to American Standard. All subjects but one were inexperienced with speech tests.

TABLE V

Means of All Subjects

	Per Cent at 20 db	Per Cent at 30 db	Per Cent at 40 db	Per Cent at 50 db	Per Cent at 60 db
List No 1	21	42	76	86	90
List No 2	21	42	77	90	94
List No 3	16	41	74	90	94
List No 4	14	40	71	93	93
Mean of All Lists	19	41	75	90	93

Recording of the Material

The lists were recorded by the author as speaker on magnetic tape at a speed of 15 in/sec on an Ampex Model 400 recorder. A 1,000 cps tone was recorded on each tape as a reference level. Each list was preceded by the following recorded instruction in Spanish: "You will hear immediately a nonsense-syllable list. Listen carefully and try to repeat each sound. Are you ready?" Ten seconds later the subject heard the carrier phrase "Repeat the word ———."

Method

In the first experimental session the subject was informed in detail about the nature of the test, the goals we wished to accomplish, and what would be expected of him. Then we made a brief otological examination and a pure-tone audiogram. A block diagram of apparatus used for the listening tests is shown in Fig. 1. In order to minimize the effects of learning, all four lists were first given at an intensity level of 60 db SPL, and a record was kept of the answers.

TABLE III

Percentage of Correct Responses
List No 3

	Per Cent at 20 db	Per Cent at 30 db	Per Cent at 40 db	Per Cent at 50 db	Per Cent at 60 db
Subject No 1	20	50	86	88	94
Subject No 2	2	24	78	86	92
Subject No 3	64	64	84	90	98
Subject No 4	0	6	58	88	90
Subject No 5	10	22	68	92	94
Subject No 6	14	56	82	88	86
Subject No 7	8	44	94	94	94
Subject No 8	6	24	64	86	98
Subject No 9	10	50	76	84	94
Subject No 10	8	50	84	92	100
Subject No 11	32	60	84	98	98
Mean	16	41	74	90	94

TABLE IV

Percentage of Correct Responses
List No 4

	Per Cent at 20 db	Per Cent at 30 db	Per Cent at 40 db	Per Cent at 50 db	Per Cent at 60 db
Subject No 1	46	38	64	78	84
Subject No 2	6	34	76	94	98
Subject No 3	34	62	92	94	100
Subject No 4	0	8	34	90	90
Subject No 5	10	22	70	88	96
Subject No 6	22	58	82	84	84
Subject No 7	6	50	76	92	90
Subject No 8	4	18	68	90	100
Subject No 9	12	28	77	86	92
Subject No 10	36	36	70	92	96
Subject No 11	16	82	90	94	96
Mean	17	40	71	93	93

tive of the Spanish language, 3 equal phonetic composition in all lists A criterion of difficulty was not established

Construction

Four lists of 50 nonsense syllables each were made according to a consonant-vowel-consonant pattern following the phonetic proportion established by Dr Tato in the initial consonant and the vowel, but not in the final consonant, however, in some instances, in order to include the single "r" in the proportion indicated by Tato, the vowel-consonant-con-

TABLE VI

Standard Error of Percentage for the Four Lists at the Five Intensity Levels

Lists	Levels				
	20 db	30 db	40 db	50 db	60 db (spl)
1	12.3	14.9	12.9	10.4	9.0
2	12.3	14.9	12.6	9.0	7.1
3	11.0	14.8	13.2	9.0	7.1
4	11.3	14.7	13.6	7.6	7.6
Mean	11.8	14.8	13.0	9.0	7.6

TABLE VII

Analysis of Variance for the Four Lists Related to the Five Intensity Levels

Source	df	ss	ms	F
Lists (L)	3	453.36	151.12	1.2038*
Levels (l)	4	184 252.08	46,063.02	366.919†
L × l	12	800.06	66.67	0.531*
Cells	200	25 108.18	125.54	
Total	219	210 613.68		

*Statistically insignificant
†Statistically significant as it should be expected

average for all levels. This latter result is shown graphically in Fig. 2.

Tables VI and VII show a statistical resume of the results. Table VI shows the standard error of the percentage for the four lists related to the five intensity levels. Table VII shows in summarized form the variability of the four lists related to the five intensity levels. From this analysis we may conclude that the variability among the lists is very small and their homogeneity is great. This is important from the clinical point of view because it permits us to use the lists interchangeably.

COMMENT

According to these results it can be said that the nonsense-syllable lists proved to be more difficult material than the disyllabic phonetically-balanced lists made by Dr. Tato (see Fig. 2), hence this test may be useful as a discrimination test with the diagnostic purpose of differentiating between a

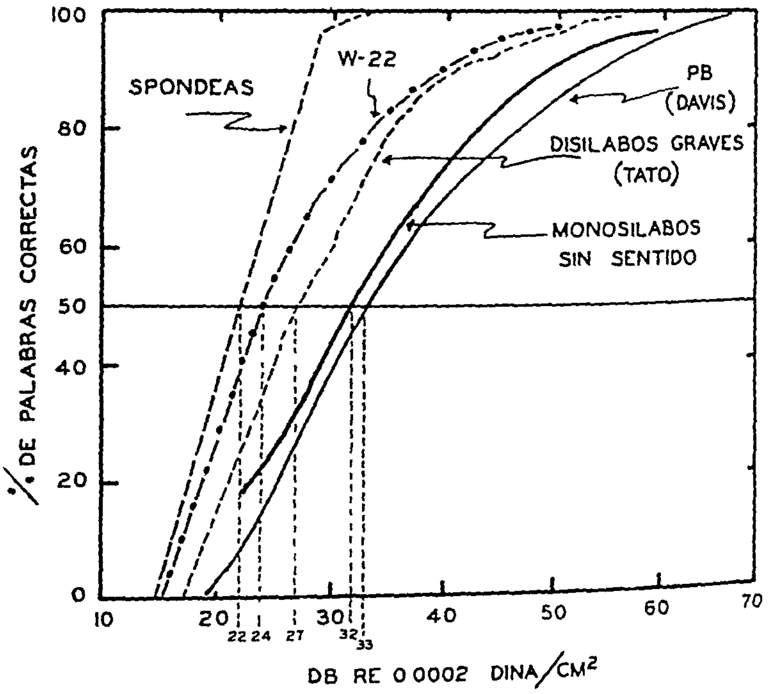

Fig 2 This graph shows the relation between the kind of speech material used and the corresponding articulation curve obtained

The curve spondees' is the classical curve obtained with English bisyllabic spondee words

The curves W-22 and PB(Davis) were both obtained with English phonetically-balanced monosyllabic word lists the 'W-22 curve being recorded by a speaker with better intelligibility

The curve 'disilabos graves(Tato)' was obtained by Dr J M Tato with Spanish phonetically-balanced trochaic word lists

The curve Monosilabos sin sentido was obtained with the nonsense syllable material discussed in this paper

During the next two (in some cases three) sessions each list was given at four different intensities 20, 30, 40, and 50 db SPL Thus each subject received the lists 16 additional times The order and intensity level at which each list was presented to each subject was randomized

RESULTS

Tables I to IV show the percentage of correct answers for each subject on each list Table V shows the means of these percentages for each list at different levels, as well as the

APPENDIX

Spanish Nonsense-Syllable Lists

List 1	List 2	List 3	List 4
1 Ses	1 Eik	1 Su	1 Ken
2 Ard	2 Mat	2 Yip	2 Sax
3 Nes	3 Sok	3 Nop	3 Dil
4 Lat	4 Nip	4 Sut	4 Neb
5 Sel	5 Uip	5 Ois	5 Kach
6 Arb	6 Sel	6 Net	6 Lib
7 Nem	7 Dap	7 Lek	7 Sok
8 Lad	8 Sun	8 Sep	8 Tex
9 Nef	9 Lab	9 Din	9 Naf
10 Sef	10 Ort	10 Kuf	10 Orx
11 Ark	11 Lep	11 Sem	11 Tet
12 Nel	12 Tok	12 Mill	12 Lad
13 Lon	13 Eis	13 Irk	13 Ers
14 Dac	14 Iik	14 Tei	14 Dot
15 Sap	15 Lod	15 Lan	15 Bal
16 Erb	16 Deñ	16 Kok	16 Kot
17 Ner	17 Tot	17 Sek	17 Pek
18 Lol	18 Sab	18 Aif	18 Sast
19 Dil	19 Ned	19 Tif	19 Ilt
20 Sat	20 Tip	20 Sab	20 Ont
21 Ern	21 Saf	21 Lod	21 Gat
22 Nag	22 Nox	22 Fan	22 Sim
23 Dur	23 Dep	23 Nach	23 Erps
24 Tis	24 Pap	24 Eim	24 Loñ
25 Sak	25 Sis	25 Dep	25 Sep
26 Tuñ	26 Pep	26 Sach	26 Dal
27 Nas	27 Tad	27 Tad	27 Sis
28 Ert	28 Dis	28 Eix	28 Fun
29 Duk	29 Esp	29 Nat	29 Nof
30 Sod	30 Yal	30 Mol	30 Tañ
31 Til	31 Kel	31 Uik	31 Dus
32 Nol	32 Gos	32 Doi	32 Ert
33 Lel	33 Unt	33 Ton	33 Jep
34 Tok	34 Kas	34 Señ	34 Fas
35 Soch	35 Elk	35 Dal	35 Esf
36 Bat	36 Dof	36 Kop	36 Med
37 Kir	37 Sil	37 Nis	37 Dap
38 Guer	38 Aix	38 Kep	38 Bek
39 Kor	39 Nek	39 Lai	39 Nir
40 Pam	40 Bon	40 Jal	40 Olp
41 Mon	41 Lit	41 Pek	41 Yex
42 Yer	42 Tai	42 Sel	42 Arp
43 Kin	43 Mep	43 Pol	43 Usp
44 Siñ	44 Nok	44 Gai	44 Pall
45 Mir	45 Ball	45 Nañ	45 Mom
46 Bor	46 Jech	46 Bet	46 Kit
47 Fes	47 Kik	47 Dax	47 Dax
48 Pos	48 Ost	48 Lu	48 Arst
49 Kad	49 Kes	49 Bol	49 Tech
50 Jin	50 Fap	50 Non	50 Sip

conductive hearing loss and a non-conductive hearing loss, however, in spite of the homogeneity shown by the statistical analysis, the value of the test in differential diagnosis can be assessed only after its use in the clinic and a careful evaluation of the results This will be the basis for future study

ACKNOWLEDGMENT

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for testing a patient is as follows for each frequency *1* determine the AC tone-pulse threshold, *2* mix the noise with the threshold-level AC tone and raise the noise level until the tone is no longer heard, *3* switch the noise to the BC vibrator, previously placed on the patient's head, and raise the noise level until the AC tone again disappears The AC tone loss found in step 1 is subtracted from the AC noise level (in db relative to the normal threshold for AC noise) found in step 2 Subtracting this difference from the BC noise level (in db relative to the normal threshold for BC noise) reached in step 3 above gives what Rainville interprets as the patient's absolute BC tone threshold for the frequency tested

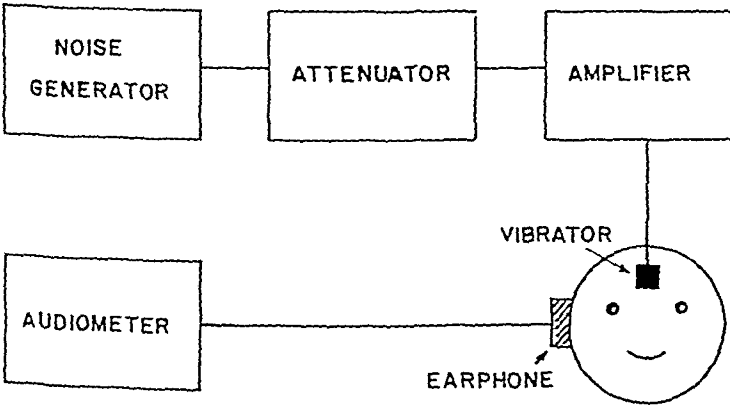

Fig 1 Diagram of apparatus required for the M-R test

The primary advantage of the Rainville procedure is that it gives little opportunity for the non-test ear to interfere with measurement of the acuity of the ear under test Moreover, the problem of cross-masking is eliminated

THE M-R PROCEDURE

The instrumental requirements for the M-R test, which are summarized graphically in Fig 1, are similar to those of the Rainville test but are simpler because there is no need to combine the noise and tone The noise channel is entirely separate from the tone channel The noise generator may be any of

THE M-R TEST OF BONE-CONDUCTION HEARING[*]

CHARLES LIGHTFOOT, Ph D ,

Chicago, Ill

Obtaining a bone-conduction threshold free of contamination by the non-test ear is frequently beyond accomplishment by means of conventional BC audiometry Even when the most elaborate formulas are followed to determine the appropriate level of masking noise, lateralization of the test signal to the non-test ear may persist—with or without the awareness of the person being tested When it is possible to mask out the non-test ear, the level of noise required is sometimes distractingly loud It may even cross over and partially mask the test ear There is no need to elaborate on contralateralization of the test signal, cross-masking and related problems associated with BC audiometry, which have been well reviewed in the literature[1,2] Of interest here is an ingenious solution to these problems provided by the BC test of Rainville[3,4] I have derived from that test a simpler, quicker procedure which I call the modified Rainville or M-R test of BC hearing It is the purpose of this paper to describe the M-R test and to enumerate its advantages and limitations

Before describing the M-R test, it may be well to review briefly the original Rainville procedure In addition to an AC audiometer capable of delivering pulses of tone, the following apparatus is required a masking-noise generator, an amplifier and attenuator for controlling the noise level, a switch which allows the noise to be delivered either to a bone vibrator or—presumably through a mixing circuit—to the earphone of the audiometer Two normative values, assuming that the audiometer is already calibrated, must be obtained before the Rainville test can be put to use the normal threshold levels for the noise, AC and BC Rainville's procedure

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[*] From the Speech and Hearing Center Department of Otolaryngology University of Illinois College of Medicine Technical contributions to the instrumentation described herein were made by Mr M P Kesnar of the center mentioned above

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the norm specified by the National Bureau of Standards fo the frequency concerned. Thus, if the obtained AC threshol were –10 db, and if the noise attenuator setting required t mask the tone at that level were 50 db, you would deduce tha a noise 10 db greater, or an attenuator setting of 40, woul be required to mask a threshold tone if the ear had conforme with the audiometric zero. For each frequency the media of several such deduced noise-attenuator settings will serv as the normative value required for the M-R test.

In M-R testing of a patient, the procedure is the same a that for getting the normative data. For each frequency yo simply determine how much the BC noise must be attenuate to interfere with the perception of AC tone pulses at threshold. You then subtract the obtained attenuator reading from th normal value. The difference is the patient's BC hearin loss.

Let us consider a few examples involving a frequency fo which we have learned that the noise attenuator must be set at 40 in order to provide just enough BC noise to mask a 0-d AC tone in the normal ear. Suppose we find that a patient ha a 30-db AC loss. We leave the hearing-loss dial set at 30 and increase the level of the BC noise—*i e*, we reduce its attenuation—by 5-db steps. Suppose that when the noise attenuato is at 40 db the patient no longer hears consistently the pulse of tone. Since the BC noise required to mask this patient i no greater than the normal amount, we learn that his BC los is 0 (= 40 – 40); hence, the entire AC loss is conductive Suppose that for another patient with a 30-db AC loss the noise attenuator reaches a setting of 25 before the AC tone is masked. We would then conclude that there is a BC loss of 15 (= 40 – 25) on which there is superimposed an additional 15-db conductive loss. Suppose that for a third patient with a 30-db AC loss the noise attenuator reaches a setting of 10 before the AC tone is masked. In this case the BC loss is equal to the AC loss, or 30 (= 40 – 10), which would impl that the entire loss is of a sensori-neural nature.

DISCUSSION

The M-R test is based upon the assumption that the mask-the several commercially available ones which provide a white noise. Saw-tooth noise generators, such as those used in most American-made audiometers, are not acceptable, for their line-hum harmonics are likely to create audible beats when transduced in the vicinity of the tone-delivering earphone. With some white-noise generators a pre-amplifier may have to be inserted in the circuit in front of the attenuator. In some situations a meter to monitor the output of the noise generator might be useful. Any stepped attenuator of the T type may be used providing it covers a range of 60 db or more; it is most convenient, however, if it is calibrated in 5-db steps. The amplifier need provide only a few watts and should have a 16-ohm or 8-ohm output tap. Although almost any commercial power amplifier would be satisfactory as far as the high-frequency end of its response characteristic might be concerned, it is of some importance to select one of uniform output down to 100 cps at the lower end. The bone vibrator may be of the ordinary hearing-aid type with which audiometers are usually furnished. With this type of vibrator the amplifier should be set to deliver about 4 volts (but not much more under ordinary circumstances) when attenuation is minimal.

As with any BC test, the M-R test requires obtaining normative data. This is relatively easy for only a few normally hearing persons with no history of ear pathology are needed; moreover, any reasonably quiet room will serve. In the case of each normal subject, you place the vibrator holder along the sagittal line so that the vibrator is held firmly on the center of the forehead. Then you set the headphone in its usual place and determine the AC threshold, which is defined here as the lowest level at which pulses of a tone presented at a rate of two per second can be heard without strain—not 50 per cent of the pulses but virtually all of them. Up to this point, although the noise generator should have been turned on, its output has been fully attenuated. Now, leaving the uninterrupted AC tone at threshold level, you decrease the attenuation of the BC noise until it just masks the AC tone. From the attenuator setting thus reached you deduce the setting which would be required if the ear's threshold were equal to cerned Thus, the first two examples cited in the preceding paragraph are not quite accurate for test frequencies below 1000 cps If the first example involved the test frequency of 500 cps, the hypothetical case with unmixed conductive loss would be more likely to yield an M-R threshold of 5 db than the zero amount previously stated

The reliability of the M-R test may prove to be slightly lower than that of conventional BC audiometry because of the shape of the masking function The reader will recall that when masking in db is plotted as a function of noise level, the curve rises elliptically at its foot and does not approach rectalinearity until the noise level is high enough to effect several db of masking[6] That is, raising the noise level 5 db above the level required to shift the threshold of a tone by a fraction of a db will bring less than 5 db of additional shift Day-to-day variability in M-R test results may be reduced if, in obtaining normative data as well as in testing patients, the AC tone is set at 5 db above threshold while the interfering noise level is obtained When using an ordinary vibrator, I do not routinely follow this altered procedure because it lowers the maximum degree of sensori-neural loss which can be measured

The maximal sensori-neural loss which can be measured by the M-R test, as by any other, varies as a function of frequency At any single frequency the primary variable governing the maximum, other than gain of the amplifier, is the bone vibrator With a maximum of 4 volts available some vibrators (I refer to ordinary hearing-aid types placed on the forehead) will allow measurement of a 60-db loss at 1000 cps, others will only handle a 40-db loss The greatest losses measurable at 500, 2000 and 4000 cps are likely to be 5 or 10 db lower than at 1000 cps It is difficult to find a vibrator, from ordinary sources, which will allow measurement of losses at 250 cps in excess of 15 or 20 db Greater losses can be handled by increasing the maximum voltage, however, such action may produce non-linearity requiring application of corrections to the attenuator readings and may ultimately result in damage to the ordinary vibrator Use of narrow-band filters would overcome these difficulties ing process in impaired ears is similar to that in normal ears. There is some reason to doubt that this assumption is completely valid.[illegible] So far, however, in using the M-R test, I have not come across any pathological ears with masking characteristics abnormal enough to interfere seriously with the accuracy of the test.

Despite Rainville's interpretation of his test as one of absolute BC, I regard the M-R as a test of relative BC. This is not to say that the occlusion of the external canal by the AC earphone has no effect; rather, the effect on the threshold is the reverse of that which accompanies sinusoidal vibration of the skull by the test tone as in conventional BC testing. With occlusion in the latter, at least with respect to the lower frequencies, the AC tone created in the canal, presumably as a result of mandibular inertia and consequent deformation of the cartilaginous portion, is more intense and hence, reinforces the BC signal more than it would were there no occlusion; therefore, unless a conductive lesion is present to block the reinforcing AC tone, the BC threshold is lowered. Occlusion of the canal by the earphone in the M-R test results in reinforcement not of the tone but of the noise, unless there is a conductive impairment. The influence of such reinforcement in M-R testing of a normal ear or of one with only sensori-neural limitation is the same as that which prevailed during the procurement of the normative data; hence in M-R testing of such ears no correction for occlusion need be made in the determination of the BC threshold. In M-R testing of a conductively impaired ear, however, the role of the AC noise created in the canal is negligible, hence, the BC vibrator has to be driven with greater intensity to effect masking than was required in establishing the norm. In other words the BC threshold obtained by means of the M-R test is somewhat higher than it should be when there is an appreciable conductive lesion, whether or not there is also sensori-neural loss. The amount of error in testing a conductively disturbed ear sometimes is as much as 10 db at 250 and 500 cps but is seldom more than 5 db at higher frequencies. The degree of conductive impairment does not appear to be critical as far as magnitude of the error is concerned Thus, the first two examples cited in the preceding paragraph are not quite accurate for test frequencies below 1000 cps If the first example involved the test frequency of 500 cps, the hypothetical case with unmixed conductive loss would be more likely to yield an M-R threshold of 5 db than the zero amount previously stated

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Several possible objections to the M-R procedure have been discussed Let me now briefly enumerate the several conspicuous advantages of the M-R test *1* lateralization to the non-test ear is not possible, *2* standardization can be accomplished easily on a few normal ears without an extraordinarily quiet room, *3* the test can be performed more quickly than can other procedures, including the conventional BC procedure as well as that described by Rainville, *4* either the poorer or the better ear can be tested first, *5* the perception required of the patient is a simple one, *6* no elaborate instructions to the patient are necessary, *7* instrumentation is not complicated

After having used the M-R test on a large number and wide variety of hypacusic patients and having weighed its advantages against its limitations, I recommend it for clinical determination of BC auditory acuity

SUMMARY

A modification of Rainville's method of measuring BC threshold is described and called the M-R test The M-R test consists in determining, for each test frequency, the lowest level of BC noise which interferes with the hearing by the ear under test of AC tone pulses at threshold The difference between this noise level and the level of BC noise which has the same interfering effect on a normal ear is taken as the BC threshold of the ear under test Possible difficulties in the administration and interpretation of the M-R test are discussed, and its particular merits are mentioned It is concluded that the several advantages of the M-R test warrant its recommendation

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ANNUAL SPRING MEETING OF THE NEW YORK EYE AND EAR INFIRMARY ALUMNI

The Annual Spring Meeting of the Alumni Association of the New York Eye and Ear Infirmary will take place April 17-19, 1961

Symposia will be offered on Otosclerosis and Benign Lesions of the Larynx There will also be a Closed Circuit Television demonstration of surgical procedures In addition there will be lectures on Plastic Surgery, Paranasal Sinuses and Carcinoma of the Head, Neck and Larynx

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CORRECTION

In the October, 1960, issue, page 1451, the last paragraph states that the Michelson drill is manufactured by Storz Instrument Co of St Louis This should read "This drill is distributed by Storz Instrument Co, St Louis, Mo"

DIFFERENCES IN DIFFICULTY AMONG THE CID W-22 AUDITORY TESTS

BARRY S. ELPERN, Ph.D.,

Chicago, Ill.

The importance of auditory speech-discrimination tests in the assessment of hearing dysfunction is attested by the great amount of clinical and experimental work which has involved such tests. The most widely used discrimination measure, at present, is the W-22 Auditory Test series developed by Hirsh and his associates[3] at Central Institute for the Deaf. This series, recorded on 12-inch unbreakable discs, comprises four, 50-word lists, each of which is recorded in six different word orders, designated A, B, C, D, E, and F. The clinical popularity of W-22 is undoubtedly attributable to the fact that the discs are commercially available and offer a simple, more-or-less standardized form of presentation.

Through preliminary trials, Hirsh sought to determine whether differences in difficulty existed among the four main lists. He examined three groups of five normal-hearing subjects and, on the basis of articulation functions yielded by these groups concluded (p. 333[3]):

> *These data show no consistent differences between scores on the four different lists. All listeners were given ample opportunity to study alphabetical lists of the words, however, and heard scramblings of each list at least three times. In a shorter clinical procedure where listeners may hear scramblings of two different lists once, some sort of differences between lists may appear from listener to listener. It can only be said that the averaged data of several listeners from several tests showed no consistent difference between lists—these results await verification from several clinics using a large number of listeners.*

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication April 1, 1960.

To the writer's knowledge, such verification has not materialized, at least, not in the form of a published report

On the basis of the initial trials, the four test lists are assumed to be, and are employed as though they are, equal in difficulty, therefore, it seems worthwhile to examine this assumption using a much larger group of subjects in order to provide broader evidence for or against the prevailing indiscriminate intercomparison of scores obtained by means of these tests

TABLE I

Distribution of Monaural Discrimination Test Scores

VA Audiology Clinic	List 1	List 2	List 3	List 4
Washington	32	26	21	21
Chicago	146	150	217	212
New York	81	76	7	10
Los Angeles	42	51	29	26
San Francisco	24	52	55	42
Dallas	73	70	16	11
List Totals	398	425	345	322
Grand Total—1,490				

PROCEDURE

A pool of 1,490 monaural discrimination scores was accumulated with the assistance of the clinical staffs of VA Audiology Clinics in Chicago, New York, Washington, Dallas, San Francisco, and Los Angeles All measurements were obtained through the use of recorded W-22 50-word lists, however, scores yielded by patients in any way suspected of having a non-organic hearing loss component were disregarded The raw data for each ear were simply the specific list used, *e g*, 2-F, and the number of words *misunderstood* The number missed was then converted to percentage of discrimination loss by multiplying by a factor of 2

The test ears were distributed in random fashion among four groups, one for each of the four W-22 lists Any difference found between two groups was assumed to be analogous to a difference between the lists to which these groups were *exposed* The distribution of monaural tests by VA Clinic and by W-22 list is shown in Table I In view of the numerical size of the groups and the geographical dispersion of the measurements, it was felt that potentially pertinent factors, such as age and hearing loss types, would tend to be inter-equated

Raw data were analyzed by applying "F" and "t" tests to the difference between variances and means, respectively, of each of the six possible pairs of groups 1 and 2, 1 and 3, 1 and 4, 2 and 3, 2 and 4, and 3 and 4 Pairs whose variances differed significantly were evaluated with respect to the difference between their means by applying the Cochran and Cox[1] approximation reviewed in Edwards[2] (p 274) The primary consideration, for the purposes of this study, was the evaluation of differences between means of each of the several pairs of groups For any given pair, a significant difference between means was interpreted as a significant difference in average difficulty, with the greater mean representing the more difficult of the two lists under comparison

TABLE II

Basic Statistical Data for the Four Groups Employed in This Study

Group (List)	N	Mean Discrimination Loss (M)	SE of M	Variance	Standard Deviation (σ)	SE of σ
1	398	13 62%	± 700	194 379	13 942%	± 494
2	425	15 64%	± 836	296 322	17 214%	± 590
3	345	16 97%	± 979	329 931	18 164%	± 691
4	322	18 30%	±1 033	342 546	18 508%	± 729

RESULTS AND DISCUSSION

Basic statistics derived from the raw data appear in Table II Inspection of this Table reveals two things of primary interest *a* the widest difference between means is *less than* five per cent, and *b* the widest difference between standard deviations is also less than five per cent. Insofar as the means and standard deviations are indices of average *level* of difficulty and average *range* of difficulty, respectively, the observed maximum disparities appear to be well within the limits of tolerance acceptable in clinical procedures, therefore, visual evaluation of the data seems to warrant the conclusion that the differences noted are, from a practical standpoint, non-significant, and that they may be disregarded in clinical practice It is, in other words, difficult to visualize a situation in which differences such as those mentioned would critically influence a clinical decision

TABLE III

Statistical Evaluation of the Difference Between Variances (s^2) for All Pair Combinations of Four Experimental Groups

The F value is the ratio of the greater variance to the lesser and the entry in column P indicates the probability of occurence of an F ratio as large as that obtained

Pair of Groups (Lists)	Greater s^2	Lesser s^2	F	df	P
1 and 2	296 322	194 379	1 524	424/397	< 02†
1 and 3	329 931	194 379	1 697	344/397	< 02†
1 and 4	342 546	194 376	1 762	321/397	< 02†
2 and 3	329 931	296 322	1 113	344/424	> 10*
2 and 4	342 546	296 322	1 156	321/424	> 10*
3 and 4	342 546	329 931	1 038	321/344	> 10*

*Not significant
†Significant

It is not unlikely, however, that in certain research applications, inter-list differences would be intolerable; therefore, to determine the suitability of the lists to the relatively rigorous standards adopted for research purposes, the differences between means and variances of the several pairs of lists were statistically evaluated Five per cent (05) was adopted as the cutoff probability value (P) This is to say that the difference between any pair of means or variances associated with a P less than five per cent (< 05) was considered significant, a difference associated with a P greater than five per cent (> 05) was considered not significant A summary of the evaluation of differences between variances appears in Table III

Significant variance ratios (F) occur only for pairs of lists of which one member is List 1 It has already been shown in Table II that the variance associated with List 1 is smaller than those of the remaining three lists, therefore, the range of difficulty of List 1, indicated by its variance, is significantly smaller, or more restricted than that of any of the other lists, which, among themselves, are relatively equal in range of difficulty

The evaluation of differences between means is summarized in Table IV. Just as a significant difference between vari-

TABLE IV

Statistical Evaluation of the Difference Between Means for All Pair Combinations of Four Experimental Groups

The t value is the ratio of the arithmetical difference between means to the standard error of this difference, and the entry in column P indicates the probability of occurrence of a t ratio as large as that obtained

Pair of Groups (Lists)	Difference Between Means	SE of Diff	t	P
1 and 2	2 02%	1 089	1 855	> 05*
1 and 3	3 35%	1 202	2 787	< 01†
1 and 4	4 68%	1 246	3 751	< 01†
2 and 3	1 33%	1 286	1 036	> 05*
2 and 4	2 66%	1 328	2 003	< 05†
3 and 4	1 33%	1 421	931	> 05*

*Not significant
†Significant

ances, denoted by the F value, indicates a systematic difference in range of difficulty, a significant difference between means, denoted by the value of t, indicates a systematic difference in average level of difficulty On the basis of t scores reported in Table IV, it is concluded that Lists 1 and 2, 2 and 3, and 3 and 4 are relatively equal in average level of difficulty, but that Lists 1 and 3, 1 and 4, and 2 and 4 are significantly different in this respect

SUMMARY

The foregoing analysis has illustrated the existence of systematic differences both in average level of difficulty and in average range of difficulty, among the four W-22 word lists It was the writer's opinion that the magnitude of the observed differences is such that they may be disregarded in clinical practice, however, in view of the fact that these differences are, nonetheless, sources of systematic bias, it became necessary to evaluate them with regard to experimental requirements The evaluation of the differences between means and variances for all possible pairs of lists indicates that the only pairs which do not differ in either respect are Lists 2 and 3, and Lists 3 and 4 These pairs of lists, then, may be employed as speech stimuli in laboratory studies without introducing bias resulting from differences in difficulty

The writer acknowledges the helpful criticism of Dr James Jerger, Northwestern University, and the co-operation of the chiefs and audiological staffs of the VA audiology clinics mentioned in the text

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THE MOUNT SINAI HOSPITAL POSTGRADUATE COURSE

In order not to conflict with the International Congress of Otolaryngology to take place in Paris in July, 1961, the annual intensive postgraduate course in Rhinoplasty, Reconstructive Surgery of the Nasal Septum and Otoplasty will be given January 7-20, 1961, by Dr Irving B Goldman and staff at the Mount Sinai Hospital, New York, in affiliation with Columbia University

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AIR DRIVEN HAND PIECE FOR TEMPORAL BONE SURGERY*

ROBIN P MICHELSON, M D ,

Redwood City, Calif

The instrument is an air operated hand piece which has many advantages over the conventional electric engine belt driven hand piece presently in use Among the many advantages are the fact that this is an extremely flexible instrument and can be driven at extremely high speeds or very low speeds and is controlled with the finger tip rather than with the foot It is also extremely flexible in that there are no arms or other attachments to the hand piece except a small flexible plastic hose which supplies the air to the hand piece The air driven hand piece develops considerably greater torque and speed than any available motor drive It is almost vibrationless, is quiet and runs cool

The instrument consists basically of a small turbine into which can be plugged either a straight or an angled drive hand piece The burrs which screw into the ends of the hand pieces are interchangable There is also a control box through which the air passes This consists of a small filter that takes out small particles of dust and water in the air, a pressure reduction valve and a pressure gauge By setting the air pressure to the hand piece the pressure reduction valve can control its maximum speed for any given depression of the trigger on the side of the hand piece The trigger on the side of the hand piece not only stops and starts the turbine, but also controls its speed

At 60 pounds of static air pressure the turbine itself will develop 175,000 revolutions per minute free running, and since it is fitted with a small reduction device, the spindle speed is around 60,000 revolutions per minute at this pressure

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*From the Department of Otolaryngology Stanford University School of Medicine Palo Alto Calif

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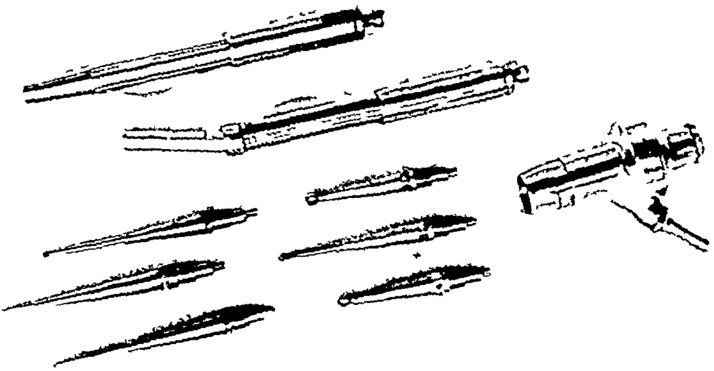


Fig 1 Straight hand piece angle hand piece turbine and assorted burrs

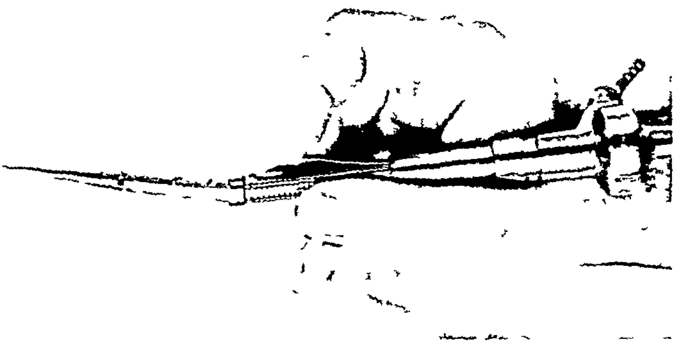


Fig 2 The angle drive plugged into the turbine with a ½ mm diamond stone

This can be continuously varied to 200 or 300 revolutions per minute

The burrs are mounted in tapered bushings, and the support of these bushings is carried as close to a cutting head of the burr as is possible The large cutting burrs are supported clear out to the head On some of the smaller ones, it has been necessary to allow the shaft to extend for some distance beyond the end of the bushing The burrs' shafts automatically engage the spindle shaft with a positive locking device when the burrs are screwed into the end of the hand piece This eliminates not only a tendency for the burrs to run eccentrically, but also eliminates the slip of the conventional collet clutch used in other types of hand pieces

The instrument is sterilized either in a hot oil bath or in dry air. Steam sterilization and water sterilization are to be avoided since the delicate, extremely fine surfaces of the ball

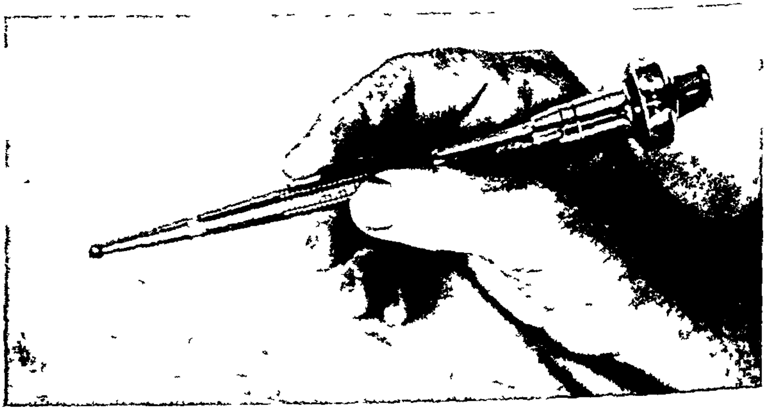


Fig 3 Straight hand piece plugged into the turbine with Jordan Day cutting burr (Note the index finger on the turbine throttle)

bearings upon which the shaft and rotor turn can be easily damaged or etched by moisture and would lose their close tolerances

In use the author has found that the high speed is very useful with the Jordan-Day cutting burr, and that one uses very little pressure against this burr to accomplish a great deal in exenterating mastoid cavity For finer delicate cutting, one can use lower speeds and a smaller finely tapered burr, thus, it is possible to thin out the footplate of the stapes with the half millimeter diamond burr just barely turning over.

A question was raised during the development of this instrument regarding the sterility of the exhausted air and the possibility that the air exhausts might be a possible source of contamination Accordingly, the air from the exhaust was blown against a blood agar plate for a period of 10 minutes, this representing about 40 cubic feet of air No growth occurred on two consecutive tests This would seem to allay this fear of a possible source of contamination

39 Birch Street

POSTGRADUATE COURSE IN ALLERGY

A continuous course of two weeks' duration is being offered by the Departments of Allergy and Applied Immunology of the Temple University Medical Center and the Graduate School of Medicine of the University of Pennsylvania Sessions will be held daily at the Temple University Medical Center from 9 00 A M to 5 00 P.M from February 2 to March 10, 1961 Tuition fee—$175 00 Enrollment limited Dr Louis Tuft is course director with Drs George Blumstein and Merle M Miller as associate directors

An outstanding faculty has been assembled to review the basic principles of immunology and allergy as applied to clinical practice Emphasis will be given to the methods of diagnosis and management of the allergic patient

The course is designed for physicians desirous of extending their knowledge of allergy It could serve as an introductory course for those about to enter the field or as a review course for practicing allergists

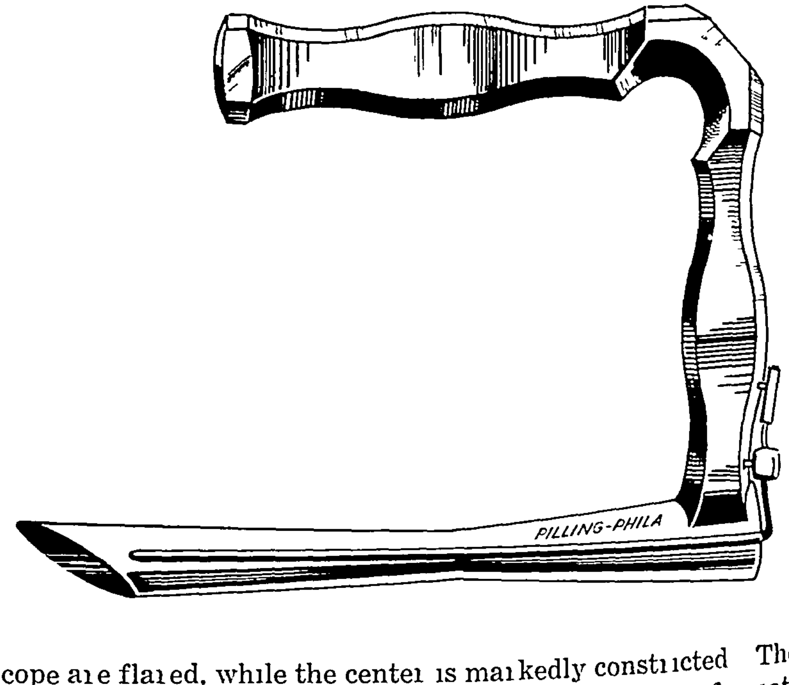
For brochure and application forms write to Dr George Blumstein, c/o Temple Medical Center, Philadelphia 40, Pa

AN HOUR-GLASS ANTERIOR COMMISSURE LARYNGOSCOPE *

PAUL H. HOLINGER, M.D.,

Chicago, Ill.

The design of this instrument incorporates a significant modification of a previously described anterior commissure laryngoscope. Accentuating the principle of the Yankauer post-nasal speculum, the proximal and distal portions of the scope are flared, while the center is markedly constricted. The narrow center section is measured to be at the point of greatest contact with both upper and lower teeth when the laryngoscope is in the position of optimum laryngeal exposure. The view of the anterior commissure is obtained with a minimum



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*From the Department of Otolaryngology of the University of Illinois College of Medicine.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication Oct. 6, 1960.

of anterior lift necessary to elevate the epiglottis The small infant lights and the light carriers are used to reduce further the external diameter Single or double illumination instruments are available, although the single carrier is preferred except for the edentulous patient The instrument is manufactured by the George P Pilling and Son Co of Philadelphia

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UNIVERSITY OF TORONTO—POSTGRADUATE COURSE IN OTOLARYNGOLOGY

On May 11, 12 and 13, 1961, there will be presented a graduate course in this specialty by the staff of the Department of Otolaryngology, assisted by two distinguished guests Dr Philip E Meltzer, Interim Chief of Otolaryngology, Massachusetts Eye and Ear Infirmary, and Acting Head of the Department of Otolaryngology, Harvard Medical School, and Dr W G Hemenway, Department of Otolaryngology, University of Chicago

The first session will begin in the afternoon of May 11th, in the Royal York Hotel, Toronto, in conjunction with the Section of Otolaryngology of the Ontario Medical Association The remainder of the sessions will be held in the clinical areas of the University of Toronto

An attempt will be made to assess, discuss and demonstrate the newer procedures employed in the surgery of deafness The present surgical treatment of head and neck problems will be presented with special consideration of the new conceptions of the responsibilities of our specialty in their management

The fee for the course will be $40 00 and will include a complimentary dinner

Please address all inquiries to the Director, Division of Postgraduate Medical Education, University of Toronto

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

PROGRAM 1960-1961

December 6, 1960

Parkland Memorial Hospital, Rooms No 101-102

5 30 P M —Otolaryngology Clinical Conference

7.30 P M —"Surgery of the Vitreous"—Retinal Detachments, Pupillary Block, etc —Dr Donald M Shafer, New York, N Y

January 10, 1961

Parkland Memorial Hospital, Rooms No 101-102

5 30 P M —Clinical Ophthalmology Conference

5 30 P M —Dr William C Huffman, Iowa City, Ia —Subject "General Problems in Re-Constructive Surgery about the Head and Neck"

7 30 P M —Dr William C Huffman—Subject "Facial Lacerations"

February 7, 1961

Parkland Memorial Hospital, Rooms No 101-102

5 30 P M —Clinical Otolaryngology and Ophthalmology Conference

7 30 P M —Dr Robert N Shaffer, San Francisco, Calif

(1) "Congenital Glaucomas"

(2) Use of Operating Microscope in Ocular Surgery

March 20-22, 1961

Dallas Southern Clinical Society, Statler Hilton Hotel

Guests Dr Ramon Castroviejo, New York, N Y
(Ophthalmology)

Dr Philip Knapp, New York, N Y (Ophthalmology)
Dr French K Hansel, St Louis, Mo (Otolaryngology)
Dr John J Shea, Memphis, Tenn (Otolaryngology)

April 4, 1961

Parkland Memorial Hospital, Rooms No 101-102

5 30 P M —Clinical Otolaryngology and Ophthalmology Conference

7 30 P M —Otolaryngology Panel "Hearing in Relation to Speech Formation and School Performance"—Dr Edward Pratt, Moderator, Dr Phillip Hood, Dr Jack Martin, and Dr Martha Helen Hale

7 30 P M —Ophthalmology "Pre-orbital and Adnexal Ocular Lesions with Basic Principals of Plastic Reconstructive Surgery" Panel Dr Billie L Arnoff, Surgeon, Dr Donald A Corgill, Otolaryngologist, Dr William C Sellman, Plastic Surgeon, Dr Coleman Jacobson, Dermatologist

May 2, 1961

Parkland Memorial Hospital, Rooms No 101-102

5 30 P M —Clinical Otolaryngology and Ophthalmology Conference

7 30 P M —Otolaryngology Dr William Wright, Houston, Tex Subject "Improved Methods of Handling Bony Deviations of the Nasal Septum" Discussion Dr Marvin G Shepard, Dallas, Tex

7 30 P M —Ophthalmology "Non-Surgical Considerations in Horizontal Ocular Deviations" Dr John Lippas, Parkland Memorial Hospital

June 6, 1961

Parkland Memorial Hospital, Rooms No 101-102

5 30 P M —Clinical Otolaryngology and Ophthalmology Conference

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

PROGRAM 1960-1961

December 6, 1960

Parkland Memorial Hospital, Rooms No 101-102.

5 30 P M —Otolaryngology Clinical Conference

7 30 P M —"Surgery of the Vitreous"—Retinal Detachments, Pupillary Block, etc —Dr Donald M Shafer, New York, N Y

January 10, 1961

Parkland Memorial Hospital, Rooms No 101-102

5 30 P M —Clinical Ophthalmology Conference

5 30 P M —Dr William C Huffman, Iowa City, Ia —Subject "General Problems in Re-Constructive Surgery about the Head and Neck"

7 30 P M —Dr William C Huffman—Subject "Facial Lacerations"

February 7, 1961

Parkland Memorial Hospital, Rooms No 101-102

5 30 P M —Clinical Otolaryngology and Ophthalmology Conference

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(1) "Congenital Glaucomas"

(2) Use of Operating Microscope in Ocular Surgery

March 20-22, 1961

Dallas Southern Clinical Society, Statler Hilton Hotel

Guests Dr Ramon Castroviejo, New York, N Y
(Ophthalmology)

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President Dr Erling W Hansen, 90 So Ninth St, Minneapolis, Minn
Executive Secretary Dr William L Benedict, Mayo Clinic, Rochester, Minn
Meeting Palmer House, Chicago, Ill, October, 1961

AMERICAN ASSOCIATION FOR CLEFT PALATE REHABILITATION

President Dr J J Longacre, 1503 Carew Tower, Cincinnati, O
President-Elect Dr D C Samuel Pruzansky, D D S, 840 So Wood St., Chicago, Ill
Secretary-Treasurer Dr Spriestersbach, Ph D, Department of Otolaryngology, University Hospital, Iowa City, Ia
Meeting

AMERICAN BOARD OF OTOLARYNGOLOGY

President Dr Gordon D Hoople, 1100 E Genesee Dr, Syracuse 10, N. Y
Secretary Dr Dean M Lierle, University Hospital, Iowa City, Ia
Meeting Palmer House, Chicago, Ill, October, 1961

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President Dr F Johnson Putney, 1712 Locust St, Philadelphia 3, Pa.
Vice-President Dr Julius W McCall, 619 Rose Bldg, 2060 E 9th St., Cleveland 15, O
Secretary Dr Daniel C Baker, Jr, 903 Park Ave, New York 21, N Y
Treasurer Dr Charles M Norris, 3401 No Broad St, Philadelphia 40, Pa
Meeting Lake Placid Club, Lake Placid, N Y, May 23-24, 1961 (afternoons only)

AMERICAN LARYNGOLOGICAL ASSOCIATION

President Dr Edwin N Broyles, Baltimore, Md
Secretary Dr Lyman G Richards, Wellesley Hills, Mass
Treasurer Dr Francis E LeJeune, New Orleans, La
Editor, Historian and Librarian Dr Francis W Davison, Danville, Pa
Meeting Lake Placid Club, Essex Co, N Y, May 21-22, 1961

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC

President Dr Fletcher D Woodward, 400 Locust Ave, Charlottesville, Va
President Elect Dr John R Lindsay, Chicago, Ill
Secretary Dr C Stewart Nash, 700 Medical Arts Bldg, Rochester 7, N Y
Treasurer Dr K M Day, 121 University Pl, Pittsburgh, Pa
Annual Meeting Lake Placid Club Essex Co, N Y May 23 24-25, 1961

7 30 P M —Otolaryngology Papers presented by Residents of our Teaching Hospitals Dr Ben Stegal—"Pseudomonas Infections in Otolaryngology" Dr Sam Huggins—"Experience in Rhinoplasty Procedures" Dr Chris Helmus—"Aortic Hemograft Reconstruction in Radical Hypopharyngeal Surgery"

7 30 P M —Ophthalmology "Ocular Problems Related to Space Medicine" Speaker to be announced

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VIIeme INTERNATIONAL CONGRESS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY

The Seventh International Congress of Otology, Rhinology and Laryngology will be held in Paris, July 23-29, 1961, at the New Faculte of Medicine, Rue des Saints-Peres The program includes *1* The Recent Acquisitions, Pathology and Therapeutics of Otosclerosis by Profs Sourdille, Sercer and Krmpotic, Weber, Ardouin, Larroude, Ferreri and Shambaugh *2* Indications and Five-Year Results in Surgery and Radium in the Treatment of Cancers of the Larynx and Hypopharynx—Profs Pietrantoni and Agazzi Alonzo, Casadesus, C L Jackson, Leicher, Leroux-Robert and Ormerod *3* A Report on Allergy and Infections of the Nose and Bronchus—Profs Van Dishoeck, Clerici, Hlavacek, Voorhorst, Mayer, Laskiewicz and Terracol

Tours and entertainment for guests and their ladies have been arranged President Dr Maurice Aubry, Secretary General Henry Guillon For further details write Dr Guillon, 6 Avenue McMahon, Paris, France

ASSOCIACAO MEDICA DO INSTITUTO PENIDO BURNIER—CAMPINAS

President Dr Alberto Gallo
First Secretary Dr Alfredo Martinelli
Second Secretary Dr Guedes de Melo Neto
Librarian-Treasurer Dr L de Souza Queiroz
Editors for the Archives of the Society Dr Antonio de Almeida, Dr Gabriel Pôrto, and Dr Roberto Franco do Amaral

ASOCIACION DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOLOGIA DE GUATEMALA

Presidente Dr Julio Quevedo, 15 Calle Oriente No 5
First Vice-Presidente Dr Héctor Cruz, 3a Avenida Sur No 72
Second Vice-Presidente Dr José Luis Escamilla, 5a Calle Poniente No 48
Secretario Tesorero Dr Horace Polanco, 13 Calle Poniente No 9 D

ASOCIACION DE OTO-RINO-LARINGOLOGIA DE BARCELONA, SPAIN

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Vice-Presidente Dr Luis Suñe Medan
Secretario Dr Jorge Perellò, 319 Provenza, Barcelona
Vice Secretario Dr A Pinart
Vocal Dr J M Ferrando

BALTIMORE NOSE AND THROAT SOCIETY

Chairman Dr Walter E Loch, 1039 No Calvert St, Baltimore, Md
Secretary-Treasurer Dr Theodore A Schwartz

BUENOS AIRES CLUB OTOLARINGOLOGICO

Presidente Dr K Segre
Vice Presidente Dr A P Belou
Secretario Dr S A Aranz
Pro Secretario Dr J M Tato
Tesorero Dr F Games
Pro Tesorero Dr J A Bello

CANADIAN OTOLARYNGOLOGICAL SOCIETY SOCIETE CANADIENNE D'OTOLARYNGOLOGIE

President Dr Fernand Montreuil, 1123 St Joseph Blvd East Montreal, Quebec
Secretary Dr Donald M MacRae 324 Spring Garden Road, Halifax Nova Scotia
Meeting Queen Elizabeth Hotel June 15-17, 1961

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President Dr G E Hartenbower, 203 N Main St., Bloomington Ill
President-Elect Dr Edgar T Blair, Springfield, Ill
Vice President Dr G LeRoy Porter, Urbana, Ill
Delegate at Large Dr S G Baldwin Danville, Ill
Secretary Treasurer Dr C L Pannabecker, Peoria, Ill

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Vice-Chairman Dr Howard P House, Los Angeles, Calif
Secretary Dr Walter E Heck, San Francisco, Calif
Delegate Dr Gordon F Harkness, Davenport, Ia
Alternate Delegate Dr Paul H Holinger, Chicago, Ill
Representative to Scientific Exhibit Dr Walter H Maloney, Cleveland, O
Meeting New York City, June 26 30, 1961

AMERICAN OTOLOGICAL SOCIETY, INC

President Dr Henry L. Williams, Rochester, Minn
Vice-President Dr Lawrence R Boies
Secretary-Treasurer Dr James A Moore, New York City, N Y
Annual Meeting Lake Placid Club, Essex Co, N Y, May 26 27, 1961

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY

President Dr Joseph Gilbert, 111 E 61st St, New York, N Y
Vice President Dr Kenneth Hinderer, 402 Medical Arts Bldg, Pittsburgh, Pa.
Secretary Dr Louis Joel Feit, 66 Park Ave, New York 16, N Y
Treasurer Dr Arnold L Caron, 36 Pleasant St, Worcester, Mass

AMERICAN RHINOLOGIC SOCIETY

President Dr Ivan W Philpott, 1801 High St, Denver, Colo
Secretary Dr Robert M Hansen, 2210 Lloyd Center, Portland 12, Ore
Annual Clinical Session Chicago, Ill, October, 1961 (definite time and place to be announced later)
Annual Meeting Chicago, Ill, October, 1961 (definite time and place to be announced later)

AMERICAN SOCIETY FOR HEAD AND NECK SURGERY

President Dr John J Conley, New York, N Y
Vice-President Dr Joseph H Ogura, St Louis, Mo
Treasurer Dr F Johnson Putney, Philadelphia, Pa
Secretary Dr George A Sisson, Syracuse, N Y
Annual Meeting Palmer House, Chicago, Ill

AMERICAN SOCIETY OF FACIAL PLASTIC SURGERY

President Dr Sam H Sanders, 1089 Madison Ave, Memphis, Tenn
Vice President Dr John T Dickinson, Mercy Hospital, Pittsburgh 19, Pa
Secretary Dr Samuel M Bloom, 123 East 83rd St New York 28, N Y
Treasurer Dr Joseph C Miceli, 879 Glenmore Ave Brooklyn, N Y
Winter Meeting February 9 10, 1961 Peabody Hotel Memphis, Tenn

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY

President Dr Leland H Prewitt, Ottumwa, Ia
Secretary-Treasurer Dr Daniel S DeStio, 121 S Highland Ave, Pittsburgh 6, Pa
Annual Meeting Palmer House, Chicago Ill October 7, 1961

FOURTH LATIN-AMERICAN CONGRESS OF OTORINOLARINGOLOGIA

President Dr Dario
Secretary
Meeting

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY

President Dr Van D Rathgeber
Vice-President Dr William Skokan
Secretary-Treasurer Dr Paul Rockwell

GEORGIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President Dr P W Rhyne, Albany, Ga
Vice-President Dr C L Pennington, Macon, Ga.
Secretary Dr James T King, 516 Baptist Professional Bldg, Atlanta 12, Ga
Meeting Oglethorpe Hotel, Wilmington Island, Savannah, Ga, March 2 4, 1961

GREATER MIAMI EYE, EAR, NOSE AND THROAT SOCIETY

President Dr Mariano C Caballero
Vice-President Dr Joseph Freeman
Secretary-Treasurer Dr H Carlton Howard
Meeting Quarterly in March, May, October and December on the second Thursday of the month, 6 30 P M, at the McAllister Hotel, Miami, Fla

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY

President Dr Jo Ono, Tokyo, Japan
Secretary Dr Chevalier L Jackson, 3401 N Broad St., Philadelphia 40, Pa., U S A.
Meeting

KANSAS CITY SOCIETY OF OTOLARYNGOLOGY AND OPHTHALMOLOGY

President Dr Clarence H Steele
President-Elect Dr Dick H Underwood
Secretary Dr James T Robison, 4620 J C Nichols Parkway, Kansas City, Mo
Meeting Third Thursday of November, January, February and April

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President Dr Max E Pohlman
Secretary-Treasurer Dr Wendell C Irvine
Chairman of Ophthalmology Section Dr Carroll A McCoy
Secretary of Ophthalmology Section Dr Philip D Shanedling
Chairman of Otolaryngology Section Dr Robert W Godwin
Secretary of Otolaryngology Section Dr Francis O'N Morris
Place Los Angeles County Medical Association Bldg, 1925 Wilshire Blvd, Los Angeles, Calif
Time 6 30 P M last Monday of each month from September to June, inclusive—Otolaryngology Section 6 30, first Thursday of each month from September to June inclusive—Ophthalmology Section

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

President Dr George Woodruff, Woodruff Clinic, Joliet, Ill
Vice-President Dr Linden Wallner, 122 So Michigan, Chicago, Ill
Secretary-Treasurer Dr Robert Lewy, 25 East Washington St., Chicago 2, Ill
Meeting First Monday of each month, October through May

CHILEAN SOCIETY OF OTOLARYNGOLOGY

President Dr Enrique Grunwald S
Vice-President Dr Agustin Estartus
Secretary Dr Marcos Chaimovich S
Treasurer Dr Benjamin Kapkan K
Director Dr Alberto Basterrica A

COLORADO OTOLARYNGOLOGY SOCIETY

President Dr James T Blair, Denver, Colo
Vice-President Dr James Rigg, Grand Junction, Colo
Secretary Dr Will P Pirkey, Denver, Colo

COLUMBUS, OHIO, OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President Dr John E Arthur
Secretary Dr M L Battles
Meetings First Monday of October through May, University Club, Columbus, O

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

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Vice-President Dr Thomas M McCrory
Secretary-Treasurer Dr James L Baldwin, 1627 Medical Arts Bldg, Dallas, Tex

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Secretary of the Exterior Dr Aldo G Remorino
Secretary Treasury Prof Dr Antonio Carrascosa
Pro Secretary of the Interior Prof Dr Carlos P Mercandino
Pro-Secretary of the Exterior Prof Dr James A del Sel
Pro Secretary of the Treasury Dr Jorge Zubizarreta.

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Secretary-Treasurer Dr Hector R. Silva, Calle Arce No 84, San Salvador, El Salvador, Central America

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President Dr George C Saunders, 644 Medical Arts Bldg, Portland 5, Ore
Secretary-Treasurer Dr Donald C Mettler, 1216 S W Yamhill St., Portland 5, Ore
Meeting Fourth Tuesday of each month from September through May Aero Club, Portland, Ore

OTOSCLEROSIS STUDY GROUP

President Dr E H Campbell, 133 So 36th St, Philadelphia 4, Pa.
Secretary-Treasurer Dr Raymond Jordan, 121 University Place, Pittsburgh, Pa.
Meeting Palmer House Chicago Ill, October, 1961

PACIFIC COAST OTO OPHTHALMOLOGICAL SOCIETY

President Dr John F Tolan, 1118 - 9th Ave, Seattle 5, Wash
Secretary-Treasurer Dr Homer E Smith, 686 Twelfth Ave, Salt Lake City, Utah
Meeting

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY

President Dr Paul Holinger, 700 No Michigan Blvd, Chicago, Ill
Executive Secretary Dr Chevalier L Jackson, 3401 No Broad St, Philadelphia 40, Pa, U S A
Meeting Seventh Pan American Congress of Oto Rhino Laryngology and Broncho-Esophagology
Time and Place

PHILADELPHIA LARYNGOLOGICAL SOCIETY

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Vice President Dr Carson S Demling, 513 Jenkins Bldg, Pittsburgh 22, Pa.
Secretary Treasurer Dr Clyde B Lamp 8101 Jenkins Arcade, Pittsburgh 22 Pa

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President Dr Arthur V Hays
Secretary Dr Edley H Jones, 1301 Washington St, Vicksburg, Miss
Meeting Edgewater Gulf Hotel, Edgewater Park, Miss, May 12 13, 1961

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman Members serve as chairman in alphabetical order monthly
Secretary-Treasurer Dr Roland H Myers, 1720 Exchange Bldg, Memphis, Tenn
Assistant Secretary-Treasurer Dr William F Murrah, Jr, Exchange Bldg, Memphis, Tenn
Meeting Second Tuesday in each month at 8 00 P M at Memphis Eye, Nose and Throat Hospital

MEXICAN ASSOCIATION OF PLASTIC SURGEONS

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Vice-President Dr M Gonzales Ulloa, Mexico, D F
Secretary Dr Juan De Dios Peza, Mexico, D F

MEXICAN SOCIETY OF OTOLARYNGOLOGY

President Dr Rafael Giorgana
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Assistant Secretary-Treasurer Dr Jacob E Reisch, Springfield, Ill

NETHERLANDS SOCIETY OF OTO-RHINO-LARYNGOLOGY
(Nederlandsche Keel-Neus-Oorheelkundige Vereeniging)

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Secretary Dr W H Struben, J J Viottastraat 1, Amsterdam
Treasurer Mrs F Velleman-Pinto, Jac Ohrechtstr 66, Amsterdam

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY

President Dr J C Peele, Kinston Clinic, Kinston, N C
Vice President Dr George E Bradord, Winston-Salem, N C
Secretary-Treasurer Dr J D Stratton, 1012 Kings Drive, Charlotte 7, N C
Meeting

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Vice-President Mr J H Otty, Frizley Old Hall Frizinghall Road Bradford, Yorkshire
Secretary and Treasurer Mr R Thomas, 27 High Petergate, York, Yorkshire

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Vice-Presidente Dr Julio Sanguily
Secretario Dr Juan Portuondo de Castro
Tesorero Dr Luis Ortega Verdes

SOCIEDAD DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOSCOPIA DE CORDOBA

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Tesorero Dr Juan Manuel Pradales
Vocales Dr Osvaldo Suárez, Dr Nondier Asis R, Dr Jorge Bergallo Yofre

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Secretary Dr Daniel Alfredo Alfaro
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Secretario General Dr D Francisco Marañés
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Monterrey 47-201
Mexico 7, D F

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Secretary Dr Carlos Valenzuela.
Treasurer Dr Benito Madariaga
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Secretario Dr José Xirau
Tesorero Dr Alfredo M Petit
Vocal Dr José Gross
Vocal Dr Pedro Hernández Gonzalo

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Secretary Dr Antonio da Costa Quinta, Avenida, de Liberdale 65, 1° Lisbon

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President-Elect Dr Carl D F Jensen, Seattle, Wash
Secretary Dr Willard F Goff, 1215 Fourth Ave, Seattle, Wash

SEVENTH INTERNATIONAL CONGRESS ON DISEASES OF THE CHEST

RESEARCH STUDY CLUB OF LOS ANGELES, INC

Chairman Dr Orrie E Ghrist, 210 N Central Ave, Glendale, Calif
Treasurer Dr Norman Jesberg, 500 So Lucas Ave, Los Angeles 17, Calif
Otolaryngology Dr Russell M Decker, 65 N Madison Ave, Pasadena 1, Calif
Ophthalmology Dr Warren A Wilson, 1930 Wilshire Blvd, Los Angeles 57, Calif
Mid-Winter Clinical Convention annually, the last two weeks in January at Los Angeles, Calif

SECTION ON OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA

Chairman Dr Morris E Krucoff
Vice-Chairman Dr Max J Fischer
Secretary Dr Adrian J Delaney
Treasurer Dr Robert D Ralph
Meetings are held the second Tuesday of September, November, January, March and May, at 6 30 P M
Place Army and Navy Club, Washington, D C

SCOTTISH OTOLARYNGOLOGICAL SOCIETY

President Dr F T Land, 13 Newton Place, Glasgow, C 3
Secretary-Treasurer Dr J F Birrell, 14 Moray Place, Edinburgh
Assistant Secretary Dr H D Brown Kelly, 11 Sandyford Place, Glasgow, C 3

SOCIEDAD COLOMBIANA DE OFTALMOLOGIA Y OTORRINOLARINGOLOGIA (BOGOTA, COLUMBIA)

Presidente Dr Alfonso Tribin P
Secretario Dr Felix E Lozano
Tesorero Dr Mario Arenas A

SOCIEDAD COLOMBIANA DE OTOLOGIA Y AUDIOLOGIA

Presidente Dr Carlos A Cleves, Carrera 12 No 20 69, Bogota Carrera
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Tesorero Secretario Dr Octavio Archila, Calle 25A No 13A35, Bogota Carrera

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President-Elect Dr Harry Ross, Anderson, S C
Vice-President Dr David Stack, Spartanburg, S C
Secretary Treasurer Dr Roderick Macdonald, Rock Hill, S C

SOUTH WESTERN LARYNGOLOGICAL ASSOCIATION
(United Kingdom)

President Mr Michael Robert Sheridan, F R C S , 7 Strangways Terrace, Truro, Cornwall.
Secretary and Treasurer Mr James Freeman, F R.C S , 22 Downs Park West, Bristol, 6

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HISTOPATHOLOGIC OBSERVATIONS IN SO-CALLED CONGENITAL FIXATION OF THE STAPEDIAL FOOTPLATE

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INTRODUCTION

Studies during the past decade by Shambaugh[1] - Holmgren,[3] House,[4] and Hoople[5] have led to the recognition of congenital footplate fixation as a clinical entity Their studies have all been based on clinical examinations and observations at operation The case reported in the present paper is the first to be studied histologically

The patient with "Congenital Footplate Fixation" is born with a severe hearing impairment If this condition is bilateral, as has been observed so far in the majority of instances, development of speech is retarded, and speech is

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the footplate and the base of the anterior crus In the areas where the footplate is not involved by the otosclerotic lesion, its margin and the annular ligament are distinct The footplate there is thin, transparent, and shows a characteristic bluish-grayish shade With the operating microscope, the appearance of otosclerotic footplate fixation is, therefore, quite different from that seen in congenital fixation

EMBRYOLOGY

The term "Congenital Footplate Fixation" appears not to be the appropriate one to characterize this malformation, since it may give the erroneous impression of an ankylosis having occurred at some developmental stage during embryonic life Bast and Anson[8,9] in their exhaustive studies on the development and the anatomy of the ear and the temporal bone in man, have clearly shown that the stapes is derived from two sources, namely, the second visceral (hyoid) bar and the lateral wall of the otic capsule

The origin in the visceral bar is primary and provides the greater mass of the stapes, the annulus stapedialis Subsequent fusion with the lamina stapedialis of the otic capsule and secondary differentiation of laminar tissue provide the stapedial rim, the vestibular surface of the base and the basal perichondrium Further modification of the peripheral laminar-tissue leads to the formation of the annular ligament around the stapes

In the 50 mm (10-11 weeks) embryo, the base of the stapes is separated from the marginal tissue of the vestibular window and through alteration of the precartilaginous tissue in what was, earlier, the lamina stapedialis Since this zone, the site of the future annular ligament, is merely one in which chondral matrix is present in smaller amount than in either the adjacent tissue of the base or that of the fenestra, the stapes at this stage of development is histologically continuous with the vestibular "window" and, in the sense in which the term fenestra is used to describe the opening in the adult bone, no window exists in the otic capsule of the early fetus

The extensive reports of Bast and Anson and their as-much impaired The clinical condition may be recognized at an early age Cochlear function studies at a later age reveal a pure conductive type of loss, averaging approximately 50 db throughout the speech frequency range Bone conduction is usually not impaired, and speech discrimination is normal, indicating an intact cochlear end-organ Repeated hearing tests on these patients over years, as they have been done by Shambaugh[1,2] and House,[4] revealed that the amount of conductive hearing loss remains unchanged The hearing impairment, therefore, is not progressive A positive family history of this malformation, even if it is associated with developmental anomalies of the middle and external ear, is extremely rare (Altmann[6,7]) Congenital Footplate Fixation may occasionally be associated with congenital atresia of the contralateral ear or with a malformation of the lower jaw

The surgical explorations that have been made reveal that the annular ligament is practically absent and that there is no evidence of an otosclerotic lesion involving the oval window The osseous plate obliterating the vestibular fenestra may vary in thickness and appearance A dehiscence in its central portion, covered by a thin transparent movable membrane, has been reported in one case by Shambaugh [1]

The patient with "Acquired Footplate Ankylosis," as a result of otosclerosis, is born with normal hearing, and development of speech is not affected Impairment of hearing begins insidiously, most often between puberty and the early thirties One ear may be impaired before the other, but usually both ears are eventually affected Cochlear function studies reveal that the hearing loss is conductive in nature Hearing impairment is usually progressive, but profound deafness, to the extent that the use of amplification is not beneficial, is rare In 50 to 60 per cent of instances, there is a positive family history of this disease

Exploratory tympanotomy in most instances reveals a single circumscribed, clearly demarcated otosclerotic lesion between the anterior border of the oval window and the cochlea Ankylosis occurs by otosclerotic bone growing across the annular ligament and involving or replacing the adjacent segment of horizontal plane at 20 micra Every fifth section was stained with hematoxylin and eosin for histologic studies

HISTOPATHOLOGY

The histological examination of both temporal bones revealed in summary

1 Developmental anomalies of the labyrinthine capsules involving the internal auditory canal, cochlea, vestibule, cochlear and vestibular aqueducts and the vestibular fenestra

2 Solid bony ankylosis of the stapedial footplates, minor structural deformities of the stapedial arches

3 Extensive malformations of the membranous labyrinth and of the first neurons of the cochlear and vestibular nerves

4 Multiple areas of bone resorption with partial replacement by granulation tissue throughout the labyrinthine capsule

5 Numerous fistulous tracts, as a result of bone resorption establishing open pathways between the internal auditory canal, cochlea, vestibule, semicircular canals and middle ear

6 Leptomeningitis, purulent, acute Labyrinthitis, purulent, acute, caused by extension along the cochlear and vestibular nerves, and by way of the numerous fistulae in the labyrinthine capsules Osteitis and mastoiditis, purulent, acute

The histopathologic findings to be reported in this communication will be limited to the area of the vestibular fenestra A separate paper will be devoted to the description and interpretation of the developmental anomalies of the inner ear and to the character and distribution of the osseous lesions encountered in the otic capsule

HISTOPATHOLOGICAL EXAMINATION OF THE OVAL WINDOW AREAS

Right Ear

There is an almost complete osseous union between the footplate and the adjacent portion of the otic capsule This sociates[8,9,10,11] give the details of the changes that result in the formation of a normal stapes, oval window and annular ligament. For the present paper, it suffices to point out that the maldevelopment resulting in congenital fixation must occur at stages later than the 50 mm. one.

CASE REPORT

These temporal bones were obtained by Dr. Sam H. Sanders of Memphis, Tenn. They are from a 29 year old white man with the following history. The patient's first hospital admission was at the age of seven for study after a fall, followed by a transient episode of unconsciousness. The records mention a recurrent chronic bilateral otitis media and almost complete deafness in both ears, but give no details. Congenital mental deficiency was the principal diagnosis.

The second admission was at the age of 18 to a State Hospital. The patient had wandered from home and, being unable to understand directions from the authorities, had had difficulties with the police. On examination he gave a past history of several episodes of pneumonia and purulent drainage from both ears until the age of 12. The physical examination revealed that he was small for his age and that his teeth were in poor condition. He was "deaf" and unable to speak intelligibly, however, he could read, write, and was a good lip reader. He was mentally deficient. Otherwise the physical examination was negative. The family history was noncontributory. The patient stayed in the hospital for seven years and worked well on several assigned duties. Since he had been doing well, he was discharged and employed with his father in a printing shop, but when after six months he wandered aimlessly away from home on several occasions and he was readmitted at the age of 25. A psychological examination during this period described the patient as mentally deficient and emotionally unstable. No mention of illness or of unusual physical change is found in the records until his terminal illness.

One day the patient was reported to have vomited, and the ward physician felt that he looked and acted ill, although he was afebrile, and the physical examination was negative. At 8:00 A.M. the following day, the patient appeared acutely ill, his neck was stiff and Lasègue's sign was positive. His throat was red, he was still afebrile. Antibiotic and sulfa therapy was started. Later in the day his temperature rose, he became quite restless and death occurred at 4:15 P.M.

The postmortem examination revealed pneumonia and an acute purulent leptomeningitis due to diplococci. Bronchiectases were found in the right middle lobe.

METHOD

The temporal bones were removed four hours after death and received four months later in the University of Chicago Ear, Nose and Throat Laboratory in 10 per cent formalin. Decalcification was done with 5 per cent trichloracetic acid and embedding in celloidin. Both bones were sectioned in the capsule where bone shows Haversian type of ossification (Rt No 300)

The inferior half of the footplate reveals a two-layered structure The inner layer, next to the vestibule, is continuous with the labyrinthine capsule, the outer layer, next to the middle ear, is continuous with the crura of the stapes The dividing zone between the two is formed by an interrupted layer of cartilage or fibrous tissue bordered by cartilage This bi-laminar structure strongly suggests an incomplete fusion of the annulus stapedialis with the lamina stapedialis (Rt No 310), also a failure of the primitive stapedial lamina to separate from the otic capsule, as it normally does

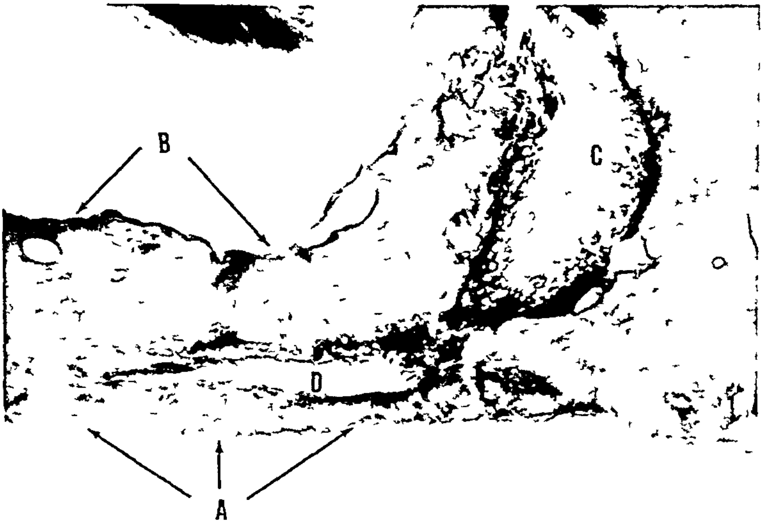

Fig 2 Right ear Section No 310 X155 Posterior end of the footplate The footplate appears as a two-layered structure an inner layer (A) continuous with the labyrinthine capsule and an outer layer (B) continuous with the crura of the stapes The dividing zone between the two is formed by an interrupted layer of cartilage (C) or fibrous tissue bordered by cartilage (D) This bilaminar structure strongly suggests an incomplete fusion of the annulus stapedialis with the lamina stapedialis and failure of the latter to separate from the otic capsule

In the superior portion of the footplate, the inner lamina is mostly resorbed except for a small lip of osseous tissue prosynostosis is extensive all along the fenestral rim except for a small area at the posterior end of the window In one section (see Fig 1) the union is fibrous only There, one encounters a very thin layer of cartilage on the stapedial rim and on the adjacent region of the vestibular fenestra In other sections (see Fig 2) the union is partly fibrous, partly cartilaginous and partly bony

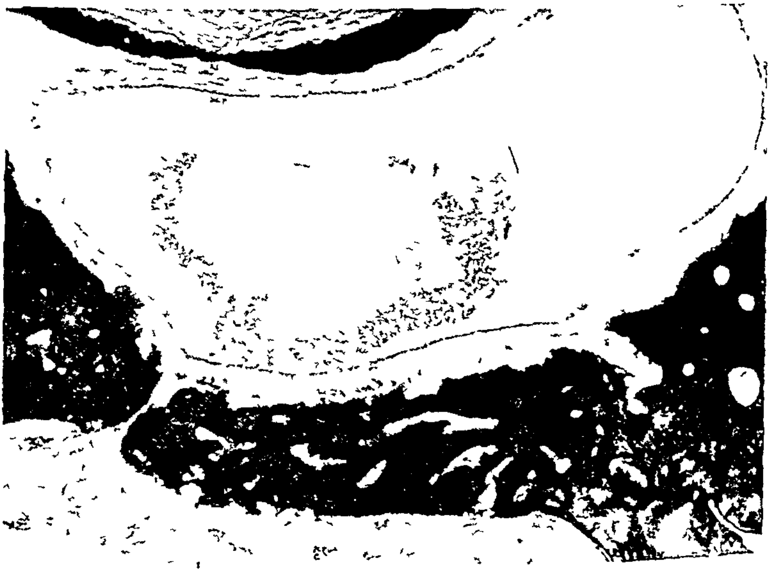

Fig 1 Right ear Section No 280 X50 Superior portion of the footplate Rudimentary annular ligament is seen at the posterior end only Anteriorly the footplate blends into the vestibular capsule

In the region of the fibrous union is one area where the collagen fibers are dense and in parallel arrangement This suggests the formation of an annular ligament (Rt No 280) There is no evidence of this ligament elsewhere The dimensions of the footplate are normal except for its thickness It is thicker in the marginal areas than in the center, but there is no dehiscence Wherever the footplate blends into the vestibular capsule, the structure of the bone does not vary from that of the otic capsule The bone reveals mainly a simple laminated structure, characteristic for endochondral bone, but there are areas in the posterior crus and in the otic

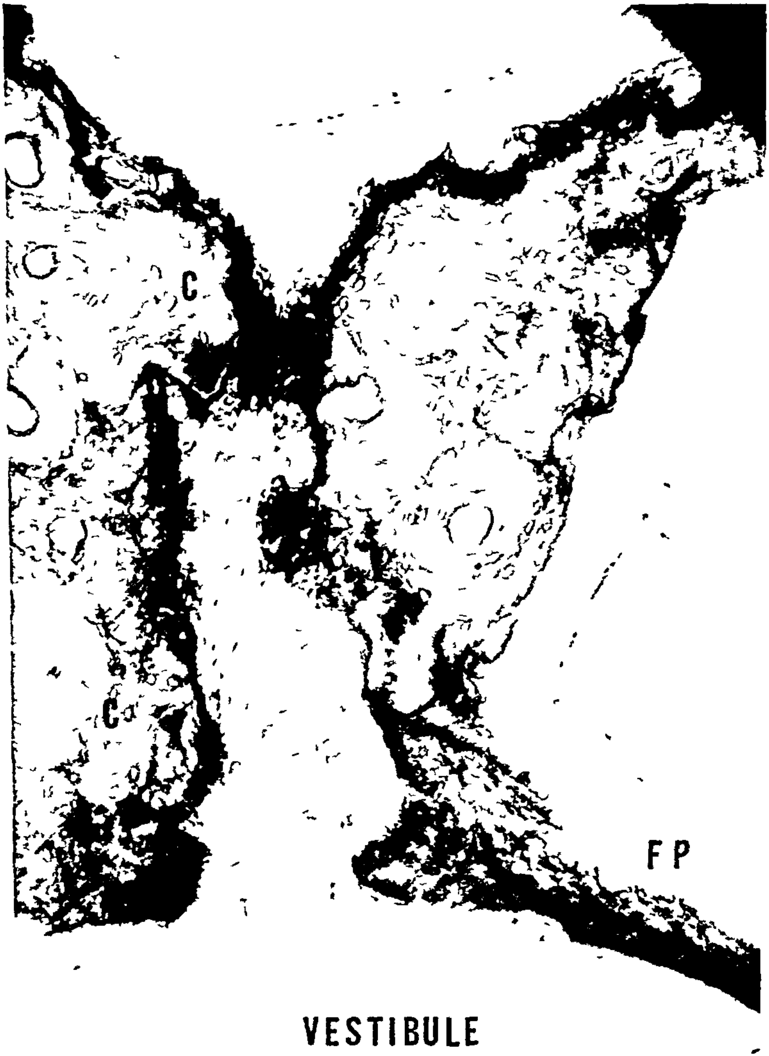

Fig 4 Left ear Section No 320 ×165 Showing the anterior margin of the footplate (F P) and the adjoining rim of the capsule (C) An annular ligament has been partially formed

truding from the anterior window margin on the vestibular side The remaining footplate consists mainly of the tympanic layer, derived from the hyoid bar (see Fig 3)

The crura are bent downward markedly, so that they join the base at an angle acute inferiorly The crural arch is ovoid, and the crura are channeled on the obturator side This hollowing extends into the capital end of the ossicle, neck and head show marked excavation The incudostapedial articulation is essentially normal Vascular channels are present in the footplate and in the base of each crus There is no evidence of any stage of an otosclerotic process anywhere in this region

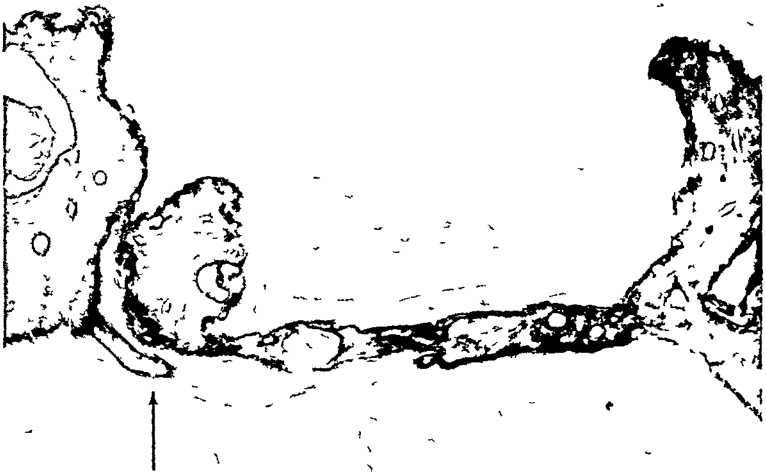

Fig 3 Right ear Section No 300 X60 Note the small lip of osseous tissue (arrow) protruding from the anterior window margin on the vestibular side The remaining footplate consists mainly of the tympanic lamella most of the inner lamella having been resorbed

Left Ear

The histopathological findings are similar to those encountered on the opposite side except in one respect the bony union is complete with the exception of a small area at the panic lamella In the superior portion of the footplate, the inner lamella is partially resorbed Its remnants are indicated by lingular osseous protrusions from the anterior and posterior window margins on the vestibular side (see Fig 7) As in the other ear, the osseous union between footplate and otic capsule is formed superiorly by the outer lamina, inferiorly, by the inner one

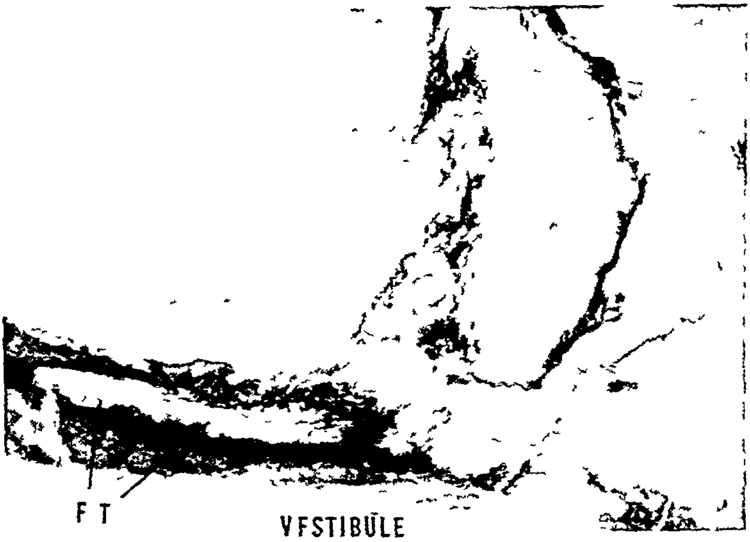

Fig 6 Left ear Section No 336 X180 The junction of the posterior margin of the footplate and the otic capsule A bilaminar structure of the footplate is suggested by the presence of a layer of fibrous tissue (F T) which apparently forms the zone which separates the inner vestibular from the outer tympanic lamella

The crura reveal the same structure, configuration and anomalies as on the opposite side Blood vessels in the bone are present in the bases of the crura and in the footplate There is no evidence of otosclerotic bone in the area of the oval window or elsewhere in the otic capsule

DISCUSSION

The anatomical studies on these temporal bones are in agreement with the clinical observations made by Shamposterior margin of the fenestra Where the union is fibrous, a calcified chondral plate forms the margin of the footplate as well as the adjoining rim of the vestibular fenestra In that area also, the concentration and arrangement of the collagen fibers is such as to indicate the partial formation of an annular ligament (see Fig 4) No cartilage is present on the vestibular side of the footplate, as on the opposite ear Elsewhere the ligament is absent or incompletely formed

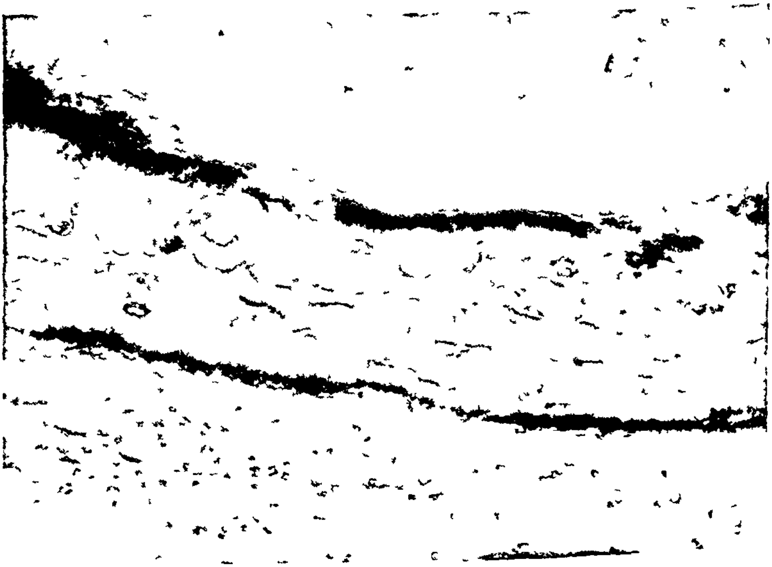

Fig 5 Left ear Section No 301 X600 Midportion of the footplate showing a simple laminated structure with the occasional formation of Haversian systems

The diameters of the footplate are normal, except its thickness, which is more marked in the marginal portion The center is thin, but shows no dehiscence The bony structure is the same as in the otic capsule It is a simple laminated structure with occasional formation of Haversian systems (see Fig 5)

A bi-laminar structure of the footplate is suggested in the inferior portion (see Fig 6) A layer of fibrous tissue bordered by cartilage or of cartilage tissue alone forms the zone which separates the inner, vestibular, from the outer, tymdeveloped temporal bone The annulus stapedialis, a derivative of the second visceral bar, fuses with the lamina stapedialis of the otic capsule to form the future stapes As the base of the stapes increases in diameter, the cartilaginous fenestral rim undergoes dedifferentiation from cartilage to mesenchyme The base of the stapes widens by a concurrent process of decrement on the capsular, and of increment on the stapedial side until the stapes has attained adult dimensions This occurs at about mid-term stage It is not until the fetus has reached this stage that an annular ligament becomes established This ligament comes into being by a transformation process of the precartilaginous tissue in what was, earlier, the lamina stapedialis (Bast and Anson[8 9])

The serial sections through the vestibular fenestrae clearly indicate a bi-laminar structure of the stapedial base These two laminae are separated by a zone of fibrous tissue bordered by cartilage or of cartilage alone At the posterior end of the footplates, where a fibrous union exists between the base and the window margin, a thin layer of cartilage exists on the circumference of the base and on the adjacent margin of the oval window, also, a parallel arrangement of collagen fibers is present in that area, but more definite in the left ear, and this could be interpreted as a partial formation of an annular ligament The sections reveal further a structural deformity of the crural arches

Obviously no osseous transformation process, whatever its etiology, can produce such pathological changes in a footplate These observations, however, can be explained on the basis of an anomalous development of the stapedial footplate and the adjoining otic capsule

Whereas investigators have been in general agreement on a branchial derivation for the malleus and incus, the opinions in the past have differed widely as to the origin of the stapes (Cauldwell and Anson[12]) The opinions were that the stapes arose from *1* the first branchial (mandibular) arch, *2* the second branchial (hyoid) arch, *3* the otic capsule, *4* the otic capsule and the second visceral arch, and *5* a "periarterial" aggregate of cells distinct from visceral or cranial baugh,[1 2] Holmgren,[3] House,[4] and Hoople[5] The vestibular fenestra may be obliterated by birth and by a process other than otosclerosis In this respect, "Congenital Fixation of the Stapedial Footplate" does exist, and it may be considered a clinical entity

Otosclerosis, Paget's disease, Endemic Goiter (iodine deficiency goiter), adhesive processes and forms of localized osteitis, etc, are among the pathological processes in the middle ear or otic capsule, which may lead to new bone formation and thus to fixation of the stapedio-vestibular articulation Perusal of our sections, however, excludes any of these morbid processes from having been the cause for a secondary ankylosis in a previously normally developed oval window, hence, the osseous closure must have a different etiology

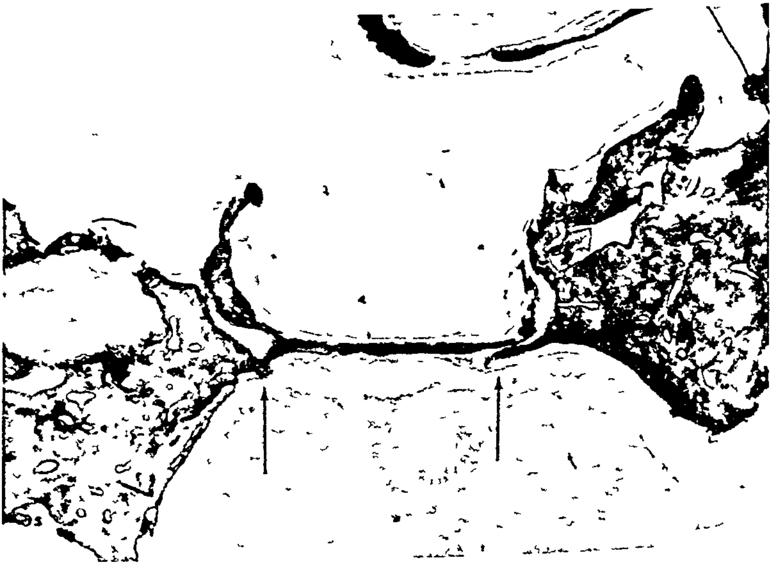

Fig 7 Left ear X28 The inner lamella is partially resorbed leaving as remnants lingular osseous protrusions (arrows) from the anterior and posterior margins

Developmental anatomy reveals that no "window" exists in the otic capsule of the early fetus in the sense in which the term fenestra is used to describe the opening in the fully the otic capsule, explains the condition found The histologic sections show no evidence of an otosclerotic process

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elements The concept of a composite origin, partly from the second visceral arch and partly from the otic capsule, was first supported by Gradenigo,[13] van Noorden,[14] later by Hertwig,[15] Jenkinson,[16] Macklin,[17,18] Reagan,[19] and McClain [20]

Bast and Anson, with their exhaustive studies, confirmed the origin of the stapes from these two sources the visceral bar provides the annulus stapedialis, subsequent fusion with the lamina stapedialis of the otic capsule and secondary differentiation of laminar tissue provide the stapedial rim, the vestibular surface of the base and the basal perichondrium The annular ligament develops through further modification of the peripheral laminar tissue

With this concept of the embryology of the stapes, the observations described in this report find their explanation in an anomalous development We are confronted here with an incomplete fusion of the annulus with the lamina stapedialis, and with a lack of further differentiation of the lamina stapedialis As a result, the stapedial base is deformed, and no true annular ligament has been developed Ossification of the cartilaginous otic capsule, therefore, brought about the osseous obliteration of the vestibular fenestra Since no "window" has ever existed in this pair of temporal bones, in the sense in which the term fenestra is used to delineate the opening into the vestibule of a fully developed otic capsule, the term ankylosis is not appropriate to characterize this malformation The conclusions are then that the stapes fixation is the result of a congenital lack of differentiation and development of the stapedial base and of its annular ligament The presence of other malformations in the inner ear corroborate this interpretation

SUMMARY

Histopathological observations in a case with bilateral congenital fixation of the stapedial footplate are reported The findings are in agreement with the clinical impression of the nature of the lesion Failure of normal development of the footplate of the stapes, with an extensive persistence of the continuity of the lamina stapedialis (of Bast and Anson) with

STAPES SURGERY

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Buenos Aires

In certain cases of otosclerosis Shea[1] removes the incus including the footplate, covers the oval window with a vein graft and inserts a polyethylene tube between the vein graft and incus I developed a variation of Shea's procedure in which a mucous flap made from the mucosa of the promontory and oval window niche is substituted for the vein graft This procedure is as follows

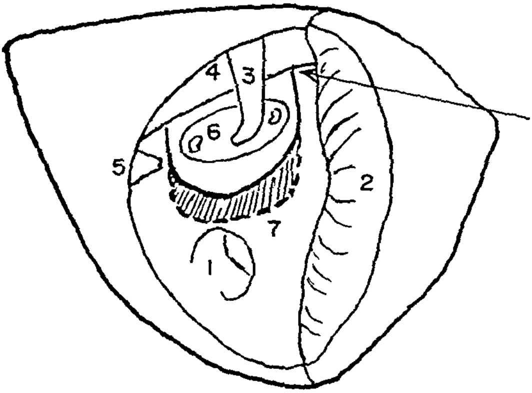

Fig 1 Diagram of the surgical field seen after completion of the operation without the polyethylene strout The arrow points to the starting point of the incision whose margin is indicated by a dotted line The striped region corresponds to the gap between the incision and the flap which has been replaced 1 round window 2 tympanic membrane 3 incus 4 wall of facial canal 5 eminentia pyramidalis 6 oval window 7 promontory

The head and crura of the stapes are removed The mucosa of the promontory is cut with either a sharp point or round knife The incision starts at the point where the cochleariform process makes contact with the wall of the facial canal and proceeds vertically downward until the level of

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May 5—Discussion Vasomotor Rhinitis

Section of Otology

Program for session 1961

February 3—Papers 1 Problems of Tympanoplasty 2 Experience with Pedicled Temporal Muscle Flaps in Radical Mastoid and Tympanoplasty Operations 3 Is Myringoplasty Worth While?

March 3—Papers 1 The Pathological Bases of Some Labyrinthine Vascular Disorders 2 Meniere's Syndrome

May 5—Paper The Present Position of Stapes Footplate Surgery

Members of the American Laryngological Association, the American Triological Society and the American Otological Society who will be in the United Kingdom at the time of any of these meetings will be very welcome

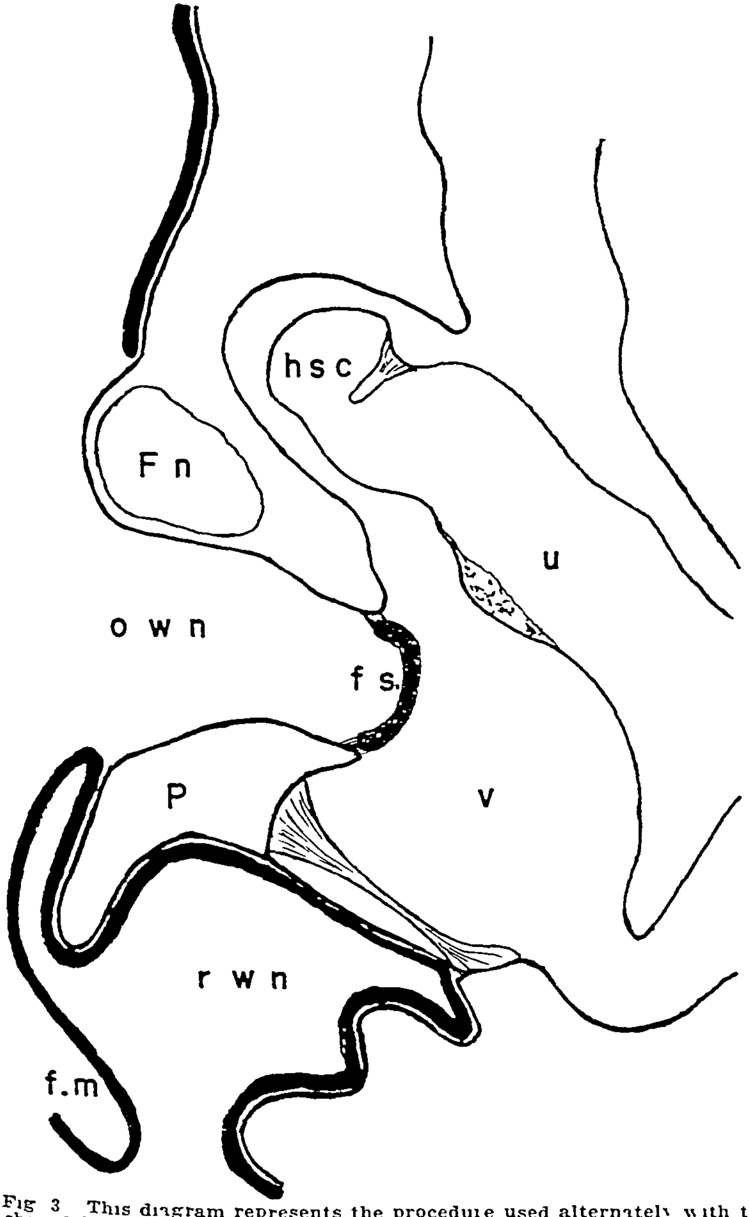

Fig 3 This diagram represents the procedure used alternately with the U shaped flap The transversal section is made along the wall of the facial canal and the flap turned over the promontory

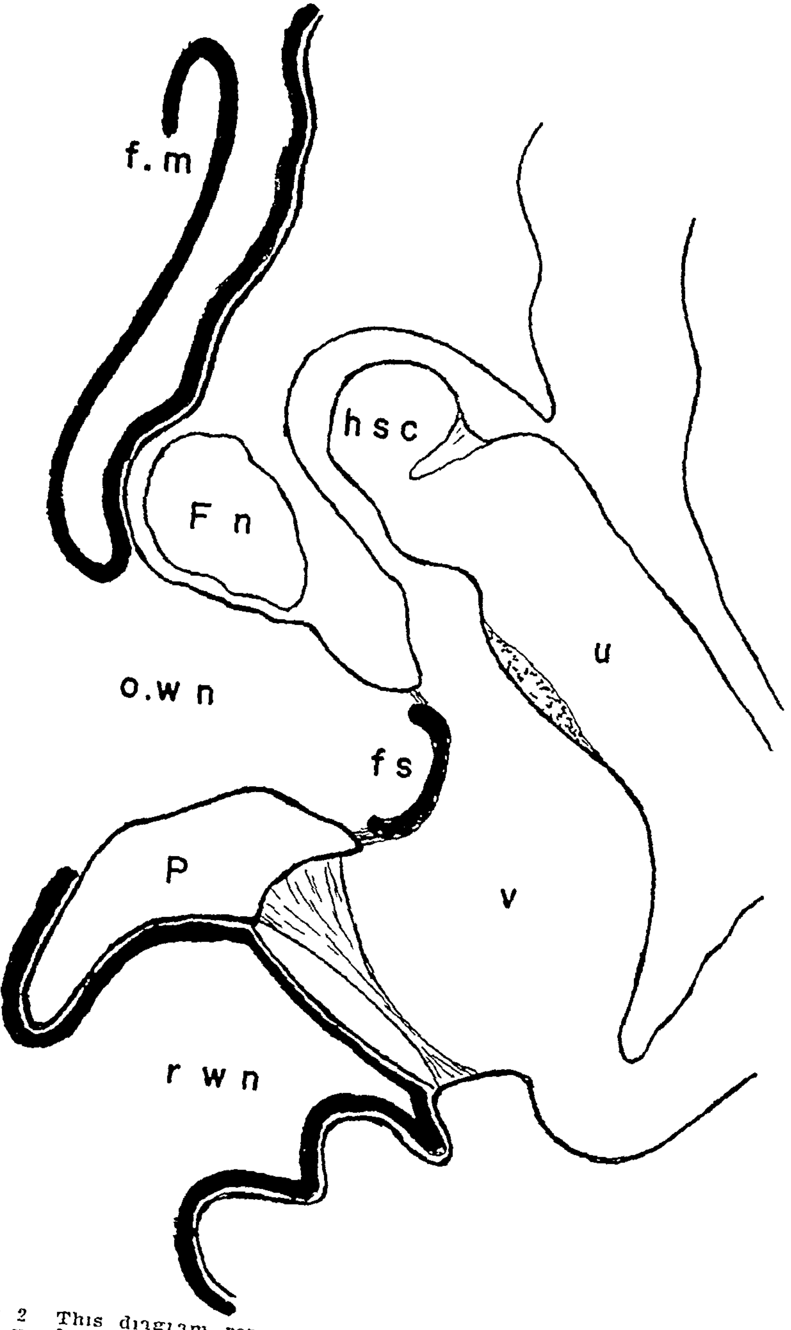

Fig 2 This diagram represents a section passing through oval and round windows after the mucosa has been detached and elevated over the wall of the facial canal The outline of this diagram was taken from a section of human temporal bone (40X) The head and crura have been removed but the footplate of the stapes still remains in place In this figure and in Fig 3 structures are identified by the following symbols F n facial nerve f m flap of mucosa u utriculus o w n oval window niche r w n round window niche f s footplate of stapes *P* promontory v vestibule h s c horizontal semicircular canal

mucosa after it is detached. The flap also presents two small openings which correspond to those of the crura which were removed.

Finally, as described in the literature, a polyethylene tube is inserted between the incus and the mucosa which lies over the oval window.

I have operated upon eight cases with this procedure. The best as well as the poorest results are here presented (see Tables I and II).

I think that the advantages of the mucous flap are clear when the results are compared with other procedures, *i. e.*, vein graft, covering the fenestra ovalis with gelfoam or leaving the oval window open with a prosthesis in place.

REFERENCE

1. SHEA, J. J.: Fenestration of the Oval Window. *Ann. Otol. Rhinol. and Laryngol.*, 67:932-951, 1958.

ANNOUNCEMENT

The University of Minnesota announces a one-day continuation course in Otolaryngology for Specialists which will be presented at the Center for Continuation Study January 26, 1961, preceding the Sectional Meeting of the American Laryngological, Rhinological, and Otological Society in Minneapolis on January 27-28, 1961. Guest registration for the meeting of the "Triological" will be welcomed. The program for the continuation course has been designed to complement the scientific program of the "Triological" Society.

Lodging and meals are available at the Center for Continuation Study.

the superior margin of the round window is reached, it then turns posteriorly and proceeds as far as possible in this direction, finally, the incision turns upward, behind the oval window, until the facial canal is reached once again The borders of the incision are shown as a dotted line in Fig 1 Hemorrhage may be controlled by electrocoagulation

The flap is made by elevating the mucosa from the transverse cut upward This procedure successfully uncovers the promontory between the round window and oval window, the walls of the oval window niche and the footplate of the stapes This flap is then turned over the walls of the facial canal Fig 2 illustrates the mucosal flap turned upward

TABLE I

Miss E C, 44 years

		128	256	512	1 024	2 048	4 096	8 192
Preoperation	Air		90	80	80	85	85	75
Sept 2, 1958	Bone		20	20	25	30	30	
Postoperation	Air	15	5	10	15	20	60	50
July 30, 1959	Bone		15	10	5	15	35	

TABLE II

Miss C H de G D

		128	256	512	1 024	2 048	4 096	8 192
Preoperation	Air	50	50	60	55	60	80	80
Aug 1, 1958	Bone		0	0	0	5		
Postoperation	Air	25	20	20	20	30	35	60
Sept 8, 1959	Bone		10	10	10	20		

The U shaped flap can be made inversely, i e, the horizontal cut running along the wall of the facial canal In this case the flap is turned over the promontory as Fig 3 illustrates After the flap is made, the footplate of the stapes is partially or totally removed in the classical manner

The flap of mucosa is replaced over the bone surface and oval window niche After replacement, a small gap remains between the margins of incision and the flap, as the striped region of Fig 1 indicates The gap is due to retraction of at no time reached more than a few dozen in the medical centers of the entire world

In order to meet this growing need in the area of communication disorders, a new profession has come into existence which is known as *audiology and speech rehabilitation* Recruited from the ranks of educators, psychologists, and academic speech experts, the pioneers in audiology and communication sciences continue their outstanding contributions to these rapidly growing fields, yet, it is curious to note that vocal pathology attracts considerably less attention than do the questions of hearing and speech As a rule, all abnormal voice conditions are treated by speech therapists under the vague heading of "voice problems"

As a result of these developments, the patient suffering from hoarseness may be handled from three entirely different viewpoints The laryngologist is primarily concerned with the detection of malignant disease, the speech therapist is trained to teach desirable speech habits, the occasionally encountered phoniatrist combines laryngologic training and experience with the objective analysis of laryngeal function, moreover, these three specialists use different professional languages for the expression of their opinions which causes constant misunderstandings among these important representatives of sciences dealing with human communication

The purpose of this paper is threefold to show how well the old authors understood various diseases and disorders, to demonstrate the need for complete medical, laryngologic, as well as psychologic evaluation in all cases of vocal pathology if the patient is to be considered a whole personality (and not an assembly of specialized interests), and to offer a theory for the psycho-dynamic explanation of hyperkinetic voice disorders as a phenomenon of regression in response to stressful situations

HISTORICAL REMARKS

It is now exactly 100 years since J N Czermak[11] observed for the first time that phonation with the ventricular folds is a pathologic phenomenon Five years after the invention of

VENTRICULAR DYSPHONIA NEW INTERPRETATION OF AN OLD OBSERVATION[*]

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PURPOSE OF THIS STUDY

The science of optics is one of the oldest branches of physics This may be one reason why eye specialists always devoted much time and study to the precise analysis of visual problems Acoustics, however, made its greatest advances since the introduction of electronic techniques a few decades ago Practicing otolaryngologists have little time to keep pace with the revolutionary development of psycho-acoustics and usually limit the evaluation of their patients' hearing losses to fundamental audiometry Compared with the pure tone audiogram, the average laryngologist's description of a vocal anomaly is even more elementary, being restricted to a subjective judgment of the respiratory efficiency of the larynx and the social adequacy of the voice

Ever since the invention of the laryngeal mirror, a few interested laryngologists devoted their efforts to the study of vocal pathology and collaborated with physiologists and acousticians in the clarification of vocal physiology Known as *phoniatrists,* these specialists tried to promote the general understanding of vocal problems and assisted their surgical colleagues in the best care of their patients Some also combined the practice of general otolaryngology and vocal rehabilitation Unfortunately, the number of such phoniatrists

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[*] From the Department of Research New York Eye and Ear Infirmary

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unconscious aggressiveness of an imbalanced personality as a reaction to the threats of daily life. This primitive neurolaryngeal mechanism can even be seen with the laryngeal mirror which shows the pathognomonic *sphincter action* of the laryngeal structures during all types of hyperkinetic phonation (see Fig. 1). Since all primitive laryngeal reflexes, like coughing, gagging, retching, vomiting, and swallowing, are characterized by the sphincter action of the epiglottis,

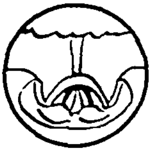

Fig. 1. Laryngoscopic image of hyperkinetic dysphonia with marked laryngeal sphincter action. Note the descent of the epiglottis, the anterior over-adduction of the ventricular folds and the posterior deficiency of vocal cord closure.

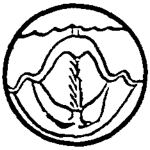

Fig. 2. Laryngoscopic image of ventricular dysphonia. The ventricular folds are over-adducted during all phonatory actions. Often this is combined with the laryngeal sphincter mechanism shown in Fig. 1.

cords and ventricular folds, this invasion by the primitive sphincter action of the highly specialized functions of human phonation indicates a regressive phenomenon.[4,7] It is interesting to note how psychiatric theory interprets this symptom of laryngophonic regression in the light of the projective test analyses of such patients.[20,21]

CLASSIFICATION

Under the influence of the primitive laryngeal sphincter action, phonation with the ventricular folds (see Fig. 2) may originate from several somatic and psychogenic causes.[20] According to our description,[3,7] one may differentiate six specific forms of ventricular dysphonia, namely the habitual, emo-the laryngeal mirror by Garcia (1854), the two fathers of laryngology, Turck and Czermak, were well engaged in presenting their new findings to the medical profession, thus, the discovery of ventricular dysphonia was one of the first clinical descriptions resulting from the use of laryngoscopy

Further developments in the knowledge of the function of the ventricular folds for phonation were studied by Panconcelli-Calzia[30 31] He showed the illustrations of various authors which clearly depict the hyperkinesis of the ventricular folds, for instance those of Merkel[25] (1862) In his textbook on laryngology in 1893, Moritz Schmidt[38] described the faulty phonation with the ventricular folds A systematic study of the ventricular folds was presented by Flatau and Gutzmann[16] in their book on ventriloquism (1894) Flatau[14 15] also dealt with therapy of ventricular dysphonia (1910, 1928) Further contributions to this subject were made by many authors as listed in the bibliography[5,9 12 13 17 22 26,27,28 39 40 41] During World War I various laryngologists described their observations of ventricular dysphonia among soldiers Similar experiences during World War II were reported by one of us[3]

DEFINITION

Ventricular dysphonia is known under many names Taschenfaltenstimme (German), Voix des bandes ventriculaires (French), Dysphonia plic*ae* ventricularis,[23] Dysphonia plica ventricularis,[37] Dysphonia plicarum ventricularum,[31] Dysphonia ventricularis,[43] etc It is caused by the faulty participation of the ventricular folds (false cords) in the act of phonation Normally, the ventricular folds represent the middle laryngeal sphincter and contribute to the firm closure of the larynx during deglutition and expectoration, hence, they belong to the *protective sphincter mechanism* of the larynx

It is fascinating to compare Furstenberg's[18] experimental findings on the neural mechanism of laryngeal closure with the clinical signs of ventricular dysphonia From such viewpoints we may now explain this voice disorder as the intrusion of a subcortical *primitive mechanism of phonation* after the regular cortical phonatory system had become inhibited by the result of the deficient or totally absent glottal closure the voice is reduced to a breathy and feeble sound (dysphonia) or to a faint whisper (aphonia). This is the form of emotional aphonia usually encountered in psychoneurotic or hysterical women.

On the other hand, the *hyperkinetic* type of psychogenic dysphonia shows the sphincter action of the laryngeal introitus. During his visibly great speaking efforts the patient tries to squeeze out his voice by forcefully overadducting the ventricular folds. This emotional type of ventricular dysphonia is distinguished from the specific disease entity of spastic dysphonia by the fact that the former affects also the singing voice, is always present, does not fluctuate between clear parts of a sentence and other words spoken with choked spasticity, does not divide the stressed vowels, and that the patient does not exhibit much anguish about his condition. These differences were analyzed in a previous monograph[1] on traumatic voice disorders and in a recent discussion of spastic dysphonia.[2]

DIAGNOSIS

With a complete history and after careful laryngoscopic inspection, the patient's complaints can be readily evaluated. Examination of the *upper airways* reveals normal conditions. If some abnormal findings are encountered, they represent coincidental complications which have nothing to do with the habitual or psychogenic vocal disorder; however, the constant vocal strain may lead to secondary signs of tissue irritation. One of us[1,2] described how the typical findings of chronic laryngitis may very well reflect the *results* of hyperkinetic types of hoarseness, and not their *causes*. For this reason we often see a moderate redness or congestion of the vocal cords in cases of ventricular dysphonia. The constant overuse of the sphincteric action of the ventricular folds can even lead to their hypertrophy.[2]

Bruno[10] demonstrated the *hypertrophic appearance* of the ventricular folds on tomographic radiographs. It could also clearly be seen how the ventricles of Morgagni were oblitertional, paralytic, cerebral, cerebellar, and the vicarious type, which are readily distinguished by history and examination

1 Most frequently, it represents the extreme end stage of hyperkinetic dysphonia due to constant vocal abuse which is usually of *habitual* origin

2 In times of general stress, or during a crucial period in the life of a psychoneurotic person, an emotional crisis may precipitate the psychogenic or *emotional* form of hyperkinetic dysphonia This is always characterized by hyperkinesis of the ventricular folds

3 The *paralytic* form is due to a rare compensatory reaction to laryngeal paralysis[6] The ventricular folds take over the function of phonic glottal closure, substituting for the true cords which cannot meet due to the paralysis

4 As a sign of dysarthria resulting from *cerebral* disease, the voice may change to an extremely low, choked, rough, intermittent, spastic, and peculiarly squeezed sound

5 Spastic and labored phonation is most typical for *cerebellar* lesions, as described by several authors[3 24] Mid-brain disease, such as in Parkinsonism, may also produce ventricular dysphonia[24 31]

6 Finally, we know the *vicarious function* of the ventricular folds when they are substituting for the defective vocal cords The systematic development of a well compensated ventricular fold voice is one of the goals of vocal rehabilitation after the loss of one or both vocal cords as a result of traumatic injury or surgical operation

J S Greene[19] discussed the differentiation of the usual types of psychogenic dysphonia and aphonia, which are often referred to as being of hysterical origin, and the specific condition of spastic dysphonia, or psychophonasthenia, as he preferred to call it It had been observed for a long time that emotional (or hysterical) aphonia and dysphonia exist in two main types the hypokinetic and the hyperkinetic

The *hypokinetic* type of psychogenic dysphonia is caused by incomplete vocal cord adduction during phonation As a result of the deficient or totally absent glottal closure the voice is reduced to a breathy and feeble sound (dysphonia) or to a faint whisper (aphonia) This is the form of emotional aphonia usually encountered in psychoneurotic or hysterical women

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Bruno[illegible] demonstrated the *hypertrophic appearance* of the ventricular folds on tomographic radiographs It could also clearly be seen how the ventricles of Morgagni were obliterated This radiologic appearance did not change with various therapeutic efforts, including local anesthesia of the pharyngeal structures In view of the recently renewed interest in contact ulcers of the vocal processes it is interesting to remember that Oltuszewski,[28a] in 1885, described two cases of contact ulcers which had developed as a result of prolonged spastic phonation All these signs of *secondary laryngitis* must not be misinterpreted as a local organic cause of the presenting vocal disability

The patient's *hoarseness* is fully explained by the ventricular hyperkinesis and the visible sphincter action of his larynx Such voices sound extremely low, harsh, and coarse, their basic pitch often reaching the "subtonal" frequencies between 50 and 100 cps If the patient complains of *pain* in the throat, especially after having spoken for a while, his vocal overexertion and the faulty use of his larynx readily explain this complaint as a sign of functional irritation

Often, we hear of a *lump* in the throat, especially below the larynx According to Tremble,[42] this complaint is frequently due to esophageal spasm Similar spasm of the m cricopharyngeus is also due to nervous tension Such signs will direct one's attention to some underlying emotional factors In case of remaining doubt a frontal tomograph will bring final clarification Once the precise type of ventricular dysphonia has been recognized, therapy is directed by the individual findings

CASE REPORTS

Case 1 White female, age 33, married housewife, complained on October 10, 1957, that she suddenly lost her voice three weeks previously

Previous History Recurrent bronchitis, once diagnosed as asthma First marriage at age 19, divorced after one year, second marriage one year later Seven pregnancies, one uterine curettage, two miscarriages, five children are alive and well Had numerous surgical operations appendectomy (age 17), ovarian cyst (22), tonsillectomy (23), D and C (24), hysterectomy (30), excision of benign tumor from left axilla (31) She was always very nervous and unstable

History of Vocal Complaints When 16 years old, she was rehearsing for a school drama performance During some dispute she fainted and awoke voiceless After four weeks of whispering she regained her voice and could perform in the leading role as she had aspired Ever since she experienced occasional attacks of sudden hoarseness Often, she felt a choking sensation in her throat, as if a hair or a lump were stuck For the past few months she had lived with her mother while her father had a kidney operation and suffered his fourth stroke Some of her five children were also ill Three weeks ago, one of her children came up to her bed at 3 a m and wanted to sleep with her She felt angry, tried to send the child to his own bed, but could not utter a sound Next morning she was still voiceless and consulted her family physician who gave her a sedative Various other ataractics were subsequently prescribed without effect on the voice

Examination showed a well developed young looking woman Ears and the upper airways showed normal findings Larynx the vocal cords were slightly congested and moved normally however, during phonation they were completely covered by the over adducted ventricular folds The voice was totally aphonic and sounded like a harsh whisper

Fig 3 Anterior laryngeal tomograph of ventricular dysphonia. Note laryngeal sphincter action with closure of ventricular folds and lowered epiglottis (Case 1)

Radiologic Analysis Lateral radiographs of the neck as well as frontal tomographs during quiet respiration were normal, however, tomography *during phonation* showed characteristic changes Fig 3 demonstrates a frontal section through the anterior half of the larynx Cords and ventricular folds are firmly approximated, Morgagni's ventricles were compressed the epiglottis was lowered and the trachea was moderately widened as a sign of high subglottic air pressure Fig 4 is a frontal section through the posterior half of the larynx Again cords and ventricular folds are completely closed Morgagni's ventricles appear as narrow slits The arytenoids are in normal adducted position The trachea is moderately *widened by high subglottic pressure For better* comparison, a normal tomogram is shown in Fig 10

Diagnosis Psychogenic ventricular (hyperkinetic) aphonia symptomatic of conversion hysteria.

Course Under verbal persuasion, the patient was able to produce several clear vowels, however, she immediately reverted to her harsh and forced whisper Obviously in need of psychiatric treatment, she was referred to our neuropsychiatric clinic for evaluation and psychotherapy A few months later she regained her voice overnight

Case 2 White male age 58, salesman, presented himself on February 23, 1957, with the complaint of hoarseness and pain in his throat His voice had weakened for several years, especially at times when he talked much No major illnesses noted

Examination showed moderate enlargement of the lower nasal turbinates Tonsils were absent The house surgeon observed moderate

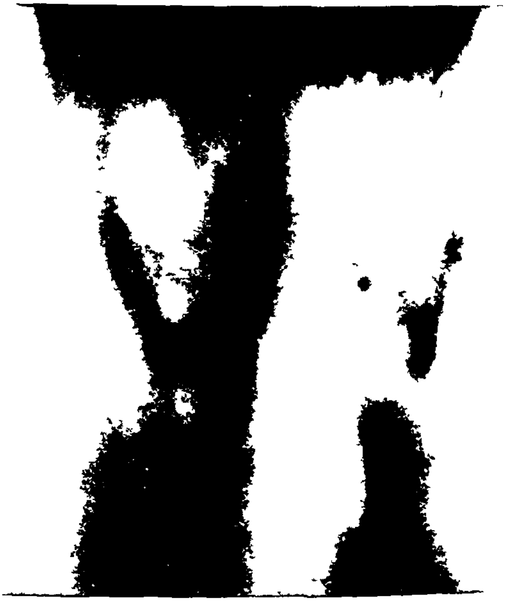

Fig 4 Posterior laryngeal tomograph of ventricular dysphonia Note compression of ventricles closure of ventricular folds and dilatation of trachea (Case 1)

hyperemia and congestion of both vocal cords which did *not* close perfectly over the posterior portion of the glottis The voice sounded *hoarse* and rough The patient gave a calm and well adjusted impression

Impression Chronic laryngitis was assumed, and a course of antibiotics and expectorants was prescribed

Course A week later, the patient returned with the same complaint and unchanged findings He was advised to use steam inhalations and rest his voice After another week, he was referred for vocal analysis This time it was noted that his larynx presented typical changes The ventricular folds appeared hypertrophic and abnormally *mobile* The vocal cords were plump and congested Observation of phonation revealed the following peculiarities at the onset of phonation the vocal cords closed normally, but the ventricular folds began to bulge over their mid portions This stage was followed by complete closure of the ventricular folds which visibly vibrated, while a low and rough voice was audible At the end of phonation, the ventricular folds began to separate anteriorly and posteriorly until shortly afterwards their central bulge disappeared This cycle was concluded by abduction of the vocal cords Fig 5 demonstrates this sequence of ventricular fold hyperkinesia The voice sounded unusually low hoarse rough and harsh Average speaking pitch was found around tone E (82 5 cps), the vocal range appeared restricted to a few tones and phonation time was shortened to 8 sec (vowel "o" on tone E)

Diagnosis Habitual ventricular (hyperkinetic) dysphonia resulting from occupational abuse of the voice

Therapy After ascertaining that all other clinical and laboratory findings were normal (sinus X-ray, CBC serologic blood sugar, urine, etc) the patient was counselled about the proper use of the voice He was advised to speak little and in a relaxed manner and was shown how to do some vocal exercises Steam inhalation and an antihistaminic were suggested for symptomatic relief

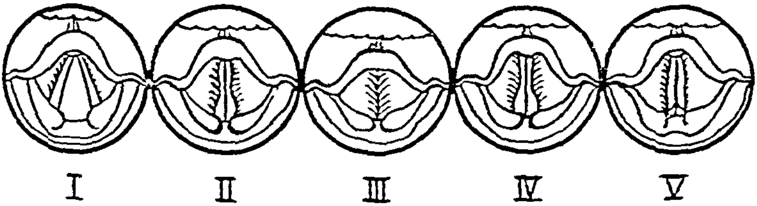

Fig 5 Sequence of laryngeal movements during one cycle of phonation in a case of ventricular dysphonia I respiration II onset of phonation with beginning hyperkinesis of ventricular folds III phonation with ventricular folds note narrowing of laryngeal introitus IV beginning relaxation of ventricular folds towards end of phonation V beginning separation of vocal cords at end of phonation note elevation of epiglottis (Case 2)

RESULTS

On April 2, 1957, the patient reported great improvement and spoke with a clearer and higher voice. His larynx appeared less irritated and the hyperkinesis of the ventricular folds was reduced so that regular vibration of the vocal cords could then be seen In the course of the next months the patient continued to improve and was able to speak with a fairly clear, loud, well placed voice Laryngeal congestion had disappeared. and the hyperkinetic movement of the ventricular folds was greatly diminished.

Radiographic analysis was undertaken at the beginning and end of this course of vocal rehabilitation. Whereas the lateral view of the larynx was always normal during quiet respiration (see Fig 6), the tomographs taken *during phonation* demonstrated the ventricular hyperkinesia In the stage of marked ventricular dysphonia (see Fig 7), the vocal cords and ventricular folds were seen in close approximation, obscuring the ventricles of Morgagni The trachea appeared markedly

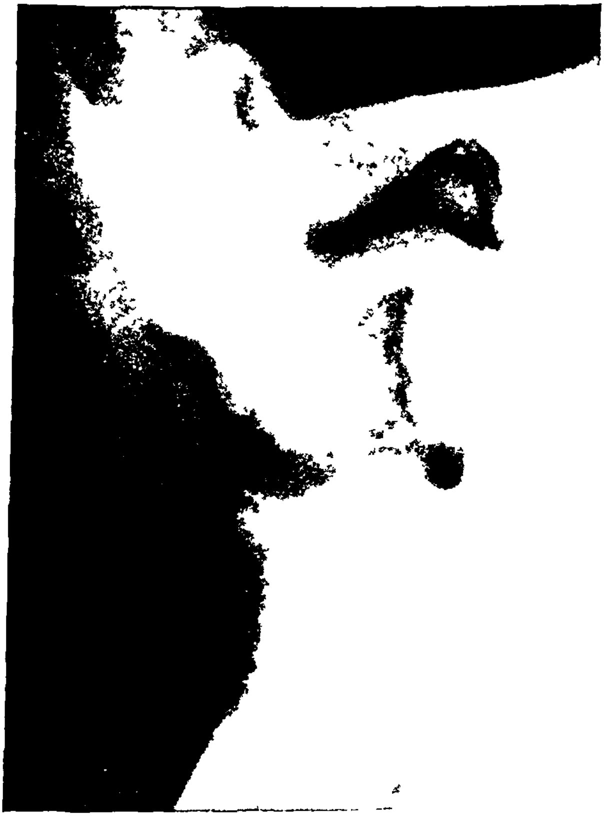

Fig 6 Lateral laryngeal radiograph during quiet respiration in a case of ventricular dysphonia showing normal structures and positions (Case 2)

dilated as a result of high subglottic pressure Since the return of more normal phonatory movements (see Fig 8), the cords close well, and the ventricular folds remain sufficiently apart to allow normal phonation

Case 3 White male, age 60, salesman, attended the N Y Eye and Ear Infirmary for several years In May, 1958, he complained of increasing hoarseness which began six months before. He does not smoke, but talks much, and loses his voice when he gets excited. He sells ladies' hats and is disturbed by lint and dust.

Previous History. Appendectomy age 13, typhoid fever age 25, tonsillectomy age 45. Had chronic sinus trouble requiring several nasal polypectomies and sinus irrigations. For the last five years he suffered from gastric ulcer, treated with diet and medication.

Examination revealed a depressed man, looking older than his age. The nasal mucosa had the so called allergic appearance and several small polyps were seen in both middle meatus. Sinus X-ray showed cloudiness of antra and ethmoids. The vocal cords were red, dry, swollen and hypertrophic. The ventricular folds appeared hypertonic and closed completely during phonation thus obstructing the true cords. Accordingly the voice sounded very rough and low with an average speaking pitch around F (87 cps) and a short phonation time (o/F = 6 sec).

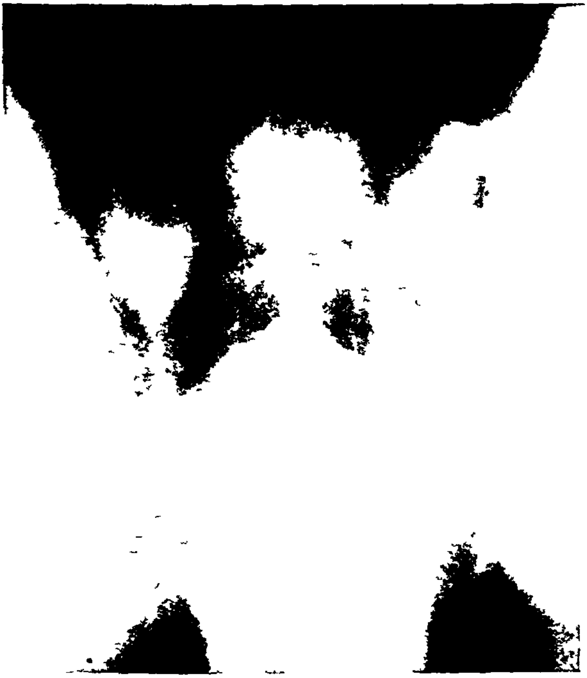

Fig 7. Frontal laryngeal tomograph of ventricular dysphonia. Note compression of ventricles, over-adduction of ventricular folds and dilatation of trachea during faulty phonation (Case 2).

Psychological Evaluation. Has been married since 1920, two sons are married and well. His wife is nervous and excitable. One year ago his daughter in law developed a psychosis following childbirth. She has fits of depression, indulges in compulsive rituals, cries often without reason and refuses to take care of her baby which had to be placed in the patient's home Her visits in his apartment usually produce quarrels, so that he refuses to see her He also had several financial losses, during the depression, after the last war, and six months ago when some speculative stocks failed He had bought them five years ago, raising the money by selling his house When confronted with the coincidence of his gastric trouble with the investment in these speculative stocks at the price of his house, and of the onset of dysphonia when he lost his money, the patient admits the emotional element of his ailments In conclusion, this man states that he is far from satisfied with his life, deplores the loss of his economic security by imprudent speculations, and resents the necessity of visiting a hospital clinic

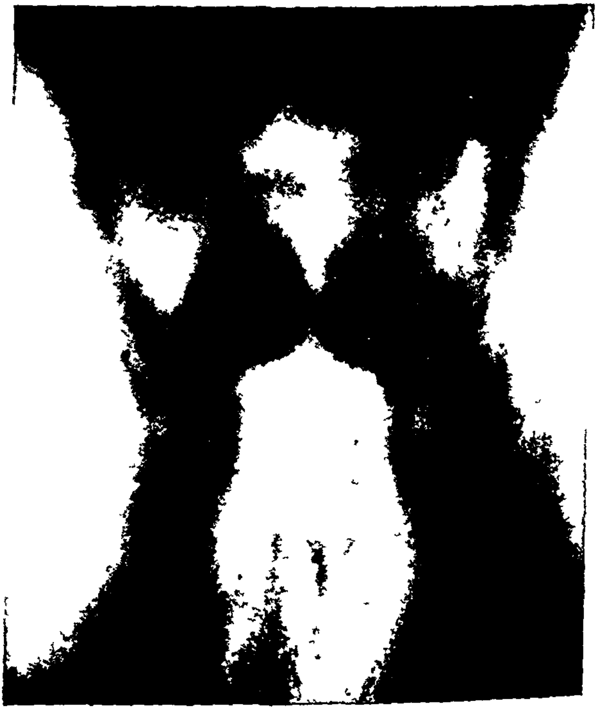

Fig 8 Frontal laryngeal tomograph of ventricular dysphonia After improvement of the faulty phonation pattern the vocal cords close well *the ventricular folds are less approximated* and the laryngeal introitus remains wider (Case 2)

Diagnosis Psychogenic ventricular dysphonia associated with other psychosomatic disorders (allergy, gastric ulcer), resulting from a tension anxiety state

Radiologic Analysis Frontal tomographs taken during *phonation* (see Fig 9) show marked *hypertrophy of the vocal cords* and of the ventricular folds Both are firmly closed The ventricles of Morgagni appear as faint slits That on the right side is seen on a lower level than on the left which is not explained by the slight tilting of the thyroid cartilage to the right side The trachea is markedly widened by high subglottic pressure

Treatment had to be confined to antiallergic medication, symptomatic care of the throat, and brief counselling on the management of his voice Since the patient was too preoccupied with his financial and domestic problems, he refused a course of vocal rehabilitation and was also disinclined to seek psychiatric care

Fig 9 Frontal laryngeal tomograph of ventricular dysphonia showing compression of the ventricles firm closure of the hypertrophic ventricular folds and dilatation of the trachea (Case 5)

TREATMENT

In all cases of vocal disability due to disorders of function, the choice of appropriate therapy depends on the individual somatic and emotional findings Obviously, the six types of ventricular dysphonia require different management

Vocal rehabilitation, based on the judicious application of orthophonic principles, is indicated in the majority of cases which present the habitual type resulting from constant vocal misuse Providing the patient demonstrates sufficient motivation for his share of the rehabilitational effort, satisfactory results can usually be achieved. The necessary techniques of vocal re-education must be studied in theory and practice for they cannot be improvised.

Psychotherapy, either by the expert psychiatrist and clinical psychologist, or offered in a supportive manner by the experienced phoniatrist, represents an integral part of the rehabilitation program. It is of primary importance for the *emotional* type of ventricular dysphonia.

Physiotherapy may be helpful in selected cases, especially those of the paralytic or central types. This includes vibratory massage of the neck for the reduction of muscular overcontraction, heat for improved circulation, or for reduction of subjective complaints such as pain, tension, or globus sensation, and the special techniques of isochronic tone massage which aid laryngeal compensation by promoting the auditory, tactile, and kinesthetic pathways for homeostatic feedback; however, electric stimulation is usually contraindicated because it tends to augment the neuro-muscular hyperkinesis.

Local laryngeal procedures may be used in a variety of techniques. Flatau[15] and H. Gutzmann, Jr.,[19a] advocated the use of a special spreading forceps. After local anesthesia, the branches of the forceps are introduced between the overcontracted ventricular folds, spreading them apart to allow free vibration of the vocal cords. Some patients then learn to maintain the re-established function of the cords. Rethi[35-36] prefers novocain injection into the palato-styloideal fold of the stylopharyngeus muscle by the endoral route. The value of this method was recently confirmed by Beckmann.[8] Sometimes, simple surface anesthesia of the larynx may be sufficient to break the faulty pattern of phonation.

Surgical procedures should be reserved for obstinate cases. Jackson and Jackson[27] recommended the excision of the hypertrophic portion of the ventricular folds. Rethi[35,36] proposed the surgical excision of the stylopharyngeus muscle by the cervical approach.

While all these techniques serve for the abolition of the

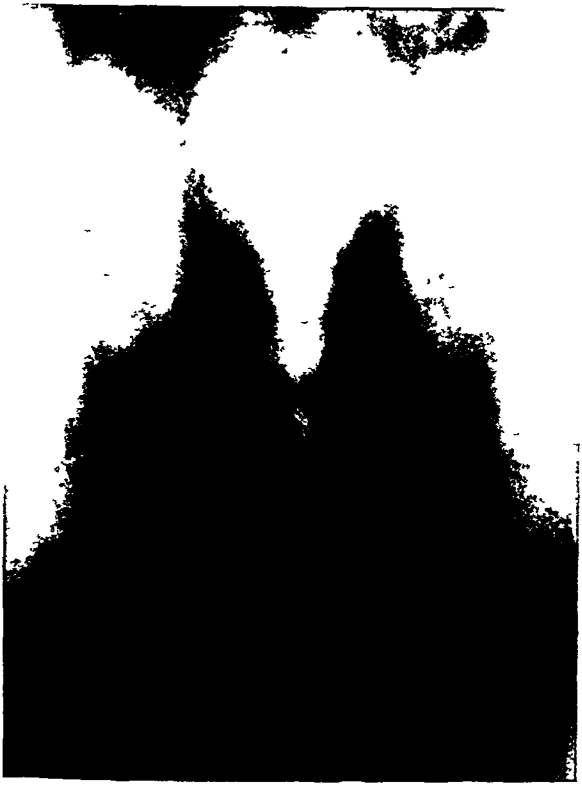

Fig 10 Frontal laryngeal tomograph of a normal male (baritone) while humming a high tone in head register Note the sharp edge of the cords the wide open ventricles the abducted ventricular folds and the wide supraglottic resonance chamber

undesirable types of ventricular dysphonia (types 1 to 5), the *vicarious type* is systematically promoted to replace vocal cord function after it had been lost by trauma or surgical intervention The development of vicarious ventricular voice is based on the techniques of orthophonic re-education and may be assisted by physiotherapy as mentioned above Further details were discussed in previous works[3,24] Surgery can be of service when protruding scars or granulations need ablation for smoothing the contours of the phonating parts

Prognosis is usually excellent when the therapeutic situation is satisfactory Therapeutic results were analyzed in a previous monograph.[3] Occasional failures are due either to diagnostic errors, or to the resistance of deeply neurotic patients. Therapeutic progress may be corroborated by radiologic control which should show a return to more normal conditions (comp. Fig. 10).

CONCLUSION

Although the phenomenon of phonation with the ventricular folds has been known for a century, and although careful studies of ventricular dysphonia were submitted by various authors during the last 50 years, this frequent disorder of the speaking voice is usually neglected in clinical practice. The faulty habit of using a primitive laryngeal mechanism for self-expression and communication produces secondary organic changes of laryngeal structure. Often, these visible alterations of laryngeal appearance (secondary or traumatic laryngitis) are misinterpreted as the *cause* of the vocal disability, while in reality they represent a secondary *result* of the primary vocal disorder.

In the light of present knowledge, all types of hyperkinetic phonation reflect a regressive psycho-dynamic phenomenon. The underlying primitive sphincter mechanism of the larynx may be activated by secondary subcortical centers of phonation. These can take over the function of the primary cortical centers of phonation when the latter become inhibited by the unconscious withdrawal of an emotionally maladjusted personality to the more primitive mechanisms of neurotic aggression and hostility.

Since, then, the "laryngitic" appearance of a larynx traumatized by ventricular dysphonia must be differentiated from true infectious laryngitis, such patients cannot be treated successfully, unless the actual underlying cause of their vocal disability has been ascertained. This differential diagnosis between true and secondary laryngitis should consider the six main types of ventricular dysphonia, namely the habitual, emotional, paralytic, cerebral, cerebellar, and the vicarious forms.

SUMMARY

The frequent problem of ventricular dysphonia is briefly discussed by first surveying the large amount of pertinent literature The laryngoscopic findings of secondary laryngitis must be distinguished from true laryngitis Correct diagnosis is established by history, analysis of laryngeal and vocal function, and consideration of the patient's personality

Since ventricular dysphonia occurs in at least six types, treatment depends on the underlying cause Three typical case histories illustrate the usual findings Laryngeal tomography is valuable for the demonstration of hyperkinetic laryngeal function The theory is advanced that laryngeal sphincter action during phonation represents a phenomenon of laryngophonic regression which the psychiatrist can utilize for his psycho-dynamic interpretation of the patient's disturbed behavior

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THE PSYCHOLOGICAL MANAGEMENT OF THE SURGICAL PATIENT VOODOO, MESMER AND ALEXANDER POPE*†

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INTRODUCTION

The subtitle of this essay contains three items designating the headings Under Part 1, Voodoo, I will attempt to convince you of the basic reality that psychological factors can have profound influence, including physical effects, on the surgical patient—really all patients—and the practice of surgery For example, Walter Cannon, the famous physiologist, collected a number of well-authenticated cases in the literature under the title "Voodoo Death " Individuals *believing* themselves hexed or hoodooed have died in a matter of hours or days without discernible organic explanation Of course, death by whatever cause ultimately is organic or physical, however, the reasons for death and the mechanism in the cases above are unknown

Part 2, Mesmer, will be concerned with the personality of the physician, and particularly the physician's beliefs or convictions, as one of the most important influences, for better or worse, on the patient

Part 3 is an elaboration on two lines from the work of the poet Alexander Pope

"Know then thyself, presume not God to scan,
The proper study of mankind is man"

The admonition "Know thyself" has been ascribed to at least six other people, but Pope's stanza best suits my purpose,

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also pertinent is the proverb "Physician, heal thyself," a reference which St Luke ascribed to Jesus Granted the fact that profound influences are possible by psychological methods, and that the physician is, himself, strong medicine, then both quotations summarize my thesis—that if a physician is to use his medical knowledge most effectively in his practice, he must really understand himself Knowing himself is the most difficult part, but it is the cornerstone of any intelligent, rational, and effective use—and explanation—of psychological principles in the management of *any* patient

PART 1—VOODOO

Many physicians pay lip service to the concept, even truism, that psychological factors are important in the general practice of medicine, or at least will acknowledge particular situations in which it is important, yet, their conviction is usually not very strong or is considered inapplicable to their own practice For example, if a patient has a headache which benefits from a "sterile hypo" or some other placebo, the implication is that the headache was not real, that is, was due to "psychological factors only" A placebo can affect the headache caused by cancer as well as the one caused by nervousness or tension Just as a placebo can relieve pain of organic origin, so can it alone produce marked physical changes and alter physical illnesses Good examples of this are found in Stewart Wolf's article[1] on marked toxic reactions from placebos, including urticaria, generalized collapse and a disseminated dermatitis Whatever an individual's illness, the illness and the patient can be conceived of only in terms of complex psychological, physiological, and environmental factors As an example of this last statement, think of a not necessarily hypothetical case in which a man and his wife have a heated argument resulting in his leaving the house and visiting several bars He becomes intoxicated, wanders about the streets in freezing weather—and ultimately develops pneumococcus pneumonia and a lung abscess requiring surgery In such a case, what would you say caused the pneumonia and the abscess? Psychological factors are most often just one facet of the genesis of the illness or its manifestations

Few physicians doubt that such things as Christian Science, or some other strong belief similar to Voodoo, can affect people with functional or psychological problems, however, most doubt that those who have a "real illness," an organic problem of some sort, can be helped by so-called mystical, superstitious, or unscientific influences. If the effect of such influence is acknowledged at all, it is considered pertinent only in the sense that psychological problems can make an organic illness worse. I am convinced that whatever one believes in strongly enough can produce functional and physical changes both in the direction of pathology—disease—as well as in the direction of recovery—health. Although my personal experience with Voodoo is limited at this time, our study of Voodoo here in Mississippi would confirm my impression.

I have used various forms of suggestion and/or hypnosis for years in the treatment of warts with excellent success. I started to use this method after I had read an article in 1948 by a dermatologist on the treatment of warts with such measures. I remember well that my grandfather used to bury pennies, and folklore is full of a variety of successful methods, to remove warts. Although warts are considered of virus origin, in two recent articles Ullman[2,3] documented the fact that in many clearly discernible instances the resolution of warts can be brought about by psychological means such as suggestion or hypnosis. Along this same line, Dubois said repeatedly that the tubercle bacillus, although *necessary*, may be one of the least important factors in the patient with tuberculosis, and this includes etiology.

Many experienced surgeons and anesthetists will freely admit that the patient who does not wish surgery or who feels that something "bad" is going to happen is a poor risk, particularly if his convictions seem fixed and strong. Some of these patients die without adequate explanation. I am reminded of several such cases which I encountered during the Korean War. In one instance a soldier was brought in who had a shoulder wound of no great consequence but who had no desire to live. His injury occurred during the Winter of 1950, when the fighting was particularly fierce, and there was little opportunity for psychiatric treatment for the man In a matter of days he died, and autopsy revealed no adequate physiological cause of death Additional proof of the power of the mind lies in the fact that thyroidectomies, hysterectomies and other operations can be performed with no anesthesia whatsoever other than hypnosis

If I have seemed to belabor the obvious, forgive me, but ten years' experience teaching medical students and graduate physicians has convinced me that there seems to be little individual personal conviction or understanding, and rarely utilization, of such truths

PART 2—MESMER

In the preceding section the importance of psychological factors, *per se*, and particularly the patient's belief and conviction have been emphasized The secret of whether voodoo will work lies in the fact that the individual who is hexed believes firmly in hoodoo and expects results therefrom The "conjurer-doctor" need not believe in hoodoo, but he must believe that it will exert a change, that it will influence the individual In other words, he doesn't need to believe it works by reason of magic, but he must expect results In this section I will be concerned with the physician's beliefs and convictions and their influence on the patients, and with the importance of the setting or trappings—call them stage effects if you like Mesmer and his career very nicely illustrate these concepts One other point—as wrong as he was about the how of what he was doing and the theory behind it, he was very right about the fact that it worked well, thus, he was very successful in its application

Franz Anton Mesmer lived from 1734 to 1815 His doctor's thesis in 1756, when he graduated, was on "The Influence of Planets" He believed that it was possible to effect cures with a magnet through the mechanism of what he termed animal magnetism Mesmer carried out his treatments in a darkened room where a group of people were seated around a large tub or baguet, from which iron bars protruded By his touch, he professed to charge these bars with animal magnetism, which flowed through the circle of people when they grasped the bars Mesmer would appear in a lilac robe carrying a wand He would touch the baguet with the wand and immediately the people in the circle would become agitated and begin to twitch and writhe Mesmer's treatment was called mesmerism and it soon became synonymous with hypnosis One of Mesmer's disciples, Puységur, rediscovered hypnotism, which in one form or another has been known for many centuries Mesmer has been dubbed a charlatan or quack, which I think is distinctly unfair, because he was a physician and went through the conventional training of that day and age After all, our medical colleagues espouse and employ many so-called scientific theories which are basically incorrect, yet this fact, *per se*, does not make them charlatans To be sure, Mesmer's explanation of his treatment-method was incorrect, but no one who wishes to study his influence on that day, and no less upon succeeding generations, can deny this influence

While I am on the subject of quackery, I would like to state my belief that in some ways we in medicine are responsible for the success of present-day charlatans or quacks We are too dependent upon science, and we belittle faith and psychological influences Charlatans can and do *help* people suffering from all kinds of ailments and illnesses, both organic and functional—and I state this categorically The danger is that these healers are untrained and unable to know what they cannot help or when they are not helping Consequently, precious time may be lost, as for instance in such conditions as cancer Another fact is very important here namely, for many people it is much more acceptable to have, for example, the chiropractor's adjustment for whatever ails them rather than to face the fact that theirs may be a psychological or psychiatric problem, particularly if referral to a psychiatrist may follow, yet this not unexpected attitude toward psychiatric referral is precisely why all physicians must be able to apply many psychiatric principles and practices The only resources the charlatan has are psychological The physician can utilize the same psychological resources *plus* all available scientific knowledge and methods for treating all facets of most illnesses, *plus* the prestige of the medical profession When necessary the psychiatrist can be utilized for advice, consultation, or referral Skillful referral is so important I could spend my whole time on the subject Discuss this with those psychiatrists to whom you refer patients

Sadly, doctors seldom miss the physical or organic facets of illness but badly neglect the others Kaufman and Bernstein[4] documented this point in rather convincing fashion Mount Sinai Hospital has a diagnostic clinic for local physicians' problem patients whose income is between $5,000 and $8,000 For a flat fee the patient receives all the studies necessary, including laboratory work and consultations with various specialists All facets of medicine are represented, including psychiatry In 1,000 unselected patients studied in this clinic 81 4 per cent had diagnosable psychiatric syndromes, and in over one-half of this 1,000 patients, the psychiatric trouble was the major or exclusive problem As psychiatrists, Kaufman and Bernstein echo the refrain "Emotional illness can and should be a positive diagnosis, not one of exclusion" It should be made by positive criteria rather than being the only diagnosis left when everything else is ruled out

To summarize this part then, let me emphasize that the physician is by far the most powerful medicine available today for the patient The old statement that the "operation was a success, but the patient died" may be nearer the truth than we like to think For in surgery there are many, many excellent technicians with superior operating skill The really great surgeons are also great physicians, albeit often unconsciously, I regret to say That is, many of them have tremendous personalities which greatly affect their work and their patients Sometimes, like Mesmer, the results are good, but the physicians' analyses of why are incorrect Many of them would be ashamed to use a placebo but will give this or that favorite prescription to the patient with the expectation that the patient will benefit from it—and the patient does, but in many cases the improvement had little to do with the medicine given

Stewart Wolf[5] illustrated this point in the field of surgery Wolf, Professor of Medicine at the University of Oklahoma, discussed a surgical meeting in the 1930's where two surgeons were reporting on their experience with gastroenterostomies. Dr. D, who was very much in favor of the operation, reported favorable results with the recurrence of marginal ulcers in only 1.6 per cent in his series. Dr. B, who had abandoned the procedure and had never been much in favor of it, reported that from the observation in his series there was 33 per cent incidence of marginal ulcers. The only explanation possible for this much difference is the personal beliefs of the individuals and the influence of those surgeons on their patients. At this point it would be well to reiterate Dr. Wolf's admonition, which I endorse enthusiastically, that in emphasizing the power of the doctor-patient relationship we be fully aware that it isn't the *only* good treatment and that it is only one facet in most cases.

Harley Williams,[6] an Englishman, wrote a book called "The Healing Touch," a series of biographical sketches on various physicians. On the jacket of the book there is a statement that "The thread running through this book is that of personality." The author has this to say, "At heart, we all believe in the healing touch. It is no mere sentimental phrase but a powerful reality. We may not possess this gift, but we have certainly experienced it at the hand of others. How are we to define it? Something nearer magic than we are likely to admit. A sort of magnetic force in the personality that puts fear to rest, releases the restless currents of our being and harmonizes them in a new direction. Some people have it, the majority do not. This powerful human faculty is felt—in ordinary affairs, in politics, in business, but its most natural place is in the ancient art of healing, and in the medicine and surgery that have grown therefrom. It is fundamental today just as in the simpler medicine of a previous age."

Apropos of this last statement, the observation has been made that when doctors could cure only a few ailments they were often held in high regard, now in an age of therapeutic excellence they often have to defend themselves. The possible explanation for this is that physicians and surgeons have become so dependent upon the miracle drugs and their vast technical accomplishments that they have neglected the interpersonal and cultural factors in the practice of medicine, that which is often called the art of medicine I am not in complete agreement with this latter designation, the art of medicine, for I think that it is coined to explain in somewhat mystical fashion why some doctors are so adept and successful in their medical or surgical practice I believe that there are more scientific explanations for their success with their patients

PART 3—ALEXANDER POPE

To repeat, "Know then thyself, presume not God to scan,/ The proper study of mankind is man" If you will accept the fact that your personality vastly influences the treatment and results you get, then some other things follow It is incumbent upon all of you to know as much about yourself as you possibly can This does not mean psychoanalysis, nor does it require any really time-consuming action, rather, it requires a willingness to know yourself, and the knowledge that to do so is most important Consider such questions as Why do some patients irritate you and how do you respond to certain patients? What can you say about the way and the manner that you practice medicine and your approach to patients? What kind of "bedside manner" do you have and are you reasonably sure that you are making the most effective possible use of yourself? What kind of conditions and diseases can you treat best, and with which do you have the best results? The answers to these questions and the process of such scrutiny could and should improve the efficacy of your practice considerably To suggest that the de-sensitization procedures in your allergic patients may not be wholly scientific need not interfere with your result What do you, or the patient for that matter, care whether the scientific explanation is satisfactory, if the treatment works? Almost anything can be good medicine and good practice of medicine if it does not harm This is the important thing How many procedures in your field of E E N T meet either criteria, and particularly the latter one—do no harm?

As an illustration of this in another field of surgery, Allbutt, in 1884, in his lecture on visceral neurosis made the following statement: "A neuralgic woman seems thus to be peculiarly unfortunate. However and repeated may be her visceral neuralgias, she is either told she is hysterical or that it is all uterus. In the first place she is comparatively fortunate, for she is only slighted; in the second case she is entangled in the net of the gynecologist who finds her uterus, like her nose, is a little on one side. Or again like that organ, is running a little, or is as flabby as her biceps, so that the unhappy viscus is impaled upon a stem, or perched upon a prop, or is painted with carbolic acid every week in the year except during the long vacation when the gynecologist is grouse shooting. Her mind thus fastened to a more or less nasty mystery becomes newly apprehensive and physically introspective and the morbid chains are riveted more strongly than ever. Arraign the uterus and you fix in the woman the arrow of hypochondria. It may be for life." As a complete aside, I am always delighted but somewhat jealous when reading articles written by our British or commonwealth colleagues. Witness this short opening paragraph from a recent article: "It makes an unpromising start to confess, as I must, that this clinical study hinges on a patient whom I never met. In fact, I was on my sick bed when her impact was foreshadowed and there I had to lie, in another part of the hospital, when she made her passage through the psychiatric department."

How else might you benefit from taking a closer look at yourself? An ophthalmologist told an elderly, somewhat poor-risk, cataract patient the following: "If you make the slightest move during the operation I can't be responsible for the outcome. This is a difficult operation. You must hold 100 per cent still." Very likely the surgeon, aware of the poor prognosis, made this statement so that he would not feel responsible (and guilty) if the operation was unsuccessful. I do not intend to impugn your motives or question your honesty; I only ask that you realize that it is necessary to look behind the obvious and to be aware of the various mental mechanisms that operate in all human beings.

Dr. Levine says that "patients are not members of a separate species but rather are slightly varied examples of human beings And, second, patients are those men, women and children who, because of a hope of a cure or the lessening of their stress, are more willing than others to reveal the truth of their secret lives and the essence of their humanity Hence, the intensive and extensive study of patients has led to a greater understanding of men and women in general"

In observing and studying yourself, not only in the context of your surgical practice but in your home and social life as well, you may benefit in various ways, not only in the practice of medicine at times by finding some unknown and neglected item of mental hygiene and enlarging your range of opportunities for action within yourself or your patient You may uncover some self-defeating trends or paradoxical personality needs which lead to frustration and lack of satisfaction You may find yourself rushing like "heck" but the reasons may not be those that you think It may be that you are running away from yourself The next time a patient broaches a topic on which you find yourself about to reassure him or interrupt him, since you note his anxiety, stop yourself Let him go on Could it be that this subject is anxiety provoking to you? If so, you should know it Anxiety is very helpful in that it is the motivating force that compels the patient to talk or to behave in such fashion and in so doing gives you much information about his problem, likewise, the absence of anxiety in a patient, when discussing some aspect of himself or some topic that logically should be anxiety provoking, may give you important insight into the patient and his attitude toward his illness For example, the presence or absence of anxiety in a patient preoperatively is not, *per se*, necessarily helpful in your prediction of what will happen during surgery Consider the presence or absence of anxiety in the context of the entire situation By the term *entire situation* I mean not only the personality of the patient, the doctor-patient relationship, the attitudes or beliefs of both physician and patient, but also the relationship of the patient's family to the illness, its effect on the family, and the possible advantages or disadvantages derived by the patient from the illness For example, if a patient has an ulcer, it vastly affects the whole family, and in a real sense, it is the family's ulcer

Identification is one of the primary mental mechanisms that often get a surgeon in trouble. For instance, one of his patients may be an individual who is a so-called pillar of the community[8] Failure to detect physical illness is upsetting to the doctor, himself a pillar, because serious questioning or attempts to discover emotional or psychological problems, the doctor thinks, might be considered an insult to the patient, since the doctor himself would be insulted by such an implication or approach. Doctors often do not trust themselves to deal with a problem when it seems to be clearly an emotional one or when emotional factors are paramount. Parenthetically, it is interesting that many physicians are very good at handling emotional problems in an intuitive fashion or for empirical reasons, yet, when they begin to analyze why, they get a kind of inferiority complex and often are not nearly so successful thereafter in controlling such problems. Somewhat analogous is the story about the thousand-legged worm who was asked how he controlled all those legs. At this point he couldn't. The patient with his problems and difficulties may activate similar difficulties, or at least difficulties in the same sphere, in the physician, forcing him to defensive maneuvers to avoid the anxiety engendered in himself. These maneuvers vary but, at the least, may involve changing the area of inquiry or the nature of the relationship. It is conceivable that the changes necessitated by the physician's own psychological reactions might render the treatment less effective for the patient than it otherwise might have been.

Let me reiterate in this way. It is obvious that both the doctor and the patient bring to the situation their own individual personalities. In addition, the doctor possesses superior knowledge and experience in the medical aspects of the situation. It should be pointed out that this meeting takes place in a certain cultural setting and that each individual is a product of his culture and responds as such. Most patients go to a physician with the idea that he will find some illness or abnormality that he will in turn do something about, probably by the use of drugs or other specific treatment, furthermore, the patients feel that they have had very little to do with becoming ill and will likewise have little to do with getting well That is, the doctor is viewed in an omnipotent role, and the patient usually longs for the doctor to live up to this role The physician's own psychological make-up will determine what his reaction to this role will be

Briefly, in the three preceding sections of this paper on the psychological management of the surgical patient, I have said to you that

1 Experience has proven that psychological factors can and do enter into the genesis, development, and recovery of virtually all illnesses heretofore thought to be of an organic nature The surgeon, like any other physician who concerns himself with only the physical factors of his patient's illness, deprives himself of much important information about the illness and deprives the patient of an essential ingredient of all types of treatment—namely, the psychological factors

2 The personality and beliefs of the surgeon can and do influence the results he will get from certain treatment The doctor-patient relationship, controlled in part by the physician's personal beliefs, sometimes determines the outcome of the treatment

3 The surgeon, like all other physicians, needs to know himself as a person and as a physician, for his own mental and emotional mechanisms, his own anxieties, frustrations, identifications, and means of escape strongly influence his own life and that of all patients whom he treats

To these ideas, which are so basic and well-known that some would call them truisms, you may respond with "Why, yes, I believe what you have said is true I have always believed that, but so what?" Actually, my talk could well have been entitled "That doesn't apply to me" —If on the other hand, you do accept these concepts personally and find that some parts of this paper do apply to you as individuals and as surgeons, the application of these principles will be of immense value to you

CONCLUSIONS—OR—WHY, HOW AND WHEN PSYCHIATRY

For your purpose, think of psychiatry from two separate aspects. In medical education, psychiatry courses give a general idea of the specialty of psychiatry, that is, when to call in a psychiatrist and what the provinces of psychiatry are. Equally important is the responsibility of psychiatry to present skills, insights, and psychological principles available from the subject and task of psychiatry itself.

Up to this time, psychiatry's association with surgery has not been the fruitful association so often true with internal medicine. For example, the literature on the subject is not very extensive, relatively speaking. There is, however, some work that I would like to call to your attention. I think that anybody remotely interested should read the various articles by Titchener, et al., at Cincinnati. They have studied the interaction of emotions and personality with surgical illnesses, treatment and convalescence. They have done this in a scientific fashion and some of the results are most interesting. In a psychiatric survey of 200 randomly selected and representative surgical patients admitted during one year to a general hospital, there was an incidence of 22 per cent of psychoses.[9] The investigators present other findings and suggestions for interpreting the data gathered. All their papers have been published within the last five years.

The literature also includes an article by Litin[10] in the *Jour. A. M. A.* on preoperative psychiatric consultations. As reported there, the psychiatrist's special skills contributed in a variety of ways to the benefit of the patient and the surgeon. Litin pointed out that patients with a diagnosis of psychosis are often not so brittle or such poor risks as they are thought to be; in fact, in some schizophrenic patients the operation may be followed by a remission, the cause of which is obscure. He emphasizes the fact that positive criteria can be used for the psychiatric diagnosis, not just making the diagnosis by exclusion.

A recent case of my own will illustrate this. I was asked to see a 45-year-old white man with unexplained abdominal pain so severe that he was addicted to morphine. Briefly, the pertinent observation was that this man had made an excellent adjustment in his life in every way and seemed to be well adjusted individually up until six or eight months ago, when he began to have this abdominal pain and some personality changes At the time I saw him he presented many of the aspects of an addicted person as well as showing a great deal of regression in his behavior It was my opinion that psychiatric problems were not responsible for this man's pain, nor could they account for the psychological picture we saw at this time That is, both the pain and the man's psychological state were secondary manifestations of some organic process Exploratory operation revealed a pancreatic tumor Among older surgeons there is an aphorism, perhaps unknown by some of their younger colleagues, that with the association of unexplained abdominal pain and sudden personality change one should suspect cancer of the pancreas The change in the personality does not seem to be related just to pain from the cancer but other explanations are not very helpful

By very similar means—that is, the absence of positive psychological findings—I was able to diagnose, or to suggest the diagnosis of, an islet cell adenoma of the pancreas which was proven by surgery Sometimes a psychiatrist must exercise a great deal of effort in holding to his opinion There is nobody so tenacious as an organically minded physician who, having looked long and hard for organic illness and being unable to find it, becomes convinced that the problem is psychiatric One such case I saw recently had had numerous tests and even an exploratory operation because of peculiar, unusual abdominal complaints He developed a severe depression which responded to some of the newer energizers, but he just did *not* fit the picture of a psychosomatic problem, and his physician was so informed Nothing was done until he developed additional complaints, at which time an abdominal cancer was found These developments were certainly no reflection on his physician, for I had made no attempt to diagnose his illness other than to rule out psychiatric problems Obviously, there are many cases in which this method of diagnosis could not be applied, *viz* , those patients who have coexisting psychiatric and physiological symptoms

In ophthalmological surgery, the field in which some of you practice, psychiatric research from a number of different projects and sources, has converged to help explain an observation that you made a long time ago concerning the postoperative psychoses seen with cataract surgery, which in the past has been followed by psychoses in as many as 7 per cent of the cases With improvement of surgical technique the incidence has been reduced tremendously, and it is possible to decrease this even further Ziskind[11] has written a very nice article pulling together the various scientific studies on the result of different kinds of isolation stress in human beings and the relationship of this stress to conditions in which the major sensory systems were interfered with, such as the eye and the ear Greater understanding of so-called sensory deprivation or sensory patterning, as studied by D O Hebb, J C Lilly, and J T Shurley, has been very helpful in elucidating certain situations encountered in eye surgery particularly

One of the first consultations that I had with a member of this audience was a telephone conversation about a postoperative patient of his I am not certain about all the details, but the case involved cataract surgery in an elderly individual whose hearing was impaired and who developed, postoperatively, disorientation, confusion and other signs of an organic psychosis I think the physician thought me a bit flippant, or worse, when my advice was *a* to cut down on sedatives and/or tranquilizers (the central nervous system already being depressed from the anesthesia and further chemical depression therefore adding to the total problem), *b* to turn the lights on in the room and unbandage the eye as soon as possible, if that had not already been done, *c* to install a radio in the room and leave it on at all times, *d* to have someone from the family, someone he knew, there to talk to him In other words, to give him as much sensory stimulation of various sorts as possible I believe that the man was reoriented in a few hours and remained so I have no doubt, because I have seen it in other similar cases, that this disorientation and confusion were directly the result of factors that I have suggested The attending physician was well aware of the importance of unbandaging the eye, but I think his case represented an accumulation of sensory deprivation or isolation in a central nervous system already depressed, and that the patient lost contact for these reasons

At this point I would like to repeat my statement that in many cases the psychiatrist is not only unnecessary, but the surgeon will be able to do a better job than could the psychiatrist As previously stated, bringing in psychiatry is often thought by the patient to be an insult, a threat, or worse The surgeon has much of the family information and other data, and this, combined with the already established rapport of the primary doctor-patient relationship, is often all that is necessary It also keeps the physician from *passing the buck*, so to speak The comment I keep hearing is that psychiatric consultations take just too much time I would be the first to admit that psychiatric diagnosis and treatment most often is a time-consuming affair, however, such specialized treatment is often not necessary and there are some short-cuts that will help you in many ways Winn,[12] in a recent issue of the *Jour A M A* , presents some principles for a brief psychiatric evaluation by the surgeon You can determine *a* what meaning the disease has to the patient and particularly the anxiety associated with the illness, *b* how the patient's symptoms alter his life, *c* whether the patient has adjusted to the reality of the therapeutic situation (Is this a defense mechanism and what are his defense mechanisms?) If this sounds difficult, I had better repeat it, because it can be approached on common-sense grounds *a* you should arrive at the meaning of the disease or the illness to the individual, *b* you need to know the result of the patient's symptoms in relation to his life, or how they have influenced and will influence his life, *c* and finally, you need to consider how he has adjusted to the illness and to the treatment proposed—whether he is too calm about something that is very serious or whether the reverse is true

The need to view the entire situation was noted in 1883 by Oliver Wendell Holmes, who gave voice to some of this concern by saying, "I have often wished that disease could be hunted by its professional antagonists in couples—a doctor and a doctor's quick-witted wife. Many a suicide would have been prevented if the doctor's wife had visited the victim the day before it happened. She would have seen in the merchant's face his impending bankruptcy, while her stupid husband was prescribing for his dyspepsia and endorsing his note. She would recognize the love-lorn maiden by an ill-adjusted ribbon—a line in the features— a droop in the attitude—a tone of voice—which means nothing to him."

From all sides we are hearing more about the whole person and about treating not just the disease but the patient, etc. This is always good advice. Likewise there is something to be gained from the writings of Dale Carnegie on "How to Win Friends and Influence People." It is also important to think positively and to appreciate the power of positive thinking. All this is sometimes good advice. I hope, however, that if I have done nothing else in this essay, I have avoided all of this kind of advice, which embodies rather shallow philosophies, oversimplifying the complexities involved in the living of a satisfying, productive life in spite of perhaps many obstacles. My objection is that these erstwhile panaceas constitute part of the block that prevents good psychological principles from being utilized by surgeons, and others, or from influencing the patient. Both of them, patient and surgeon, think their acceptance of these popular concepts indicates that they are psychologically sophisticated and that this is what is necessary. If reason, logic, good manners, lots of attention, rest and vacation, reduction of environmental tension, and the like, don't help, then there just isn't anything wrong with the patient, and there is some kind of irresponsibility or lack of cooperation on his part—or this is simply a bad patient. We owe more to our patient than an attentive ear and good advice. Ours must be a discerning ear, and we must be capable of understanding and explaining after we have listened. Above all, we must be capable of establishing a doctor-patient relationship that is a corrective and satisfying interpersonal experience for the patient.

Perhaps the biggest mistake we make is in the idea of selling the patient on the idea of living with his illness. It is one thing to *face* an illness or disability It is another to accept that condition as a permanent handicap And the placid acceptance I refer to implies not only lack of hope but also unwillingness to compensate for the loss of function The only way to live with an illness is to fight it, always The blind cannot see and the deaf cannot hear, but they have other senses with which they can compensate for their losses—with which they can *fight* their losses That is, *if* they have spirit and hope As Kant said, "Take hope and sleep away from man, and he is the most miserable creature on earth"

At the risk of seeming contradictory, I must emphasize that in some quarters there is a tendency to overdo the psychiatric approach It shouldn't be necessary to remind you that attention to psychological causation should not prevent an adequate diagnosis and emphasis on physical factors Our scientific knowledge has been too dearly won to discard My plea is that we combine all of our knowledge in the practice of comprehensive medicine, regardless of specialty

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2500 North State Street

COURSE IN LARYNGOLOGY AND BRONCHOESOPHAGOLOGY

The Department of Otolaryngology, University of Illinois College of Medicine, will conduct a postgraduate course in Laryngology and Bronchoesophagology from March 13 through March 25, 1961, under the direction of Paul H Holinger, M D

Registration will be limited to 15 physicians who will receive instruction by means of animal demonstrations and practice in bronchoscopy and esophagoscopy, diagnostic and surgical clinics, as well as didactic lectures

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk St, Chicago 12, Ill

MULTIPLE PRIMARY MALIGNANT TUMORS

Phillip A Dibble, Captain, MC, USA,[*]
and
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Washington, D C

In 1879, Billroth[1] first described multiple primary malignancies and proposed three conditions which had to be satisfied before that diagnosis could be established

1 Each tumor must have an independent histologic appearance

2 Each tumor must arise in a different situation

3 Each tumor must produce its own metastasis

Wallace[2] felt these postulates were too strict, proposing instead the following three postulates

1 Macroscopic and microscopic distinctness

2 Each tumor must grow in an individual way

3 The diagnosis is confirmed by a different nature of metastasis in each case, and the probability of one metastasizing from the other must be excluded

Warren and Gates[3] concur with Wallace regarding the strictness of Billroth's postulates P J Wooley[4] divided multiple primary malignant tumors into four groups

1 Malignant tumors may affect the same tissue and produce identical tumors or tumors of different types

2 Malignant tumors may affect both of a pair of organs The tumors need not be of the same histological type

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3 Malignant tumors may affect different regions of the same system but need not be of the same histological type

4 Malignant tumors may present as different tumors involving different systems

Epstein's[5] classification of multiple primary malignant tumors is as follows

1 The tumors are multicentric if they arise in the same area

TABLE I

Sex Ratio and Occurrence Rate

Date	Sex Ratio Male/Female	Total Tumors	Multiple Primary Tumors	Per Cent	Author
1931	—	950	18	0 2	Hanlon[9]
1932	1/1 6	1,078	40	3 7	Warren and Gates
1933	1/1 2	2,124	71	3 3	Hurt and Broders[10]
1936	2/1	583	46	7 8	Burke[11]
1938	2 2/1	1,411	25	1 77	Kirschbaum[12]
1939	1/1 3	2,500	113	4 52	Stalker and Phillips
1943	—	685	30	4 3	Hellendahl[13]
1944	2 1/1	2,829	194	6 8	Peller[14]
1957	—	3,096	112	9 7	Wallace
1958	—	1,514	24	1 52	Fried[15]

2 They are systemic if they arise in anatomically or functionally allied organs of the same system

3 They are coincidental if they occur as a random and accidental association in unrelated areas

Watson[6] demanded a stricter definition of multiple primary tumor, since he considered that the multiple primary tumor must involve different organs or systems, however, he stands as the exception to the usual thinking of today

Most of the articles written on multiple primary tumors are based upon autopsy studies involving a great number of cancer deaths, and are certainly statistically reliable The incidence of multiple primary tumors, on the basis of European and American data, was 1 8 per cent, and on the basis of American data, 3 9 per cent In a survey of 2,829 consecutive autopsies on cases of cancer from 1932 to 1943 Warren and Ehrenreich[8] found 194 cases of multiple primary malignant tumors, an incidence of 6 8 per cent This was a continuation of an earlier study (see Table I) by Warren and Gates covering the period from 1926 to 1931 in which the incidence of multiple primary tumor was 3 7 per cent They felt that this reflected a current awareness to cancer and a greater tendency to biopsy suspicious lesions for histologic examination

The average age of onset is apparently dependent upon the sex, there being an apparent tendency for earlier onset in women than in men, and these tumors tend to occur at approximately the same age as single tumors (see Table II)

TABLE II

Age of Onset

Author	Age of Onset in Men	Age of Onset in Women
Fried	61 5	50
Warren	67 7	58 2
Kirschbaum	66 0	57 2

Watson found that the interval between the diagnosis of the primary tumor and the development of a second tumor was 41 months Stalker, et al , had a 45 1 per cent occurrence of synchronous multiple primary tumors and a 54 9 per cent occurrence of metachronous multiple primary tumors Of the latter, 26 5 per cent had an average interval of 12 3 years Barrett[16] reports a 3 22 year interval between the primary and the development of the second tumor Peller reported a 3 1 year interval, and Warren and Ehrenreich found a 3 1 year interval between tumors

As indicated in Table I, multiple primary tumors tend to be statistically more frequent in males than in females

Hellendahl stated that the familial incidence is no higher in multiple primary tumors than in single ones, however, Stalker suggested a hereditary tendency and quoted a 26 6 per cent incidence, whereas the range in the literature is 15-30 1 per cent

Some interesting observations on pathogenesis have been made. Hellendahl found that congenital anomalies are more frequently found in cases of multiple primary tumors This relation to congenital anomalies raises the question of whether or not cells giving rise to cancer may in themselves be congenitally abnormal Warren and Gates theorize a congenital predisposition or susceptibility Watson does not believe in *constitutional* predisposition to multiple cancers of different systems, but he does admit that some people do have a marked predisposition to multiple tumors of the same organ He felt that this indicated a response by different parts of similar tissues to the same stimulus but would not consider this as an indication of general predisposition Epstein discussed a biologic accident of cancer visited on one or a group of cells versus a prepared or initiated field, perhaps of considerable extent White proposed a predisposition on the basis of a gene for cancer inheritance, a gene for localization, plus environmental stimuli Fried mentioned a possible endocrine basis He raised the question of coincidence versus a susceptibility to epithelial malignant disease Hurt and Broders, and Bugher[17] doubt the coincidence theories Wooley suggested six possible theories of pathogenesis

1 Are there anomalies of cell division?

2. Are there mechanical developmental anomalies or multiple foci of irritation?

3 Are there abnormal chromosomes distributed in the nuclei?

4 Are there toxins acting in two different locations?

5 Is there a complex influence of an existing tumor or neighboring cells with a predisposition for tumor growths?

6 Are there multiple foci of embryonal displaced or superfluous cells?

Hurt and Broders reported a study which was made to determine the relationship of the grade of malignancy to the frequency of occurrence of multiple primary tumors They concluded that if a person has one tumor, the probability of his developing another varied directly with the grade of the first malignancy They hypothesized that the average grade of malignancy in multiple primary tumors should be higher than a like group of single tumors

Peller presented material to support the contention that a cured cancer secured protection against the development of another cancer This was disputed by Warren and Gates who showed the carcinoma attack rate of organs other than skin to be twice that of the expected rate Warren and Ehrenreich calculated that multiple primary malignant tumors occurred 11 times more frequently than chance would allow Brunschwig[18] felt that the body must have some degree of resistance to the growth and invasion of cancer cells To support his argument he discussed various animal studies, as well as the documented cases of spontaneous regression He agreed that any immunological mechanism is relatively feeble in most people Bashford[19] showed that animals in which tumors had developed and then been absorbed were highly refractory to subsequent inoculations The same was true following radium exposure Fried stated that multiple primary malignant tumors were rare because of an immunity which developed Green and Strauss[20] found that in animals there was no increased tendency to multiple primary tumors in affected animals over unaffected animals Boyd[21] also maintained that one cancer afforded immunity against the development of a second Wallace stated that of 20 patients with a primary malignancy one would later develop another primary malignant tumor Epstein wrote that the incidence of multiple primary tumors was high when at least one of them occurred in the oral cavity Watson admitted that some patients demonstrate a marked predisposition for multiple cancers of the same organ He felt this indicated a response by different parts of similar tissue to the same stimulus In experiments with animal inoculation, Bashford, et al , found that the protection following absorption of tumor material from the same species as the protected animal was greatest against subsequent inoculation of the tumor absorbed and was less against other tumors of similar histogenesis They found that mice which were highly protected by inoculation subsequently developed spontaneous tumors of their own

The areas most frequently involved with multiple primary malignant tumors are the colon, pharynx, stomach, and uterus Barrett noted a tendency for multiplicity of squamous cell cancer in the buccal mucosa Fried found that cancer of the colon is the tumor most frequently associated with another primary tumor Eisenstadt[22] found that tumors of the gastrointestinal and gentiourinary tracts are the most frequently associated Warren and Ehrenreich found in 194 multiple primary cancers that 95 involved the colon, 44 the skin, and 34 the pharynx (including the buccal cavity)

Since the cases to be reported in this paper are mostly of the oral cavity let us examine some of the literature dealing with this area Wynder[23] discussed the not infrequent occurrence of primary carcinoma in the larynx, lung, and oral cavity Epstein felt that because of the small area of the oral cavity, serial sectioning of the intervening tissues might be required to confirm the clinical impression of distinct origin Moertel and Foss[24] used two criteria for multicentric origin of oral cancer

1 The tumors are stimultaneous if they are clearly separated by intervals of normal mucosa

2 They are multiple and nonsimultaneous if the most recent lesion is clearly separated from the site of excision of the initial lesion

Lund[25] discussed the occurrence of second primary tumors of the buccal mucosa He considered a cancer developing in mucous membrane distant to the first primary to be a new lesion rather than a metastasis He found the incidence of a second primary tumor in the oral cavity to be 15 times greater than that expected by chance alone In a study of mixed tumors of the oral cavity, New and Childrey[26] found a tendency toward multiplicity

We have encountered 14 cases of multiple primary malig-

TABLE III

Multiple Foci in Oral Cavity

First Cancer			Second Cancer			Third Cancer	
Type	Location	Interval	Type	Location	Interval	Type	Location
Squamous	Gingiva, upper anterior	3 yrs	Squamous	Gingiva, lower posterior	3 yrs	Squamous	Antrum, right
Squamous	Gingiva, left lower	8 mos	Squamous	Jaw, right lower			
Squamous	Larynx	2 yrs	Squamous	Tongue, anterior			
Squamous	Floor of Mouth, left	13 mos	Squamous	Jaw, right			
Squamous	Tonsil, left	21 mos	Squamous	Tonsil, right			
Squamous	Tonsil, left	1 mos	Squamous	Tonsil, right	14 mos	Squamous	Floor of Mouth, left, anterior
Squamous	Larynx and Esophagogastric Junction—simultaneous						
Squamous	Floor of Mouth, anterior and Palate—simultaneous						
Squamous	Tonsil, right	3¼ yrs	Squamous	Floor of Mouth, left, posterior			
Squamous	Floor of Mouth, anterior	10 mos	Squamous	Tonsillar pillar, right	16 mos	Squamous	Floor of Mouth, right
Squamous	Tonsillar Pillar, left	1 mos	Squamous	Tonsillar pillar, right			
Squamous	Palate, left	18 mos	Squamous	Palate, right			
Squamous	Floor of Mouth, right, anterior	13 mos	Squamous	Tonsillar pillar, right			

nant tumors of the oral cavity plus 12 cases of carcinoma of the oral cavity associated with tumors elsewhere in the body. In a study of 80 patients with intraoral cancer, Slaughter found that 18 per cent had grossly apparent multicentric lesions. Byars and Anderson[28] reviewed 166 cases of cancer of the oral cavity and found that 12 per cent had multiple oral cancer. Wilkins and Vogler[29] found that in carcinoma of the gingiva, 21 per cent of 81 cases had multiple lesions of the oral cavity.

TABLE IV

Cancer of Oral Cavity Associated with Cancer Elsewhere

First Cancer		Second Cancer	
Location	Type	Location	Type
Lip	Squamous	Thyroid	Follicular Carcinoma
Tongue	Squamous	Rectum	Adenocarcinoma
Gingiva	Squamous	Uterus	Adenocarcinoma
Tongue	Squamous	Stomach	Leiomyosarcoma
Floor of Mouth	Squamous	Lung	Oat Cell Carcinoma
Tongue	Squamous	Parotid	Mucoepidermoid Carcinoma
Floor of Mouth	Squamous	Lung	Adenocarcinoma
Tonsil	Squamous	Skin	Squamous Cell Carcinoma
Tongue	Squamous	Parotid	Warthin's Tumor
Larynx	Squamous	Prostate	Adenocarcinoma
Uterus	Adenocarcinoma	Right Tonsil	Squamous
Prostate	Adenocarcinoma	Lip	Squamous

Table III analyzes the 14 cases of multiple primary malignant tumors of the oral cavity that we have collected. The average interval between tumors in our series was 17.3 months. The family history did not indicate a hereditary tendency in any of our patients. We noted no tendency to increased undifferentiation between the first tumor and those developing later. All of our cases of multicentric tumors of the oral cavity occurred in patients who had a long history of heavy alcohol intake. All of them continued to drink heavily following the treatment of their first tumor. Moertel, Wynder[30] and many others have commented on the relationship between smoking and oral cancer as well as alcohol intake and oral cancer. The evidence is convincing enough to behoove the physician to advise patients who develop oral cancer to stop using both tobacco and alcohol

Table IV lists our cases of cancer of the oral cavity associated with cancer elsewhere in the body

SUMMARY

1 Billroth first described multiple primary malignant tumors in 1879 His postulates have been found too strict by later investigators

2 The incidence of multiple primary tumors is in the range of 4 6 per cent

3 Multiple primary malignant tumors are more common in males

4 It is apparent that multiple primary malignant tumors occur more frequently than chance allows

5 Some observations on pathogenesis have been surveyed

6 The question of immunity following the development of cancer is discussed

7 There is a tendency for multiplicity of squamous cell carcinoma in the oral cavity

8 The incidence of multiple primary malignant tumors is higher when at least one occurs in the oral cavity

9 Fourteen cases of multicentric carcinomas of the oral cavity and 12 cases of cancer of the oral cavity associated with cancer elsewhere have been reported

10 The association of tobacco and alcohol with cancer of the oral cavity has been noted

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Tongue	Squamous	Parotid	Warthin's Tumor
Larynx	Squamous	Prostate	Adenocarcinoma
Uterus	Adenocarcinoma	Right Tonsil	Squamous
Prostate	Adenocarcinoma	Lip	Squamous

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THE WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

The West Virginia Academy of Ophthalmology and Otolaryngology will hold its annual meeting at the Greenbrier Hotel, White Sulphur Springs, W Va, on April 6-8, 1961

The guest speakers on Ophthalmology are Dr Irving H Leopold, Philadelphia, Pa, and Dr Harvey E Thorpe, Pittsburgh, Pa, for Otorhinolaryngology Dr John J Shea, Memphis, Tenn, and Dr F Johnson Putney, Philadelphia, Pa

In addition to the scientific program, arrangements have been made with Mr Philip Salvatori of Obrig Laboratories to discuss and show techniques of contact lens fitting

For additional information please contact the secretary, Dr Worthy W McKinney, 109 East Main St, Beckley, W Va

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accomplished in a minimum of time An additional advantage is the fact that S-shaped external auditory meatus whose anterior wall obstructs the anterior and antero-inferior portion of the tympanic membrane and its annular region, can easily be drilled away with the same instrument The action of the burr also provides proper hemostasis in case any bone work should be necessary

Squamous epithelium growing around the edge of the perforation to the mucosal surface of the tympanic membrane can also be removed by the same technique with a wide margin of safety

The manubrium of the malleus is frequently wrapped in squamous epithelium in many cases of a long-standing defect of the pars tensa Technically, it is no particular problem to remove this tissue from the handle of the malleus by the sanding technique, however, because of the considerable vibratory action of the burr, the possibility of acoustic trauma to the inner ear structures should be kept in mind Further investigations concerning this subject are being carried out From my own experience, I do not believe that the short irritation of the inner ear fluids, resulting from the action of the burr on the soft tissue structures of the tympanic membrane, is any more harmful than the conventional manipulation of these structures

SUMMARY

A new method of denuding the tympanic membrane and adjacent external auditory meatus from its squamous epithelial layers by use of the "sanding technique" with a diamond burr is presented

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A NEW TECHNIQUE FOR MYRINGOPLASTIC PROCEDURES

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The two main problems facing the surgeon during myringoplastic procedures are:

1. The exact preparation of a bed for the free graft by denuding the tympanic membrane and part of the adjacent external auditory meatal wall of its squamous epithelial layers.

2. The proper positioning of the skin graft.

If both these conditions are met, then, in my opinion, most myringoplastic procedures yield quite satisfactory results, providing the usual medical and surgical principles concerning indication for surgery, aseptic technique, and proper follow-up are strictly observed.

The denuding of the tympanic membrane and canal wall of squamous epithelium is the most time-consuming part of the operation, if done in the conventional manner. It becomes a rather short and comparatively easy procedure if the "sanding method" is employed. This is best done with a globe-shaped fine diamond burr. These burrs have been in common use among dentists for a long time. In dentistry, of course, their main use is restricted to working on extremely hard material, namely, the enamel; however, when fitted to an angulated hand-piece, they are well suited to this procedure.

The mechanical principle involved resembles that used by the plastic surgeons in their "face-sanding" procedure. Using the diamond burr under the operating microscope, employing high speed and slight pressure, should be no problem for the surgeon familiar with temporal bone surgery. There is no tearing of the soft tissues if the instrument is used with proper care and the preparation of the bed for the graft is

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there is no feasible way by which the patient's respiratory efforts can be assisted Work by Elam and Brown[3] has indicated that ether as an anesthetic agent causes a significant increase in the production of endogenous carbon dioxide Concomitantly, the same investigators concluded that alveolar carbon dioxide accumulation was consistently found during surgical planes of ether anesthesia, unless the patient's ventilation was vigorously assisted In view of such findings, the authors undertook a clinical study of blood pH and blood gas values to determine the extent of respiratory dysfunction that might result from the use of ether as the principal anesthetic agent for tonsil surgery without the benefit of assisted respirations

METHOD

A total of 20 patients was used in this study, ranging in age from four to 11 years A combination of pentobarbital and scopolamine was given intramuscularly one hour preoperatively All of the anesthesias were given by two physician-anesthesiologists, each of whom managed ten cases The techniques varied only with respect to the induction phase One of the anesthesiologists accomplished induction with open-drop Vinethene® and ether with an oxygen flow of 500 cc per minute beneath the mask The other anesthesiologist carried out induction by means of a cyclopropane and ether sequence administered by a to-and-fro non-absorption system (reservoir bag and mask) with a total gas flow of one liter per minute With this technique respirations were assisted throughout induction During the initial one to two minutes after the change-over to the insufflation technique, varying flows (200-400 cc/min) of cyclopropane were added to the mixture of oxygen and ether

Maintenance of anesthesia in both groups of patients was by means of oxygen and ether insufflation through an appropriate size Davis-Crow mouth gag A Foregger Copper Kettle® was utilized to vaporize the ether Total flow of oxygen during maintenance was between 1 4 and 2 liters For the purpose of the study all the patients were kept in clinical Stage

ETHER ANESTHESIA FOR TONSILLECTOMY STUDIES IN RESPIRATORY ADEQUACY*

THOMAS D GRAFF, M D ,
LEROY C HARRIS, M D ,
THOMAS D MICHAEL, M D ,
OTTO C PHILLIPS, M D ,
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and
EDWARD T FOX, M D

With the impressive technical advancements that have been made in the field of thoracic and cardiovascular surgery during the past decade, there has been an increased demand for greater knowledge of biochemical measurements that might reflect aberrant respiratory physiology When deviations in respiratory function have been demonstrated by such means, causative factors proposed have included the position of the patient during the operation, the surgical procedure itself, and the anesthetic technique or agent[1] While the surgical and anesthetic challenge for tonsil and adenoid surgery may not approach the magnitude of that for intrathoracic procedures, the importance and gravity of tonsil surgery, if judged by the number of cases done as well as by the incidence of mortality,[2] leads one to suppose that these patients deserve closer scrutiny than they now receive in order to detect possible physiologic trespass during anesthesia

The purpose of this study was to determine the degree to which respiratory chemistries deviate during the clinical conditions imposed by an ether insufflation anesthesia technique Insufflation of oxygen and ether by means of a suitable mouth gag has been found, clinically, to be a highly satisfactory means by which to maintain anesthesia in children undergoing tonsil and adenoid surgery With this technique, however,

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there is no feasible way by which the patient's respiratory efforts can be assisted. Work by Elam and Brown[3] has indicated that ether as an anesthetic agent causes a significant increase in the production of endogenous carbon dioxide. Concomitantly, the same investigators concluded that alveolar carbon dioxide accumulation was consistently found during surgical planes of ether anesthesia, unless the patient's ventilation was vigorously assisted. In view of such findings, the authors undertook a clinical study of blood pH and blood gas values to determine the extent of respiratory dysfunction that might result from the use of ether as the principal anesthetic agent for tonsil surgery without the benefit of assisted respirations.

METHOD

A total of 20 patients was used in this study, ranging in age from four to 11 years. A combination of pentobarbital and scopolamine was given intramuscularly one hour preoperatively. All of the anesthesias were given by two physician-anesthesiologists, each of whom managed ten cases. The techniques varied only with respect to the induction phase. One of the anesthesiologists accomplished induction with open-drop Vinethene® and ether with an oxygen flow of 500 cc. per minute beneath the mask. The other anesthesiologist carried out induction by means of a cyclopropane and ether sequence administered by a to-and-fro non-absorption system (reservoir bag and mask) with a total gas flow of one liter per minute. With this technique respirations were assisted throughout induction. During the initial one to two minutes after the change-over to the insufflation technique, varying flows (200-400 cc./min.) of cyclopropane were added to the mixture of oxygen and ether.

Maintenance of anesthesia in both groups of patients was by means of oxygen and ether insufflation through an appropriate size Davis-Crowe mouth gag. A Foregger Copper Kettle® was utilized to vaporize the ether. Total flow of oxygen during maintenance was between 1.4 and 2 liters. For the purpose of the study all the patients were kept in clinical Stage

III, plane 2 to 3 surgical anesthesia A 10-degree Trendelenburg position was used during surgery

Venous blood, arterialized according to the technique of Goldschmidt and Light,[4] was drawn at the following times *1* when a state of unconsciousness had been accomplished, *2* just prior to the change-over to insufflation, *3* on beginning the dissection of the second tonsil, *4* at the termination of the surgical procedure

Chemical determinations performed on the arterialized venous blood included oxygen saturation (with a cuvette oximeter), carbon dioxide content (by a modified Van Slyke-Neil manometric technique),[5] and pH (glass electrode pH meter) Calibration of the cuvette was carried out as decribed by the manufacturer[6] utilizing bloods of known oxygen saturation Such values were calculated from oxygen content (Van Slyke-Neil analysis) and hemoglobin determinations The accuracy of the oximeter readings was checked at intervals during the experimental period and was found to be within ±2 per cent of calculated values Measurements of pH were read only after blood samples and buffer solutions had been brought to constant temperatures and suitable adjustments had been made with the manual temperature compensation control of the pH meter

Continuous electrocardiographic tracings were recorded on an oscilloscope screen, permanent tracings were made when indicated Additional observations were made as to the effect of the preanesthetic medication, the general course of anesthesia, amount of blood loss during surgery, and the immediate postoperative course in the recovery room

RESULTS

General Clinical Course

All of the children were awake on arrival at the operating room During the preliminary preparations for the study, they expressed no undue concern or anxiety, and all were classified as being fully cooperative Table I gives the patients' ages and body weights as well as their hemoglobin values and the duration of anesthesia Airway problems during the course of anesthesia were limited to an occasional mild laryngeal stridor early during the surgical procedure while traction was being applied to one or the other tonsils Transient breath-holding occurred not uncommonly immediately following the change-over from the to-and-fro technique

TABLE I

Preoperative Data and Duration of Anesthesia

To and Fro

Patient	Age Yrs	Wt lbs	Duration Anesth Min	HGB Gms
1 S S	6	42	20	11 8
2 P D	5	34	30	11 8
3 F H	5	45	25	13 0
4 D S	7	55	25	13 5
5 D B	6	40	23	12 5
6 M L	6	45	35	13 0
7 A P	11	64	30	13 2
8 P S	6	70	28	10 0
9 L W	5	40	32	11 0
10 W H	5	54	25	12 8
Average	6 2	48 9	27 3	12 3

Open Drop

Patient	Age Yrs	Wt lbs	Duration Anesth Min	HGB Gms
11 C P	7	90	50	11 5
12 C H	6	46	40	13 0
13 S S	6	41	35	12 8
14 C B	6	44	35	13 0
15 E S	9	80	30	12 0
16 F S	7	50	25	10 8
17 A.S	5	44	40	11 8
18 B H	4	41	40	11 8
19 J F	5	46	33	12 6
20 M P	5	49	32	13 8
Average	6 0	53 1	36 0	12 3

to insufflation, but in each case this was remedied by adding cyclopropane to the oxygen-ether mixture for two to three minutes. Blood loss was considered to have been minimal to average in all cases except one (C P), in whom difficulty with oozing was not only marked during the procedure, but also continued into the postoperative period at which time the child was transfused with one unit of blood

Blood Chemistries During the Anesthesia

We note from Table II that oxygen saturations were in excess of 93 per cent during maintenance of anesthesia (insufflation) with one exception, (C. P.), whose saturation value fell to 76 per cent at the time of the commencement of dissection of the second tonsil. As would be expected, the to-and-fro assisted respiration technique of induction resulted in better oxygenation during this phase of anesthesia than did the open-drop technique. With respect to carbon dioxide contents (see Table III), blood samples drawn during the open-drop to insufflation sequence gave no indication of CO_2 retention during maintenance anesthesia. The initial average

TABLE II

Arterialized Blood Oxygen Saturation (Per Cent)

To and Fro

	Patient	a	b	c	d
1	S S	98.5	99.5	99.5	99.5
2	P D	100.0	100.0	100.0	99.5
3	F H	95.5	98.5	100.0	99.5
4	D S	93.0	98.5	98.5	98.0
5	D B	94.0	99.5	99.5	99.0
6	M L	96.5	98.0	94.5	97.0
7	A P	98.0	100.0	99.5	98.0
8	P S	95.5	100.0	99.5	99.0
9	L W	96.5	98.5	99.0	98.5
10	W H	99.0	99.0	99.5	99.5
Average		96.7	99.2	99.0	98.4

Open Drop

	Patient	a	b	c	d
11	C P	88.0	80.0	76.0	91.5
12	C H	96.5	98.5	98.5	98.5
13	S S	81.5	98.5	99.5	98.5
14	C B	89.5	88.0	93.5	96.5
15	E S	91.0	98.0	97.5	98.5
16	F S	94.5	98.5	97.0	96.5
17	A S	95.0	90.0	97.5	97.5
18	B H	93.0	96.5	96.5	98.5
19	J F	93.5	96.0	96.0	95.0
20	M P	83.0	99.5	97.0	—
Average		90.6	94.4	95.0	96.8

a.—Induction
b.—Changing from to and fro to insufflation
c.—Beginning dissection of second tonsil
d.—Termination of surgical procedure

value of 46.9 Vols. per cent undoubtedly represents the effect of breath-holding just prior to loss of consciousness. The closed to-and-fro induction technique brought about an average increase in the carbon dioxide content of 5 Vols. per cent over the initial reading. This accumulated carbon dioxide was completely eliminated by the time the first tonsil had been resected. pH determinations (see Table IV) showed no remarkable variations in acid-base balance, except for a mild respiratory acidosis during induction with the to-and-fro technique. With the change-over to insufflation, the average pH returned to a presumably normal value.

TABLE III

Arterialized Blood CO_2 Content (Vol %)

To and Fro

	Patient	a	b	c	d
1	S. S.	41.3	46.1	40.7	40.4
2	P. D.	36.7	46.8	39.5	41.5
3	F. H.	43.2	47.6	40.0	37.6
4	D. S.	44.3	49.1	43.1	42.6
5	D. B.	45.8	49.0	46.1	43.4
6	M. L.	35.6	43.2	40.0	34.0
7	A. P.	47.2	50.5	44.4	44.4
8	P. S.	41.0	44.0	42.0	39.5
9	L. W.	39.3	45.2	38.8	39.0
10	W. H.	41.4	44.5	41.2	39.5
Average		41.6	46.6	41.6	40.2

Open Drop

	Patient	a	b	c	d
11	C. P.	44.6	40.3	38.7	39.6
12	C. H.	47.5	42.2	39.2	39.8
13	S. S.	49.7	42.1	41.7	41.7
14	C. B.	48.4	48.6	45.7	43.5
15	E. S.	48.4	42.4	42.1	43.1
16	F. S.	52.3	44.1	42.8	42.0
17	A. S.	47.5	44.6	40.3	40.6
18	B. H.	42.8	41.3	41.4	37.5
19	J. F.	39.3	34.2	35.0	39.3
20	M. P.	48.6	42.3	41.7	—
Average		46.9	42.2	40.9	40.8

a—Induction
b—Changing from to and fro to insufflation
c—Beginning dissection of second tonsil
d—Termination of surgical procedure

TABLE IV

Arterialized Blood pH

To and Fro

Patient		a	b	c	d
1	S S	7 41	7 38	7 43	7 44
2	P D	7 40	7 37	7 41	7 38
3	F H	7 46	7 42	7 42	—
4	D S	7 35	7 30	7 38	7 41
5	D B	7 36	7 34	7 40	7 44
6	M L	7 38	7 35	7 38	7 40
7	A P	7 38	7 34	7 42	7 44
8	P S	7 30	7 38	7 41	7 49
9	L W	7 40	7 35	7 48	7 48
10	W H	7 41	7 37	7 45	7 47
Average		7 39	7 35	7 42	7 44

Open Drop

Patient		a	b	c	d
11	C P	7 38	7 38	7 40	7 40
12	C H	7 36	7 38	7 38	7 38
13	S S	7 38	7 40	7 41	7 40
14	C B	7 43	7 42	7 45	7 48
15	E S	7 42	7 46	—	—
16	F S	7 40	7 48	7 50	7 50
17	A S	7 36	7 41	7 43	7 40
18	B H	7 42	7 35	7 33	7 38
19	J F	7 40	7 45	7 42	7 40
20	M P	7 45	7 49	7 47	—
Average		7 40	7 42	7 42	7 42

a —Induction
b —Changing from to and fro to insufflation
c —Beginning dissection of second tonsil
d —Termination of surgical procedure

Electrocardiographic Changes

Aside from minimal changes in cardiac rate, 18 of the children showed no variation in their electrocardiographic tracings The remaining two patients developed conduction defects during the latter part of the maintenance phase of anesthesia This consisted of a nodal rhythm in one child (J F), and a first-degree heart block in the other (C B) Both of these abnormal patterns returned to normal sinus rhythms before the termination of the procedures

DISCUSSION.

Whitteridge[7] voiced the classical description of the influence of ether on respiratory behavior in stating that the respiratory center was stimulated reflexly by ether until the deeper planes of surgical anesthesia were reached, at which time there was a reduction in both tidal volume and minute volume In a recently edited textbook of pharmacology,[8] the sectional author states that body metabolism is reduced by ether, and, in the absence of anoxia or carbon dioxide retention, there is no upset in acid-base balance

In an excellent review of pediatric anesthesia, Stephen[9] defined good anesthesia as that which caused "minimal disturbance of the patient's basic physiologic functions" Included in this review was a critical analysis of the open-drop technique in which Stephen pointed out that the technique was essentially limited to ether, which produces metabolic acidosis when used in relaxing concentrations Watrous[10] stressed the importance of actively supporting respirations during all types of inhalational anesthesia so as to combat the tendency of such agents to cause hypoxia and respiratory acidosis Beecher[1] has presented data to show that patients receiving ether for thoracic surgery develop a combined respiratory and metabolic acidosis unless the patient's ventilatory efforts are vigorously assisted He called attention to the fact that the oxygen tensions were generally satisfactory at the time when the carbon dioxide tensions were dangerously high Similar studies[11 12] substantiated this noteworthy clinical report and provided further evidence as to the possible disturbance in acid-base balance resulting from ether anesthesia Bunker[13] found that most infants and about 50 per cent of children between the ages of one to 12 years develop moderate degrees of metabolic acidosis during ether anesthesia

In contrast to patients undergoing thoracic procedures, Beecher[14] was able to show that adults anesthetized with ether, but kept in a supine position, failed to develop metabolic acidosis, furthermore, they have no tendency to accumulate alveolar carbon dioxide or build up fixed acids In extrathoracic procedures under deep ether anesthesia, Gabbard[12] noted that alveolar ventilation was sufficient to maintain normal arterial carbon dioxide tensions In several instances,

TABLE IV

Arterialized Blood pH

To and Fro

Patient		a	b	c	d
1	S S	7 41	7 38	7 43	7 44
2	P D	7 40	7 37	7 41	7 38
3	F H	7 46	7 42	7 42	—
4	D S	7 35	7 30	7 38	7 41
5	D B	7 36	7 34	7 40	7 44
6	M L	7 38	7 35	7 38	7 40
7	A P	7 38	7 34	7 42	7 44
8	P S	7 30	7 38	7 41	7 49
9	L W	7 40	7 35	7 48	7 48
10	W H	7 41	7 37	7 45	7 47
Average		7 39	7 35	7 42	7 44

Open Drop

Patient		a	b	c	d
11	C P	7 38	7 38	7 40	7 40
12	C H	7 36	7 38	7 38	7 38
13	S S	7 38	7 40	7 41	7 40
14	C B	7 43	7 42	7 45	7 48
15	E S	7 42	7 46	—	—
16	F S	7 40	7 48	7 50	7 50
17	A S	7 36	7 41	7 43	7 40
18	B H	7 42	7 35	7 33	7 38
19	J F	7 40	7 45	7 42	7 40
20	M P	7 45	7 49	7 47	—
Average		7 40	7 42	7 42	7 42

a —Induction
b —Changing from to and fro to insufflation
c —Beginning dissection of second tonsil
d —Termination of surgical procedure

Electrocardiographic Changes

Aside from minimal changes in cardiac rate, 18 of the children showed no variation in their electrocardiographic tracings The remaining two patients developed conduction defects during the latter part of the maintenance phase of anesthesia This consisted of a nodal rhythm in one child (J F), and a first-degree heart block in the other (C B) Both of these abnormal patterns returned to normal sinus rhythms before the termination of the procedures

DISCUSSION

Whitteridge[7] voiced the classical description of the influence of carbon dioxide during the induction phase (Lurie, et al[15]), as well as during the period immediately following the change-over to an open insufflation system when the accumulated carbon dioxide might be rapidly eliminated (Brown and Miller[16]) Such fears failed to materialize, as all the children in the to-and-fro induction group showed normal electrocardiographic tracings throughout their courses of induction and maintenance Reference to Table II shows that these patients were extremely well oxygenated, a factor which, according to Jacoby,[17] is most important in preventing abnormal cardiac rhythms

It is important to point out that all of these cases were managed in conjunction with cooperative and proficient otolaryngologists, highly cognizant of airway problems We certainly do not mean to infer that patients undergoing tonsillectomies with this method never develop laryngeal stridor, hypoxic episodes, or cardiac arrhythmias We do believe, however, that the information presented here shows that a properly conducted insufflation technique can fulfill Stephen's criteria of good anesthesia by causing "minimal disturbance of the patient's basic physiologic function", and thus provide safe and reliable anesthesia for children undergoing such elective surgery, nevertheless, there should be no question on the part of the anesthesiologist or the surgeon that an alternative endotracheal technique and a means of aiding of supplanting the patient's exchange be immediately available in the event respirations become depressed, obstructed or otherwise ineffective

SUMMARY

A study was conducted to determine the presence or absence of aberrant respiratory function during ether-insufflation anesthesia for tonsil and adenoid surgery Analysis of serial blood chemistries showed that oxygen saturation in all but one patient was in excess of 93 per cent during maintenance anesthesia, while during the same phase carbon dioxide and pH values showed no remarkable variations

Electrocardiographic monitoring failed to show any ab-the ventilation was actually increased to the point of causing respiratory alkalosis

In the present study, there are numerous clinical factors which might possibly lead to findings of respiratory inadequacy These are

A Depressed minute volume due to deep surgical planes of anesthesia

B Unsupported respirations

C Trendelenburg position, in which diaphragmatic efficiency is impaired

D The age of the patients

E Surgical manipulation, retraction and packing in the immediate vicinity of the glottis

The composition and analysis of blood chemistries in our series of patients failed to show that the above factors materially affected respiratory function With the exception of one patient (C P) in the open-drop induction group, all the children showed oxygen saturations of 93 per cent or better throughout the maintenance period (after change-over to insufflation) We have no explanation for the exceptionally low oxygen readings of this one patient who demonstrated no unusual airway problem Of some significance perhaps is the fact that this child had a chronic upper respiratory-tract infection and a rectal temperature of 100° F During the procedure, the blood was considered to be dark by both the anesthesiologist and the surgeon This same patient had excessive oozing postoperatively

During the maintenance phase of anesthesia, the carbon dioxide and pH readings showed no indication of either respiratory or metabolic acidosis Utilizing the to-and-fro nonabsorption technique there was the expected carbon dioxide retention which was reflected in a small drop in the average arterial pH The greatest concern over such a retention of carbon dioxide was the possibility that abnormal ventricular rhythms might occur in association with the high blood levels

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GILL MEMORIAL EYE, EAR AND THROAT HOSPITAL

34TH ANNUAL SPRING CONGRESS

The Gill Memorial Eye, Ear and Throat Hospital will hold its Thirty-Fourth Annual Spring Congress in Ophthalmology and Otolaryngology and Allied Specialties, April 3-8, 1961 There will be 20 guest speakers and 50 lectures normal rhythms during induction or maintenance anesthesia Minor conduction changes were noted in two cases late in the course of anesthesia

Such data have demonstrated that in children with an age range of from four to 11 years satisfactory oxygenation and acid-base balance may be maintained in deep surgical phases of ether anesthesia when administered by an insufflation technique

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tween supraglottic lesions and lesions involving the cartilaginous box of the larynx[3] These deserve particular separation in teaching, so that an improvement in practice may occur

Eve[4] has pointed out how obstruction to the airway may occur when the epiglottis is inadvertently forced into the airway in such a manner that it cannot be displaced He examined a victim of electrocution The epiglottis was firmly wedged in the superior laryngeal aperture by the muscular forces generated by the current Eve felt that if the electrical current had not killed the patient, he would have been asphyxiated He further suggests that certain sudden deaths observed during bombing attacks may be due to the force of the shock wave of the explosion jamming the epiglottis into the superior laryngeal aperture at a time when the patient was in the phase of expiration Under these circumstances, having insufficient air in the lung to force the epiglottis out of the superior laryngeal aperture, the patient is thereby asphyxiated He suggested this as an explanation for the observed fact that two persons in a blast area may be standing side by side uninjured by any flying particles One lives, and the other dies In Eve's opinion, the one who dies may have been at the end of an expiratory cycle when the shock wave struck and was, therefore, unable to force the occluding epiglottis out of the superior laryngeal aperture

A similar situation has been described by Miller[6] with regard to acute epiglottitis in children One of the characteristic features of the dyspnea in acute epiglottitis is its variability This is due to the fact that swollen and edematous tissues of the epiglottis and superior laryngeal aperture are not fixed by the laryngeal cartilages These tissues produce obstruction which varies with the degree of swelling, the position of the head, the contraction of the pharyngeal musculature, and the force of respiration With a swollen mass of tissue overlying the superior laryngeal aperture, it is easy to understand how a situation such as Eve has suggested could occur The patient suffers a bout of paroxysmal coughing, exhausting air in the lung, and then attempts to inspire quickly, pulling the swollen tissues of the supraglottic area into

CURRENT UTILIZATION OF TRACHEOTOMY AS A THERAPEUTIC MEASURE[*][†]

A Review of the Literature and an Analysis of 526 Cases

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Tracheotomy has long been a fascinating operation in medical annals. Caelius Aurelianus[1] records the performance of a tracheotomy in Rome in the First Century, B.C. It has been referred to by Galen and was certainly performed in 1546 by Antonio Musa Brasavola.[2] The word "tracheotomy" appeared in the works of Thomas Fienus in 1649. The operation at that time was done for the relief of high obstruction in the airway. For this purpose, tracheotomy came to be a clearly indicated procedure and has been accepted by physicians to this day. In recent years, a changing concept of the function of tracheotomy has developed which has extended the usefulness of tracheotomy and changed many of the basic ideas which previously surrounded this operation. It is the purpose of this paper to examine the present ideas with relation to current teaching, as well as current practice in utilizing the procedure and to consider possible avenues whereby future progress may be made.

OBSTRUCTION TO THE AIRWAY AS AN INDICATION FOR TRACHEOTOMY

Although the classical indication for tracheotomy has been high obstruction in the airway, it is noteworthy that today many authors tend to create a single category without defining the variations in high obstruction which drastically alter one's clinical judgment with regard to management. In high respiratory obstruction, a distinct difference exists be-

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contribution of the otolaryngologist. As early as 1911, Chevalier Jackson[8] called attention to the fact that a patient may literally drown in his own secretion, but it remained for Lee, Tucker, and Clerf[9] to demonstrate by bronchoscopy that atelectasis was caused by the mechanical obstruction of bronchi by secretion and that removal of the obstruction restored pulmonary ventilation. This concept was also aided by the contributions of Coryllos.[10] The acceptance of this idea by the profession generally was slow, but in hospitals with well established bronchoscopic services, postoperative atelectasis was frequently managed by bronchoscopic aspiration. Where the conditions that produced the accumulation of secretion were non-recurring, bronchoscopy proved to be the ideal method of treatment and remains so today. The success of bronchoscopy was less satisfactory where failure of the expulsive mechanism of the lung permitted the re-accumulation of secretion. The work of Galloway[11 12 13 14 15] demonstrated the importance of airway obstruction by secretion in poliomyelitis, and the usefulness of tracheotomy in overcoming this constantly recurring obstruction. The accuracy of his observations was rapidly attested by numerous observers,[16 17 18 19 20 21 22 23 24] so that tracheotomy in bulbar poliomyelitis became an accepted method in this country. In England, it was not until Lassen's[25] report of the Copenhagen epidemic of poliomyelitis in 1953 that the use of tracheotomy for secretory obstruction was widely accepted.[26]

Tracheotomy came to be applied to a great many different disease situations, including tetanus,[27 28 29] botulism,[30] barbiturate poisoning,[31] eclampsia,[32 33 34] leprosy,[35] hydrophobia,[29] and certain allergic emergencies.[36] In our own clinic we were able to demonstrate marked improvement in the management of neurosurgical patients as was reported by Taylor and Austin.[37] Tracheotomy has come to be accepted as an essential part of the management of patients with depression of the central nervous system related to neurosurgery,[38] acute head trauma,[39 40 41] and other causes of unconsciousness.

Kime[42] has recommended that the depth of unconsciousness be related to levels of anesthesia as a criterion for election of the glottis like a cork into a bottle The patient with insufficient air in the lung is unable to expel the edematous tissues from the airway and is instantly suffocated

The entire supraglottic airway is subject in greater or less degree to this same sort of variable obstruction This type of obstruction should be recognized as a clinical entity distinctly different from obstruction in the region of the cartilaginous box, where the size of the airway tends to diminish by regular progression Glottic and subglottic obstruction involves this cartilaginous box and produces a clinical picture where a clear-cut relationship exists between the symptoms of obstruction and the need for tracheotomy It has been our practice to separate sharply those obstructive lesions arising in the supraglottic area where obstruction is variable and the moment of asphyxia is completely unpredictable For these, only three possible courses can be observed *1* relieve the obstruction, *2* literally stand by the patient prepared to do an immediate tracheotomy, or *3* tracheotomize in order to guarantee a free airway until such time as the supraglottic obstruction is relieved With regard to obstruction at the level of the glottis, it is possible to allow an obstruction to exist until that point is reached where the patient shows, in addition to stridor and retraction, the evidence of inadequate ventilation as demonstrated by pallor, restlessness, fatigue, sweating, and the inability to sleep

The concept of tracheotomy as a method of relieving high obstruction exclusively persisted to modern times As late as 1934, Figi could report 200 consecutive patients on whom 206 tracheotomies were performed In this group, 137 were done for carcinoma of the larynx, 25 for goiter, 14 for foreign body, and eight for laryngeal papillomata In none of the cases reported by Figi was there any mention of tracheotomy for the relief of bronchial obstruction

THE ROLE OF TRACHEOTOMY IN THE MANAGEMENT OF PULMONARY AND VENTILATORY PROBLEMS

The remarkable extension of tracheotomy to the treatment of pulmonary and ventilatory problems has been largely a contribution of the otolaryngologist. As early as 1911, Chevalier Jackson[8] called attention to the fact that a patient may literally drown in his own secretion, but it remained for Lee, Tucker, and Clerf[9] to demonstrate by bronchoscopy that atelectasis was caused by the mechanical obstruction of bronchi by secretion and that removal of the obstruction restored pulmonary ventilation. This concept was also aided by the contributions of Coryllos.[10] The acceptance of this idea by the profession generally was slow, but in hospitals with well established bronchoscopic services, postoperative atelectasis was frequently managed by bronchoscopic aspiration. Where the conditions that produced the accumulation of secretion were non-recurring, bronchoscopy proved to be the ideal method of treatment and remains so today. The success of bronchoscopy was less satisfactory where failure of the expulsive mechanism of the lung permitted the re-accumulation of secretion. The work of Galloway[11, 12, 13, 14, 15] demonstrated the importance of airway obstruction by secretion in poliomyelitis and the usefulness of tracheotomy in overcoming this constantly recurring obstruction. The accuracy of his observations was rapidly attested by numerous observers,[16, 17, 18, 19, 20, 21, 22, 23, 24] so that tracheotomy in bulbar poliomyelitis became an accepted method in this country. In England, it was not until Lassen's[25] report of the Copenhagen epidemic of poliomyelitis in 1953 that the use of tracheotomy for secretory obstruction was widely accepted.[26]

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Kime[42] has recommended that the depth of unconsciousness be related to levels of anesthesia as a criterion for election of tracheotomy A unique application of tracheotomy in neurosurgical problems is its use by Jackson and Carter[43] to prevent the building up of air pressures in the upper respiratory tract, in order to prevent the escape of air into the meninges from a skull fracture communicating with the upper respiratory tract

The acceptance of tracheotomy in general surgical services was somewhat slow, though everyone recognized the necessity of tracheotomy in thyroid surgery[44] where the indication was usually high obstructive dyspnea The demonstration by Haight[45] in 1938 that the introduction of an aspirating catheter into the tracheobronchial tree would reduce postoperative pulmonary complications by mechanically removing secretions and stimulating cough to fortify the expulsive mechanism of the lung showed the usefulness of this concept in general surgery [46,47,48]

In thoracic surgery, tracheotomy has become an important part of the armamentarium for a number of reasons [49,50,51] Where there has been a pulmonary resection and a marginal ventilating area remains, the accumulation of secretion is particularly intolerable to the patient A similar interference with respiration is encountered in crushing injuries of the chest

D'Abreu[52] in his recent text states that tracheotomy should be performed without hesitation as early as possible in crush injuries of the chest whenever paradoxical movements, ineffective respiration, dyspnea, cyanosis, and the inability of the patient to cough up bronchial secretion are obvious or are developing

The treatment of burns constitutes a peculiar indication for tracheotomy because a variety of elements enter into the indications Burns of the neck may be associated with edema of the burn area If the face is involved, the eyes and mouth may become closed by swelling, and when vomiting occurs, there is danger of aspiration An extension of this edema to the deeper tissues may interfere with respiration and cough A further factor is the possibility of injury by the inhalation of hot or irritating fumes Moncrief[53] feels that air can be cooled so rapidly that burns from this cause are rare, but notes that steam and irritant smokes can produce thermal or caustic injury to the tracheobronchial tree itself Under these circumstances, we have seen serious damage to the respiratory tract with a threat to the airway from edema, exudate, and slough of the mucosa with long term serious impairment to pulmonary function Of a thousand burn cases observed by Moncrief, 84 required tracheotomy

TRACHEOTOMY IN RESPIRATORY FAILURE

The current interest in pulmonary function has encouraged observers to relate respiratory problems to the total function of respiration In addition to the local problem occurring in the lung, the failure of sufficient central nervous system drive to maintain adequate ventilation and the effects of such loss must be considered[54 55 56 57 58] Among the earliest attempts to maintain ventilation under these circumstances, the Drinker Respirator was a definite advance It soon became apparent, however, that accumulation of secretion could nullify the usefulness of this type of treatment Tracheotomy, therefore, was employed to permit access to the airway The use of this cumbersome apparatus which competed with the tracheotomy tube for the neck area complicated the treatment but constituted a distinct advance

Another solution to the problem of loss of respiratory drive was the use of a cuffed oro-tracheal tube with forced ventilation through the mouth Such tubes are quite effective for brief periods of time, but if it is necessary to maintain ventilation in this way, several disadvantages appear They are not well tolerated by a conscious patient, they render aspiration of bronchial secretion difficult, they frequently lose lumen by encrustation of secretion within the tube itself, and they tend to increase the amount of dead space Respiratory failure is encountered not only in disease of the central nervous system and muscles, but also in emphysema and acute respiratory infections Westlake[59] studied the arterial blood gases of 30 patients with acute respiratory infection In six of these who had no previous chest difficulties, the arterial carbon dioxide pressure was normal, however, 18 of 24 with a history of chronic bronchitis and emphysema disclosed an arterial carbon dioxide tension five to 32 millimeters above that obtained on recovery. He considered these to be in acute respiratory failure. The accumulation of carbon dioxide in sufficient concentration produces a profound narcotic effect on respiratory drive, so that functionally there is, in addition to hypercapnia, increasing anoxemia and respiratory acidosis. Westlake enumerates the following symptoms: increased intracranial pressure, choked discs, headache, mental confusion, hallucinations, sweating, myoclonic jerking, increased cardiac output, pulmonary hypertension, and congestive heart failure. In patients such as these, the administration of oxygen reduces the anoxic drive and thereby diminishes ventilation with the production of a vicious circle consisting of increasing hypercapnia and increasing central nervous system narcosis. By increasing ventilation, the carbon dioxide can be removed, and respiratory function tends to be restored. In these circumstances, the use of a tracheotomy tube with a cuff surrounding it gives a sealed airway for ventilation which can be maintained satisfactorily by the use of one of the newer mechanical respirators, such as that described by Bennett. With this type of apparatus, a firm connection between the tracheotomy tube and respirator is necessary, but when an air-tight system has been achieved, the patency of the airway is easily maintained and ventilation can be satisfactorily carried on.

A tracheotomy tube devised by Dr. Thomas Cannard and the author has been found extremely satisfactory in situations where assistance to respiration is required. The fundamental ingredients of this tube are the inflatable cuff which surrounds the tube and the fitting on the inner cannula. When the tube lies in place, this cuff is in the trachea and is inflated so that a tight system is developed between the airway and the tracheotomy tube. The inner cannula of this tube is provided with a fitting that allows a tight connection to be made by the gas apparatus using the Foregger fitting. This permits the full diameter of the tracheal tube to be used for ventilation and diminishes resistance within the system It can be used for long periods of time, the cuff does not cause injury to the trachea, and it can be fitted easily to various types of ventilating apparatus which are now available (see Fig 1)

TRACHEOTOMY IN CHRONIC PULMONARY INSUFFICIENCY

Recent interest has been demonstrated in the treatment of chronic pulmonary disease, such as emphysema and the pulmonary aspects of cystic fibrosis by a technique called tracheal fenestration.[illegible] This is a surgical procedure that results in a firmly epithelialized mucocutaneous opening into the trachea provided with lip-like valves that can act as a closing mechanism. It is intentionally designed to obviate the wearing of a metal cannula The opening thereby produced in the trachea admits semi-rigid catheters for the purpose of aspi-

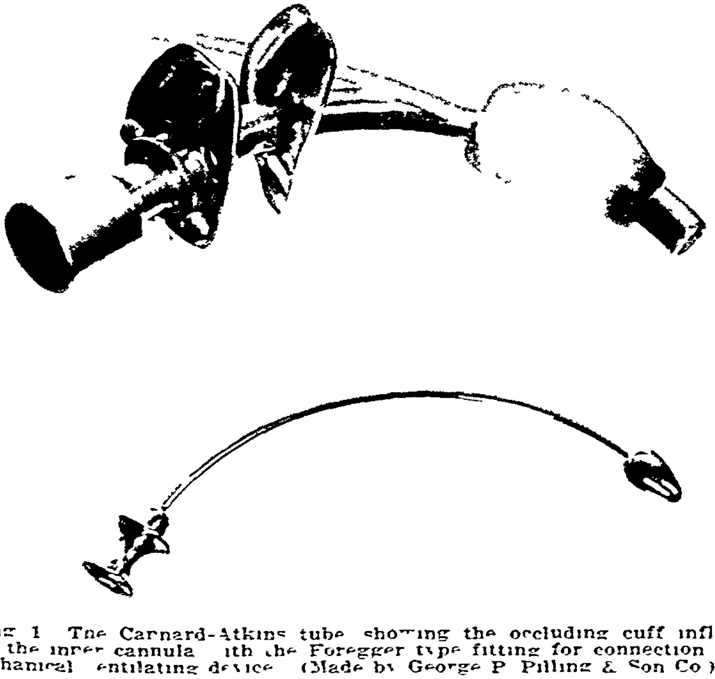

Fig 1 The Carnard-Atkins tube showing the occluding cuff inflated and the inner cannula with the Foregger type fitting for connection to a mechanical ventilating device (Made by George P Pilling & Son Co)

rating bronchial secretion from these patients with chronic pulmonary disease The advocates of the method contend that the critical factor in the disability of such disease is not the quantity of secretion but the reduced pulmonary reserve associated with a failure of the cough mechanism They claim the effects of aspiration extend beyond the immediate relief from discomfort and asphyxia Tracheal aspiration is often followed by prolonged improvement so that the frequency of aspiration becomes less and less, and exercise tolerance improves This would parallel the experience one sees in patients wearing conventional tracheotomy tubes, where the improvement is attributed to the elimination of the effects of infection which almost automatically follows an improvement in pulmonary drainage Following the fenestration operation, patients aspirate themselves and learn to control the flaps with digital pressure, so that cough and expectoration via the mouth can be carried out normally The proponents of this procedure warn against indiscriminate use and find it no short-cut therapy for profuse expectoration, cough, or pulmonary suppuration The technique described by Rockey involves an operative procedure of some four hours' duration with a temporary tracheotomy placed about the seventh tracheal cartilage Our personal experience with the method is insufficient to evaluate it The idea appears to have merit, though a simpler approach to the airway would seem possible and highly desirable A further trial of the method is justified

TECHNICAL CONDITIONS EFFECTING SUCCESS IN TRACHEOTOMY

Perhaps the most important single element in the successful application of the operation of tracheotomy is timing The tracheotomy which is done too late is generally recognized as having a severely limited value[64, 65, 66] This is true regardless of the indication for which the procedure is done For this reason, prophylactic tracheotomy has been employed more and more in those situations where experience indicates that a need for tracheotomy may be reasonably expected

Almost as important as timing, is planning The proper respect for tracheotomy as an operative procedure with inherent hazards is frequently overlooked, particularly by the inexperienced A failure to appreciate that the patent airway is the thing to be acquired rather than the tracheotomy has caused physicians to make injudicious moves in unpropitious circumstances Wherever possible, an airway should be obtained and maintained Davis, Kretchmer, and Bryce-Smith[67] encountered complications in 54 of 155 tracheotomies Of these 54, there were 20 major complications Fourteen of these, or 70 per cent, occurred in tracheotomies which were done as emergencies The acquiring of an airway is usually best accomplished by the introduction of a bronchoscope This not only gives an airway but also permits one to examine and sometimes correct the causes of obstruction An unrecognized foreign body or exudate may be removed with restoration of the airway, so that tracheotomy may be avoided If a bronchoscope is not available, the use of an oro-tracheal or naso-tracheal tube will permit one to carry out ventilation and aspirate secretion As a temporary expedient, the oral path to the airway is usually safer and faster Once an airway has been achieved, the patient should be taken to the operating room where the tracheotomy can be done without haste with the advantage of good light, good position, good assistants, and the safeguards for emergency treatment which are normally found in an operating room An attempt to do tracheotomy in a bed or respirator with poor light and poor assistants is not to the patient's best interest[68] Seldom is a patient so sick that he cannot be taken to the operating room in his bed

Of particular concern is the use of tracheotomy in small children[69-70] It has long been our practice to stabilize the trachea and guarantee an airway by the use of an oro-tracheal airway, usually a bronchoscope which is held by an assistant and withdrawn as the tracheotomy tube is introduced In the absence of a dependable assistant, the bronchoscope can be used as a diagnostic instrument and then removed A fairly rigid endotracheal tube is then introduced to maintain the airway and stabilize the trachea An anesthesiologist or nurse anesthetist can be depended on to handle such an airway expertly This avoids the many complications which are peculiar to tracheotomy in the very young[71]

A constantly present problem in the management of the tracheotomized patient is postoperative care[72,73,74,75,76] This is complicated by the constantly changing personnel which exists in modern hospitals, so there is the necessity of maintaining a continuous program of education for the nursing staff

The ideal in postoperative care of the tracheotomized patient is to maintain the airway as it would be if the normal expulsive and ventilatory forces were at work This can usually be accomplished when the cough reflex is intact, if there is no serious underlying pulmonary disease and if there is meticulous nursing care The problem becomes extremely difficult, however, when there is serious impairment of the cough reflex or of ventilation Some concern has been expressed about the effect of aspiration on the circulatory status of debilitated patients by Brattstrom[77] and on the effect of rapidly lowering carbon dioxide tension following the opening of the airway in hypercapnic patients by Negus[78] In our practice, we have never recognized any difficulty in the postoperative management of the tracheotomized patient from these causes

EVALUATION OF CLINICAL RESULTS

In an effort to evaluate the clinical application of tracheotomy in modern hospital practice, the tracheotomies performed in two teaching hospitals from 1954 to 1958 have been reviewed The findings are indicated by the table prepared from the survey It is to be noted that among the 526 cases, there was no serious or significant difference in emphasis between the practice of the two hospitals, so that the figures of one would fairly accurately reflect the practice of the other Of the 526 cases, 13 initially had cricothyroidotomies Among the reasons for doing the cricothyroidotomy, the most common was laryngoptosis in a very ill patient, where it was considered that the simplest possible procedure was the most appropriate for that particular moment In all instances in which the patient survived, the tracheotomy tube was replaced in the trachea at a subsequent procedure It is the practice in

TABLE I

Survey of Tracheotomies—1954 1958, Inclusive

A Total number of tracheotomies	526
1 Cricothyroidotomies	13
2 Tracheotomies	513
B Number done by bronchoesophagology	408
1 Associated bronchoscopies	66
2 Preliminary intubation	33
C Number done by other services	118
D Service primarily responsible	
1 Neurosurgery	124
2 Neurology	30
3 General Surgery	146
4 Bronchology	65
5 Medicine	104
6 Pediatrics	21
7 Plastic Surgery	20
8 Otolaryngology	6
9 Urology	6
10 Ophthalmology	1
11 Gynecology	1
12 Radiology	2
E Children Under 12	44
1 Children under three	34
2 Decannulated at discharge	25
3 Not decannulated at discharge	14
F Indication	
1 Laryngeal or above (mechanical obstruction)	
a tumor	72
b inflammatory	27
c foreign body	1
d trauma	28
e congenital	2
2 Tracheal	38
3 Tracheobronchial (secretory obstruction)	327
4 CNS depression	
a neurologic	155
b metabolic	34
c toxic	33
d trauma	1
5 Prophylactic	99
6 Cardiac	51
G Complication	
1 Bleeding	1
2 Pneumothorax	2
3 Stricture	1
H Results	
1 Number recovered	226
a decannulated	165
b not decannulated	61
2 Deaths	300
a primarily due to tracheotomy	2
b other causes	298
3 Number autopsied	129
a tracheitis	4
b pneumonia	99
c lung abscess	21
d thermal burns to lungs	2

these hospitals that the Broncho-esophagologic Service, which is intimately associated with the Otolaryngologic Service, carries the responsibility for the management of tracheotomy problems In 99 of the 408 cases done by the service, there was sufficient evidence of obstruction at the time of tracheotomy to require the use of an auxiliary airway during the operation One may recognize the role which tracheotomy performs in a wide range of situations by observing the variety of services to which the patients were primarily assigned The small figure for the Neurology Service can be attributed to the fact that for the most part cerebral vascular accidents are admitted to the medical service for care, with admission to the neurology service reserved for difficult diagnostic problems

The long duration of cannula life which is necessary in certain children is shown by the 14 who were discharged from the hospital wearing their tracheotomy cannulas Of 23 children tracheotomized for acute inflammatory lesions of the larynx, seven were done for supraglottic obstruction, and 16 for involvement in the glottic or subglottic area Obstructive lesions in the trachea or above represented the indication for tracheotomy in a total of 168 cases It is to be noted in reviewing the indications for which the tracheotomy was done that in certain cases, more than single indication was encountered

Some progress with the idea of doing tracheotomy early is indicated by the 99 prophylactic tracheotomies represented in this group Serious operative complications were few, but point out the need for maximum safety in the performance of tracheotomy Results indicate the number of patients recovered and discharged from the hospital Certain of these patients were discharged wearing their cannulas for various reasons, including the children noted above plus patients whose obstruction was not completely relieved, or patients with tumors discharged to continue X-ray therapy as outpatients The 300 deaths in this group indicate the application of tracheotomy to the seriously ill patient A death due to respiratory obstruction would represent loss of a patient by default

As indicated above, the problem of maintaining a clear airway in the patient with serious impairment of ventilation and cough constitutes a real challenge in the postoperative care of these patients Undoubtedly, many of these patients had their pulmonary complication at the time tracheotomy was done, but it is to be hoped that more effective control of inflammatory pulmonary complications can be achieved in these desperately ill patients At the same time, it must be stressed, in connection with the autopsy findings, that the pulmonary lesions were not the primary cause of death

COMMENT

The above findings are in accord with the findings of Fitz-Hugh[79 80 81 82] and his colleagues, who have noted the change in the application of tracheotomy to a broad group of problems There is some indication that this concept is not universally practiced, particularly in the non-teaching hospitals Whitaker and Lee,[83] who noted the performance of 25 tracheotomies in a hospital performing 10,709 operations in the same period, felt that the procedure should have been more widely used Baker and Wise[84] found that high obstruction overshadowed lower airway obstruction as an indication for tracheotomy by two to one It would seem, therefore, that a category or syndrome should be presented to the medical profession in which these newer indications for tracheotomy are clearly outlined without being attached to any particular disease entity, so that whenever this physiologic disturbance is encountered, the needs of the situation will be clearly recognized

The present concept of the indications for tracheotomy[26 85 86 87 88 89] is not in dispute What seems to be necessary to develop clearly in the mind of the profession are three concepts *1* When tracheotomy is needed, the sooner it is done the better *2* Obstructive lesions of the supraglottic area are dangerous, and the degree of obstruction is inconstant, whereas, obstructions arising in the cartilaginous box of the larynx tend to be regular and predictable *3* Failure of the expulsive mechanism of the lung from whatever cause threatens life and is an indication for tracheotomy The operation offers the following advantages *a* It removes obstructive secretion from the airway and gives easy access to it *b* It discourages aspiration of pharyngeal or gastric contents *c* It reduces the dead air space *d* It permits assistance of inadequate ventilation by positive pressure breathing apparatus

SUMMARY

A review of the literature and analysis of the utilization of tracheotomy in 526 cases is presented

The changing and growing scope of tracheotomy can be seen by comparing earlier reports of its use with those more recently published and with the clinical material presented

Obstructive lesions of the upper airway should be separated clinically into supraglottic and laryngeal categories

Obstructive lesions of the lower airway should be recognized as a single category found in many different disease situations, all characterized by a failure of the expulsive mechanism of the lung

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CORNEAL GRAFTS IN MIDDLE EAR SURGERY

A Preliminary Report[*]

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Viable tissue has previously been used in surgery for the restoration of hearing The tissue used for this has been skin, either as a free graft, any of the methods of sliding skin grafts,[1] vein grafts,[2,3] and mucous membrane grafts Closure of perforations has likewise been accomplished by the use of cigarette paper, egg membrane, silver nitrate, trichloracetic acid, with or without antibiotic agents[4] Foreign, non-viable material (gelfoam) has been used in stapedial footplate surgery

Four essentials are necessary for the maintenance of the viability of a graft in middle ear surgery first, the field must be free of infection, second, there must be immobility of the graft, third, there must be adequate blood supply, fourth, in tympanoplasties the graft must have free mobility in order to have an air containing middle ear cavity To acquire the fourth demand, an epithelial and "endothelial" lined graft is best suited, otherwise the graft becomes undesirably fixed to the inner wall of the middle ear

Corneal grafts are ideally suited for middle ear surgery Previously, corneal transplants, both homografts and heterografts, have been largely confined to improve vision or in therapeutic grafting of the eye

The human cornea averages 11 6 mm in the horizontal diameter, in the adult, with the vertical diameter slightly less It is slightly thinner by 0 8 to 0 9 mm in the center than at the periphery where it is 1 0 to 1 1 mm in thickness The cornea possesses no blood vessels except at the periphery where it receives its nutrition from the capillary loops The

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[*] From the Hearing Institute of the Colorado Springs Medical Center

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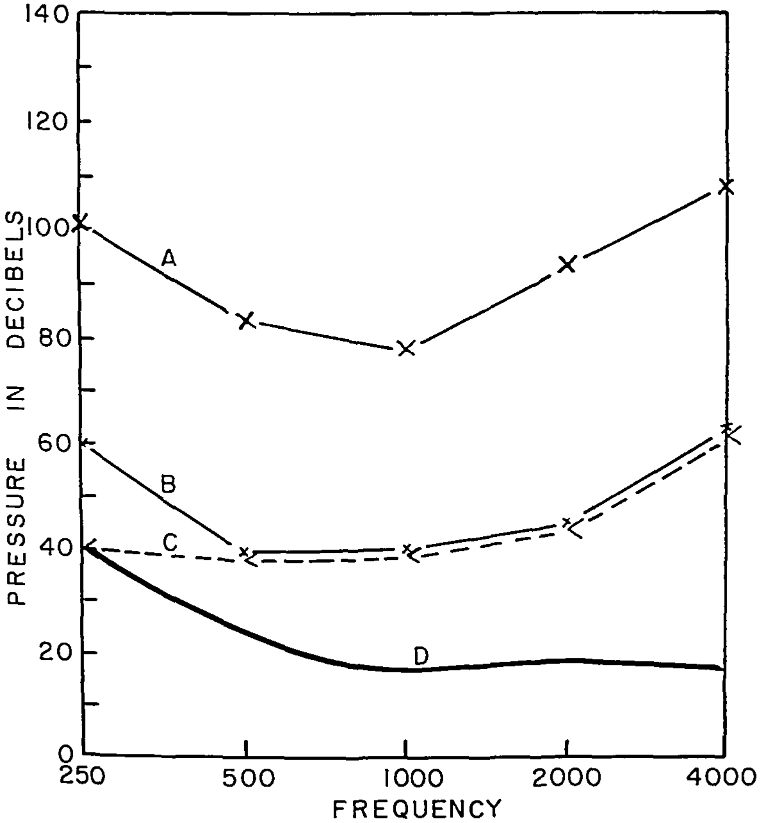

Fig 2

methods of obtaining, storing, and preserving corneas have been thoroughly studied and described[6-8]

The procedures used to preserve corneas for use in keratoplasty are very specific, because ultimate transparency of the cornea is important Since the utilization of the cornea in the ear is not concerned with opacification, corneas, not of value because of the ultimate opacity, can be used in ear surgery Fresh penetrating or lamellar, and freeze-dry penetrating or lamellar corneas have been used in the ear

The comparative greater thickness of the corneal graft aids in handling and positioning Apparently, the thickness does

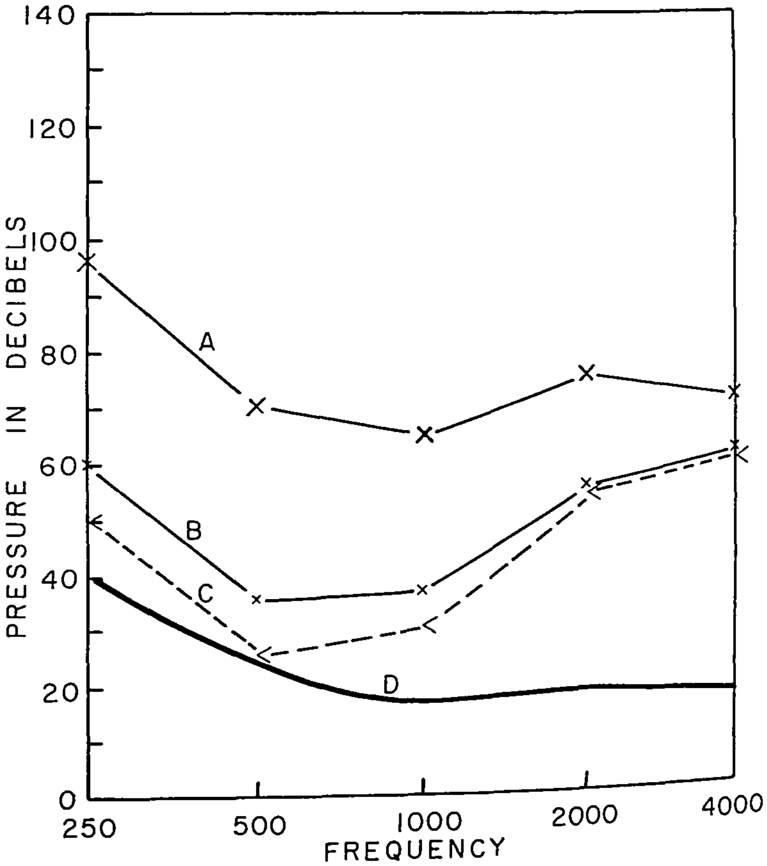

Fig 1

cornea lives almost by osmosis alone and is ectodermal in origin The endothelium is not a true endothelium in the embryological sense

Corneal grafts can be considered either lamellar or penetrating The lamellar graft is one which will basically include only the epithelium, Bowman's membrane, and stroma It is a split thickness corneal graft A penetrating corneal graft is a full thickness graft, including the entire cornea's being lined with epithelium and "endothelium"

Corneal transplants are not new, being described by Dieffenbach[5] as an "audacious fantasy" over 100 years ago The

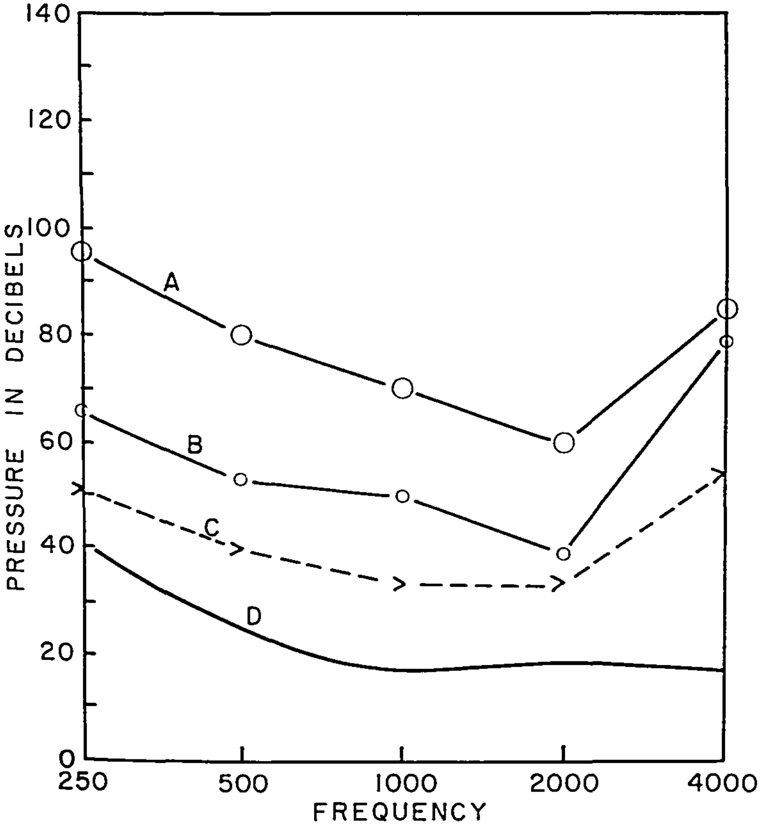

Fig 4

THE TECHNIQUES OF SURGERY

It is not the purpose of this paper to discuss the surgical technique of tympanoplasties or stapedectomies The technique of tympanoplasty and stapedial surgery was adequately discussed in the Workshop held in Chicago for Reconstructive Middle Ear Surgery in March, 1959

This is a preliminary report of the use of corneal grafts in tympanoplasties and stapedectomies Preparation of a donor site is done in the customary manner with special emphasis on hemostasis, otherwise, the advantage of transparency is lost When a remaining viable part of the tympanic mem-

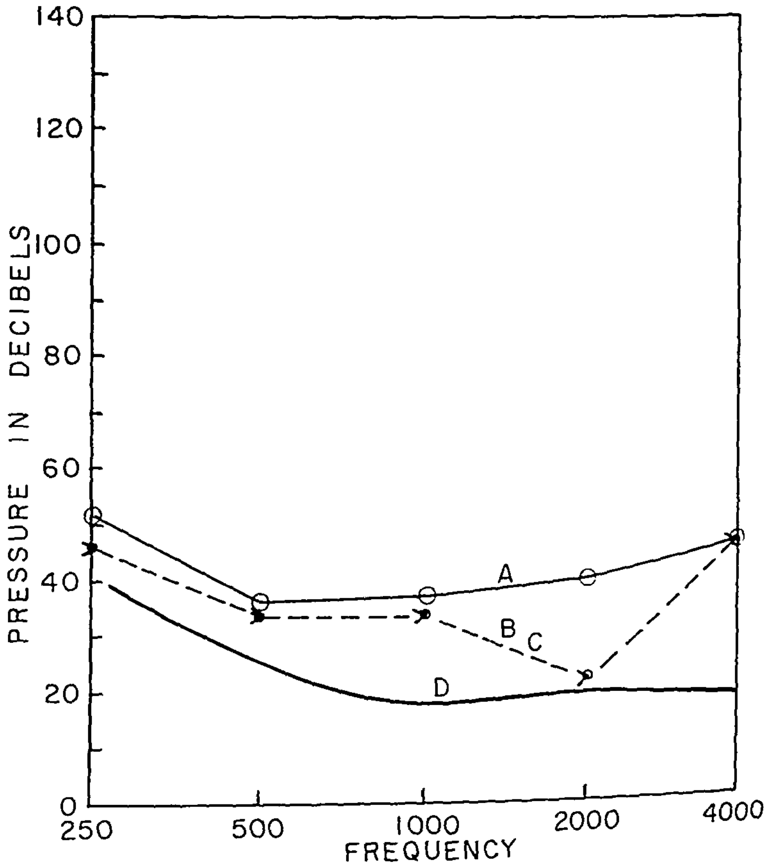

Fig 3

not interfere with the transmission of sound Because of ease of handling, suturing of the graft to the remaining drum has been done using a special suture ligature and single strand raw silk worm gut The immediate transparency in fresh grafts makes accurate positioning of the corneal graft easier

Naturally, there are disadvantages to corneal grafts first, human corneas are not readily available, second, the size of the cornea limits its use, third, antigen antibody reactions can occur, resulting in slough and sensitization The first two can be eliminated by using heterografts

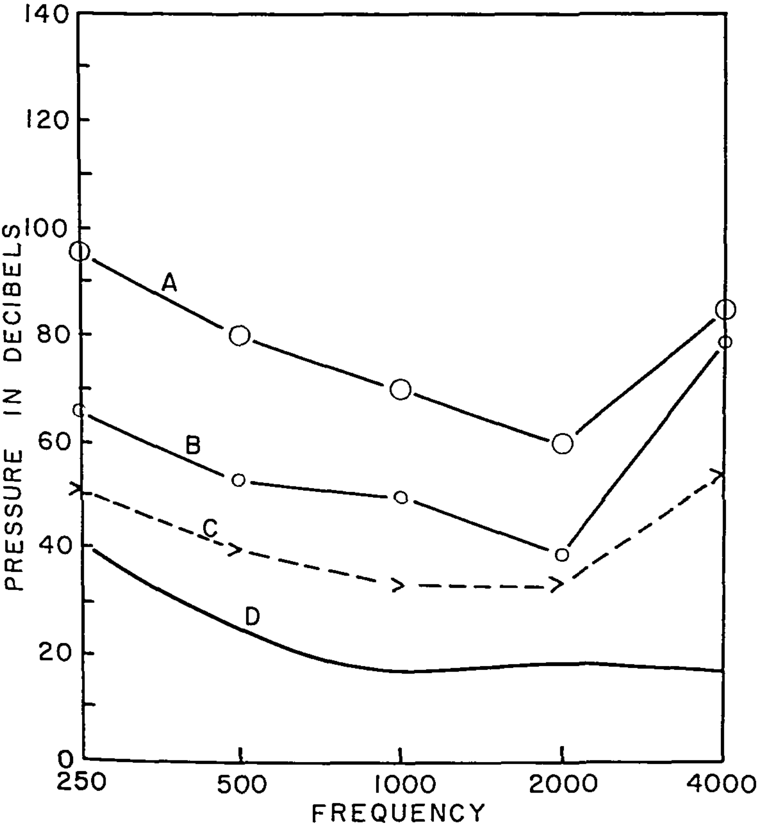

Fig 4

THE TECHNIQUES OF SURGERY

It is not the purpose of this paper to discuss the surgical technique of tympanoplasties or stapedectomies The technique of tympanoplasty and stapedial surgery was adequately discussed in the Workshop held in Chicago for Reconstructive Middle Ear Surgery in March, 1959

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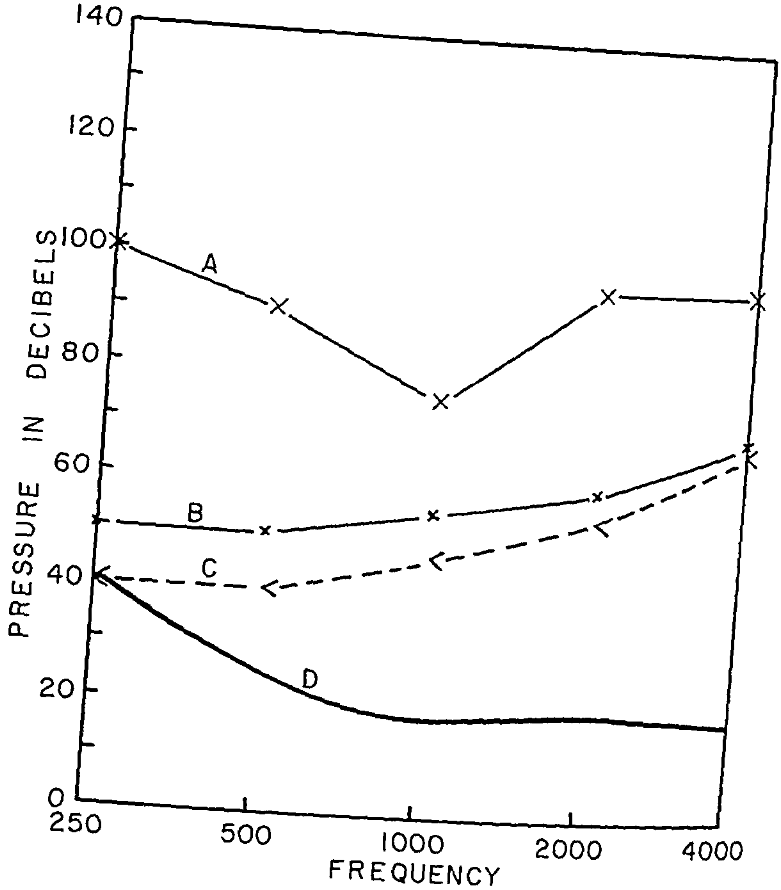

Fig 5

brane is left, the epithelium is removed from the drum, and a suitable sized cornea is sewed to the drum

When exenteration of the mastoid cavity is necessary, the mastoid cavity is lined with a three-fourths thickness skin graft from a non-hairy portion of the body The area tympanic membrane is replaced with a full thickness viable corneal graft, placed against the remaining movable ossicle or polyethylene tube

Corneal homografts have been used in the place of vein grafts and gelfoam in stapedectomies The cornea may be used either as a plug, similar to the gelfoam technique, or as a larger graft in the vein graft technique of Shea

RESULTS

Thus far in our limited number of cases, we have had uniformly good results It has been interesting to note that in audiograms the low tones return sooner in stapedectomies than by using the gelfoam or vein graft techniques The accompanying audiograms show the results in various cases

The hearing pressure charts as illustrated utilize absolute pressures 0002 dyne per square centimeter as the base, rather than the customary corrective audiogram representing 0 as a normal level The pressure grams are utilized as they show hearing levels rather than hearing losses

In the pressure grams, Line A represents the preoperative hearing level, Line B, the postoperative hearing level, Line C, bone conduction, Line D, the average normal hearing pressure level Figs 1 and 2 are stapedectomies Fig 3 is a myringoplasty with a central perforation, having very little hearing loss Fig 4 represents a marginal perforation with destruction of the long process of the malleus Fig 5 represents the results of a Type III tympanoplasty in which the entire drum, incus, and malleus were destroyed

CONCLUSION

This is a preliminary report of the use of corneal homografts in the restoration of hearing in middle ear surgery Corneal homografts appear to be a very satisfactory material for use in the middle ear because of the transparency and ease of handling The results so far have been good

A more exhaustive report of results and description of the individual techniques in the use of corneal grafts, both homografts and heterografts, will be presented in the future A more detailed discussion of technique is being prepared and will accompany a future report

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ANNUAL SPRING MEETING OF THE NEW YORK EYE AND EAR INFIRMARY ALUMNI

The Annual Spring Meeting of the Alumni Association of the New York Eye and Ear Infirmary will take place April 17-19, 1961

Symposia will be offered on Otosclerosis and Benign Lesions of the Larynx There will also be a Closed Circuit Television demonstration of surgical procedures In addition there will be lectures on Plastic Surgery, Paranasal Sinuses and Carcinoma of the Head, Neck and Larynx

For further information write Dr John R Finlay, Secretary, Alumni Association, 218 Second Avenue, New York 3, N Y

NEW METHOD OF TYMPANOPLASTY

Preliminary Report

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Montreal, Canada

Tremendous progress has been achieved in reconstructive surgery of the middle ear in the last few years, and unaided hearing has been restored to many patients suffering from cholesteatoma or chronic otitis media, however there are still some failures largely attributed to *1* the formation of scar tissue and adhesions to the stapes in the middle ear, and *2* the necrosis and perforation of the skin graft intended to seal the middle ear

Necrosis of the flap is due to poor blood supply to the free skin graft, and to postoperative infection[1] A pedicled flap done according to established plastic surgery principles has a blood supply sufficient to prevent necrosis of the extreme end of the flap[2] For this reason it should be no more than one and a half times longer than the width of the base, but in a highly vascularized area, when the blood vessels run parallel to the length of the flap, it can be made longer without danger of necrosis

With these principles in mind I worked out a technique to use a pedicled post-auricular flap and to transpose it in the mastoid cavity and over the middle ear space This flap may be used in Types II, III, and V

I have used this technique most often in Type III and occasionally in Types II and V, therefore, I will describe my procedure as it is used in Type III

STEP I EXPOSURE OF THE MIDDLE EAR AND ATTIC

An endaural incision is made, the middle ear, the attic, the

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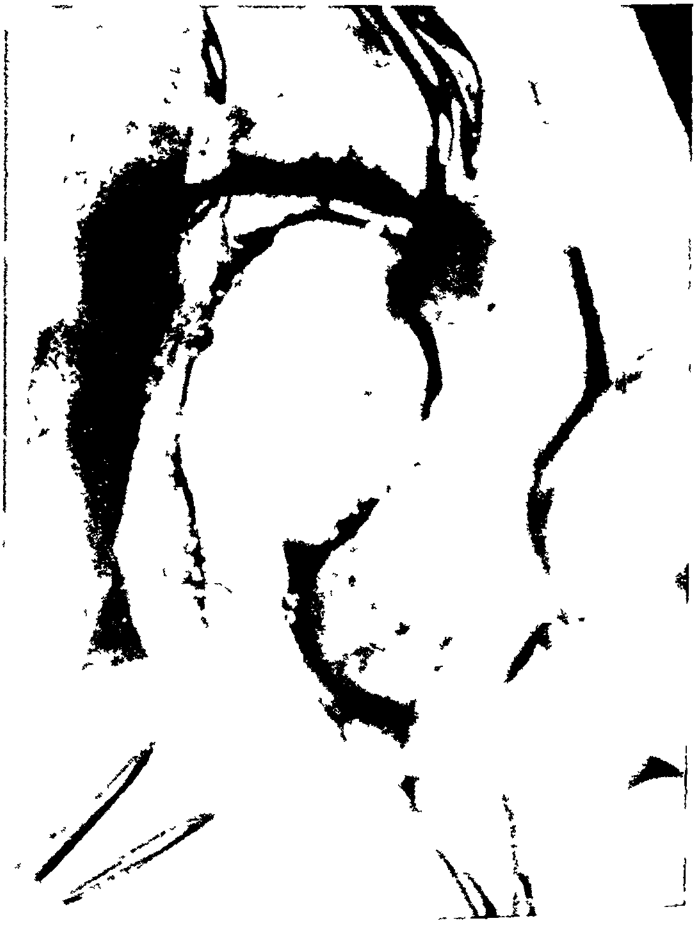

Fig 1 The incision is made from the hair line to the posterior surface of the auricle through the skin and subcutaneous tissues as illustrated

ossicles and the antrum are exposed either by transcortical or subcortical approach

STEP II REMOVAL OF THE PATHOLOGY

The pathology is removed from the middle ear, the antrum and the mastoid When we are dealing with a cholesteatoma, it is removed completely under magnification, including the matrix In cases of chronic infection, granulations, infected mucosa and infected bone are removed Since the ossicular

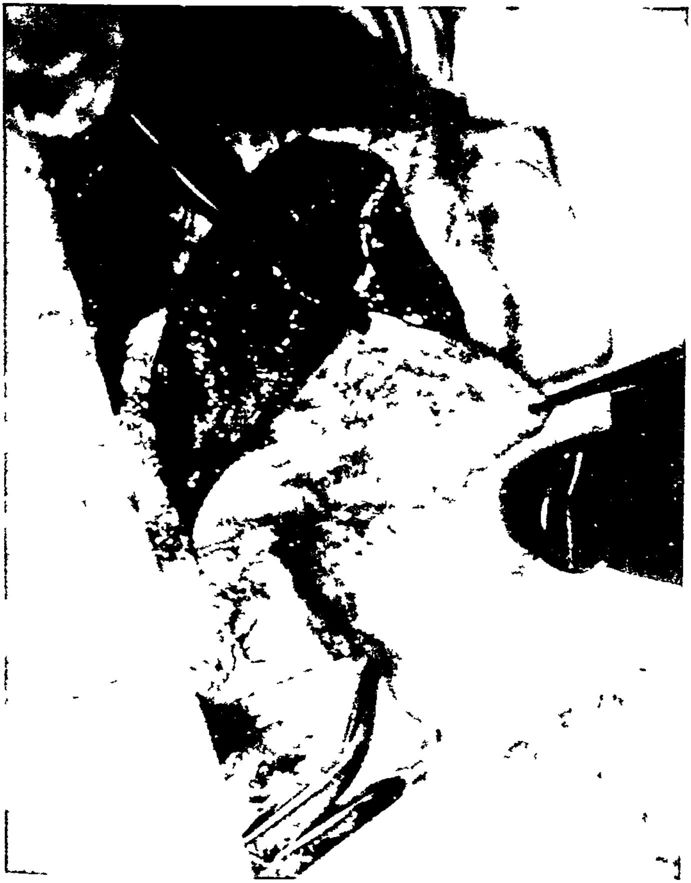

Fig 2 The flap is raised with blunt curved scissors from the postauricular donor area

chain is interrupted and the incus, the malleus and the tympanic membrane are badly damaged in these cases, they are removed, the remnants of the drum are also removed, but the stapes is preserved with as much mucous membrane as possible in the middle ear [4]

STEP III MOBILIZATION OF THE STAPES

The stapes is mobilized by careful removal of adhesions, hypertrophied mucous membrane and cholesteatoma in this

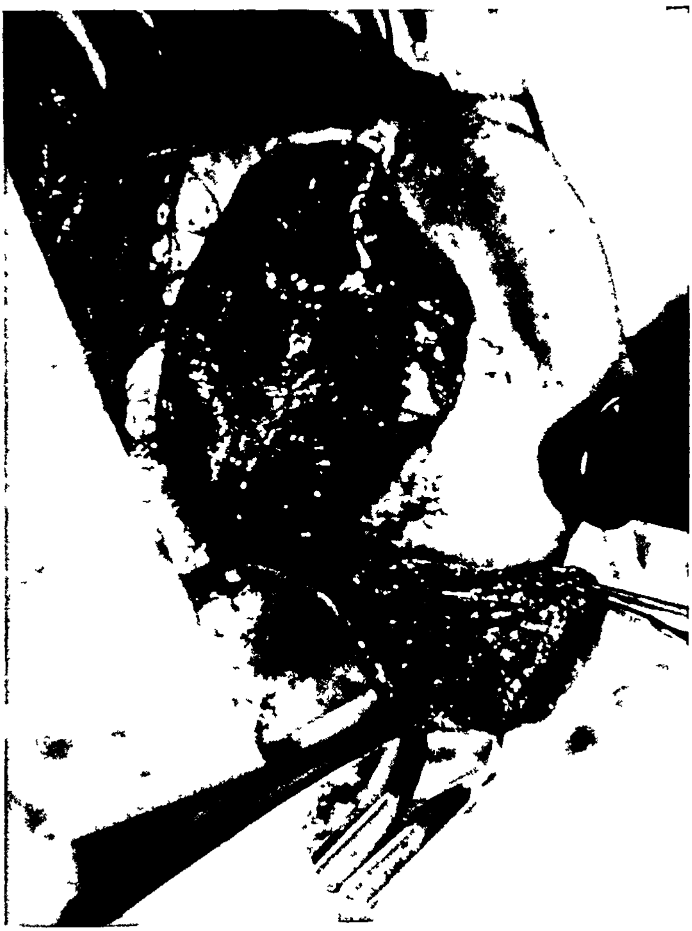

Fig 3 The flap has been raised and its pedicle is shown near the mastoid tip

area The patency of the Eustachian tube is checked by means of a dull probe applied gently

STEP IV RAISING OF THE PEDICLED FLAP

A pedicled skin flap is raised from behind the ear, the pedicle being at the mastoid tip and the free end near the upper attachment of the auricle as seen in Figs 2 and 3 All the skin from the hair line to half way behind the pinna is raised It is important not to include hair follicles that would

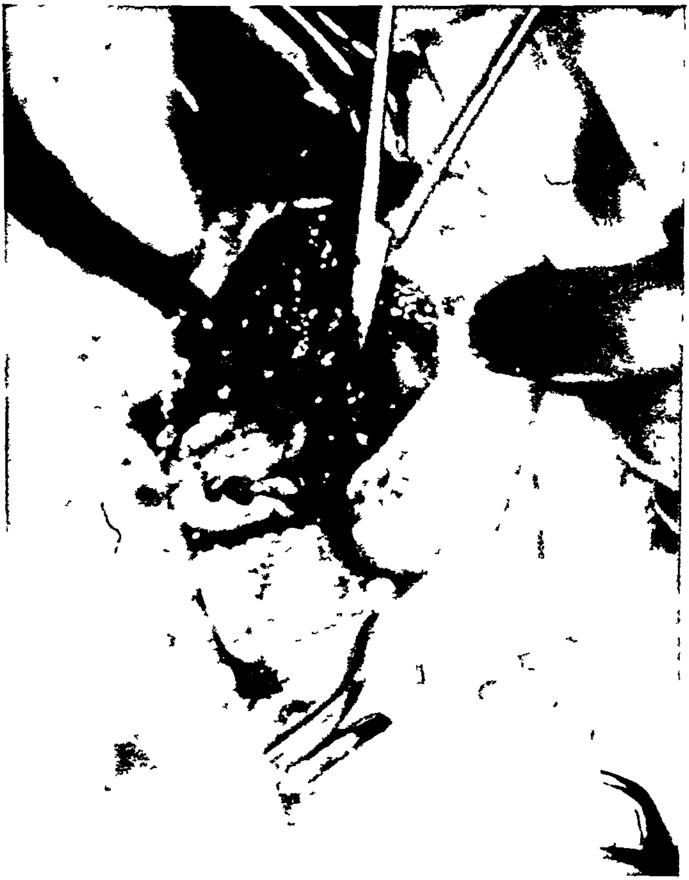

Fig 4 A small incision is made through the periosteum near the mastoid tip to permit transposition of the flap to the middle ear through this incision

otherwise grow in the mastoid cavity The flap is made thin in the distal half and thicker in the proximal half

STEP V TRANSPOSITION OF THE FLAP

The flap is transposed in the mastoid cavity The soft tissues and periosteum are incised down to the mastoid process for a length of 2 cms , near the mastoid tip, this allows enough room for the transposition of the pedicled flap in the mastoid cavity and over the middle ear through the lower end of the

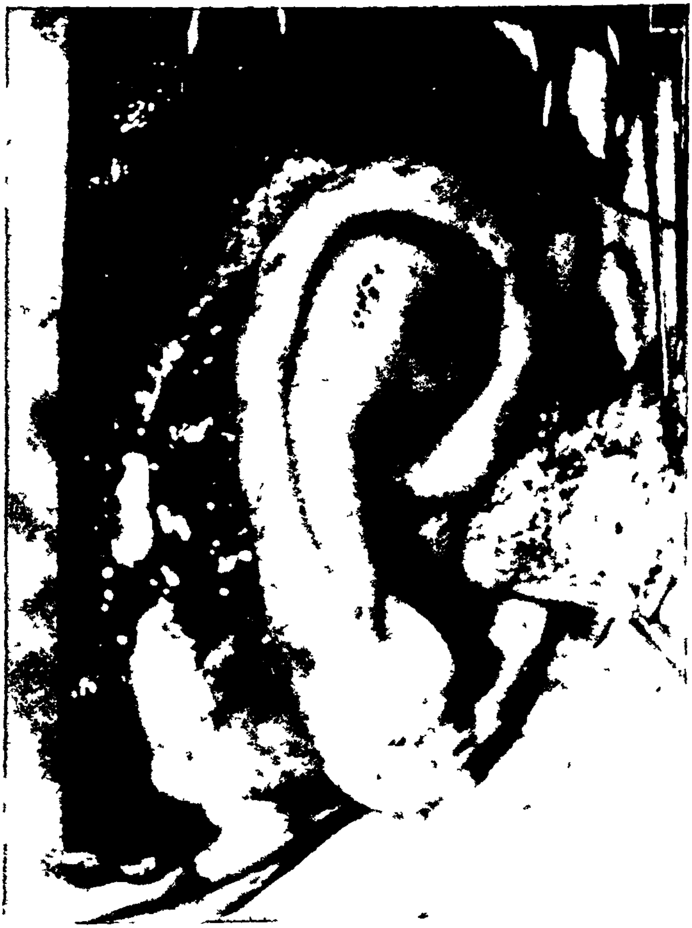

Fig 5 The flap has been transposed to the mastoid cavity and is shown through the endaural incision

post-auricular wound The flap is placed in such a way as to seal the middle ear space and to be in close contact with the head of the stapes [5] Packing is done with parresin mesh, and the endaural incision is closed

STEP VI CLOSURE OF THE DONOR AREA

The edges of the post-auricular wound are undermined and are approximated with No 000 silk mattress sutures, except for the lower third of the wound

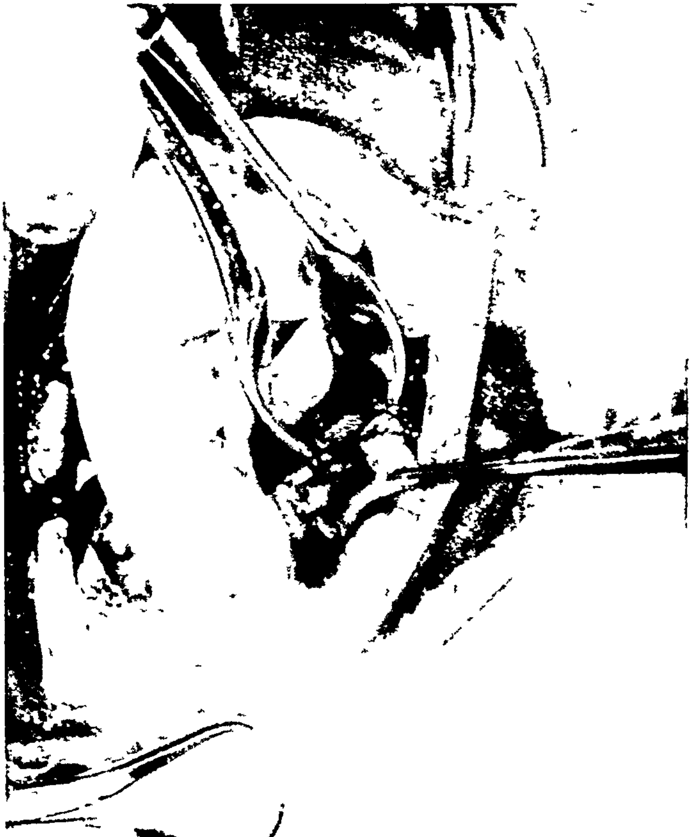

Fig 6 The flap is being laid over the antrum and the middle ear

Two or three months later the skin of the pedicle is incised, raised on the distal side, and the lower third of the wound is closed with No 000 silk sutures

In Type V, several months after the first operation has been done, and when the mastoid cavity is completely epithelized and there is no infection whatsoever, a tympanomeatal flap is incised and raised to expose the lateral semicircular canal as in fenestration surgery A fenestration is made over the ampulla, the skin of the flap is thinned down and invaginated over the fenestra

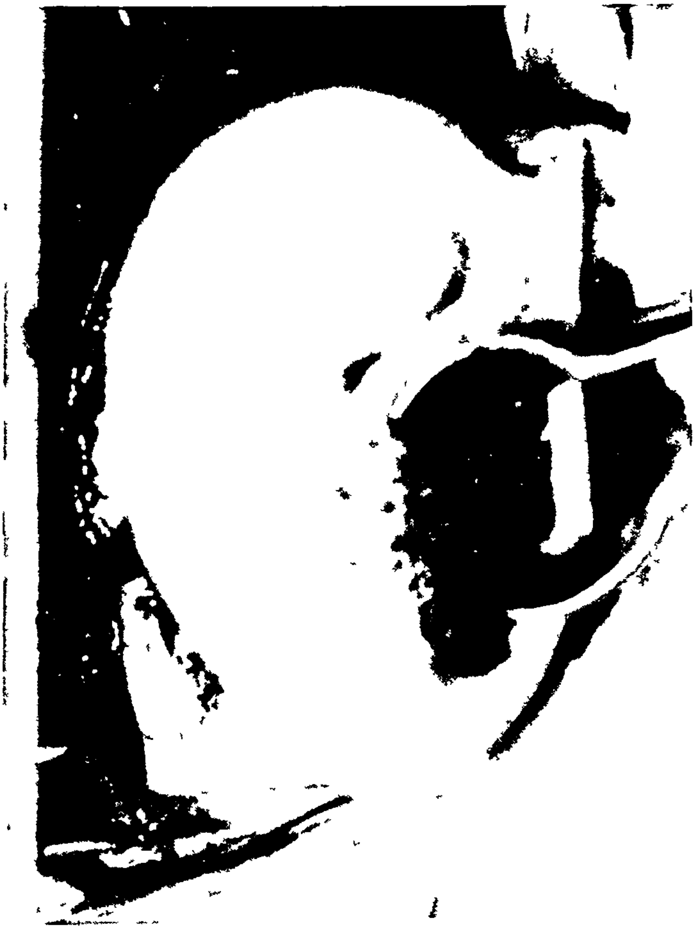

Fig 7 The flap is shown sealing the middle ear and attic

COMMENT

The flap, if properly made, is long enough to reach and seal the middle ear space without tension on the pedicle[1], in some instances there was even too much skin and some had to be cut off to allow the flap to fit properly The transposition of the flap can be done without undue twisting of the pedicle which could interfere with the blood supply -

The blood is supplied in this region by branches of the posterior auricular artery which run parallel to the length

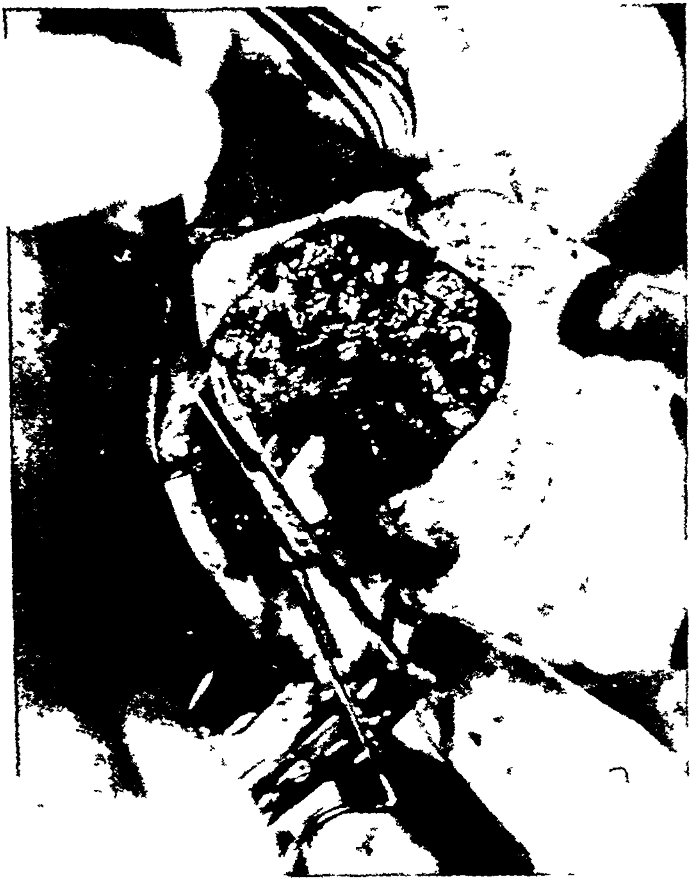

Fig 8 Undermining is done with blunt scissors on each side of the wound for closure

of the flap, this permits a length more than one-and-a-half the width of the base, without cyanosis and necrosis

Inflation of the Eustachian tube is started ten days after the operation to prevent adhesions and to maintain an air space in the middle ear.

After closure of the post-auricular wound the auricle is somewhat closer to the skull than previously, but after a month or two the auricle has regained its original position There is no deformity if the skin is raised no further than

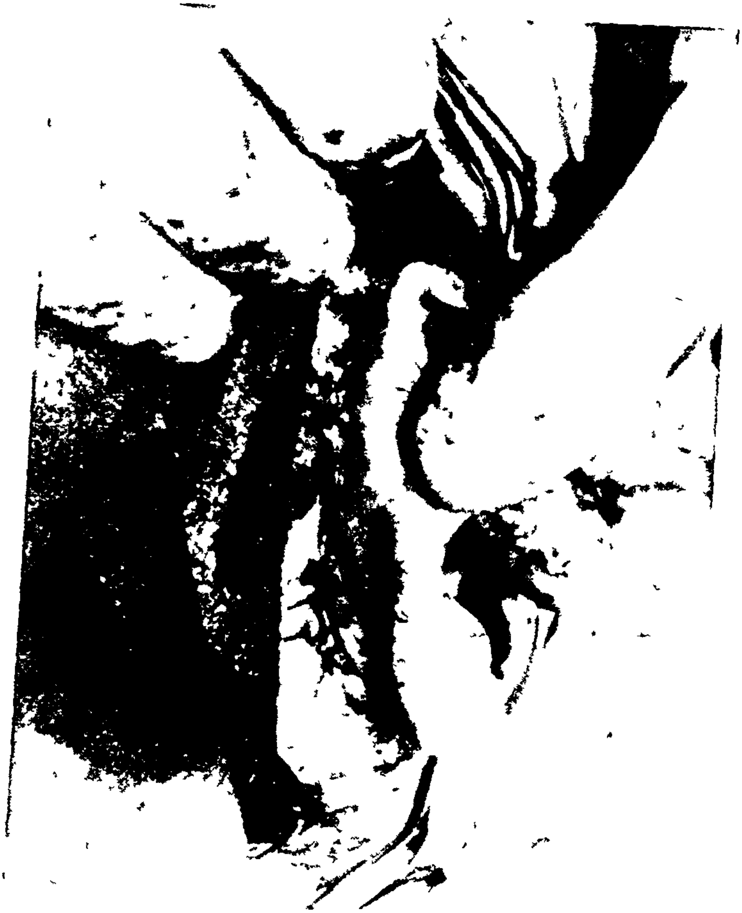

Fig 9 The donor area is closed with No 000 silk mattress sutures

half way from the post-auricular fold to the helix on the posterior surface of the auricle

All this work is done extra-cartilaginous, the cartilage is not exposed or cut through at any time

With this method, epithelization of the mastoid cavity is completed much earlier, and the danger of postoperative suppuration is much reduced on account of the rich blood supply that is brought in the mastoid cavity [1]

I suggest the name "Middle Ear Reconstruction" to describe

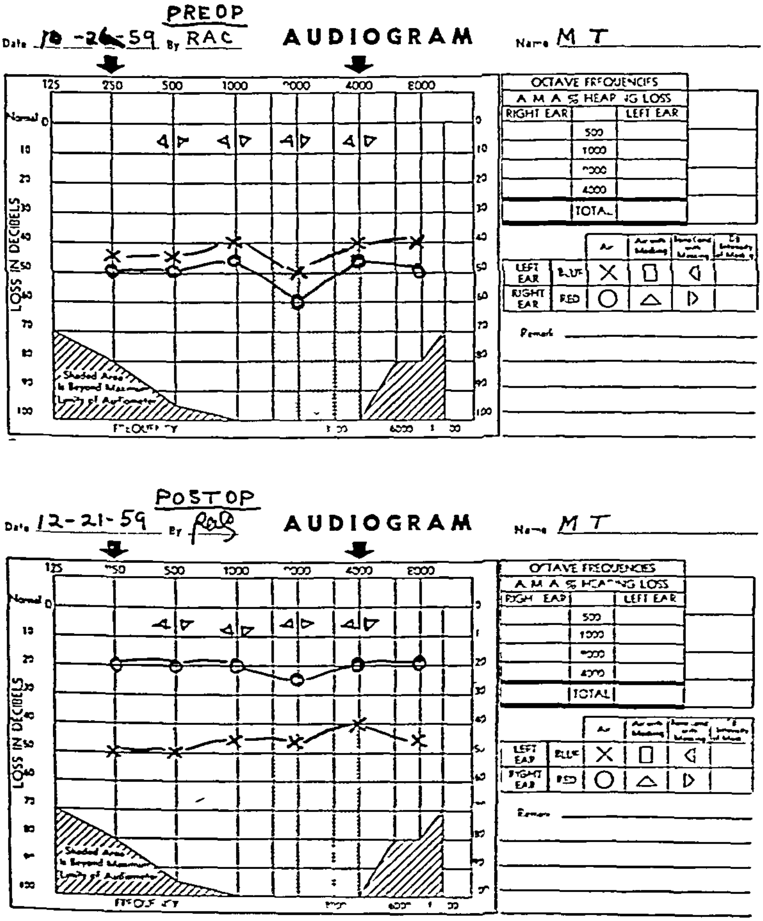

Fig 10 Preoperative and postoperative audiograms in Type II tympanoplasty with post-auricular pedicle graft

this procedure, whenever the pedicled post-auricular flap is used

SUMMARY

1 A pedicled post-auricular flap is recommended in tympanoplasty Type III and Type V, instead of a free graft

2 This flap is intended to eliminate the postoperative necrosis and perforation of the graft that sometimes occur.

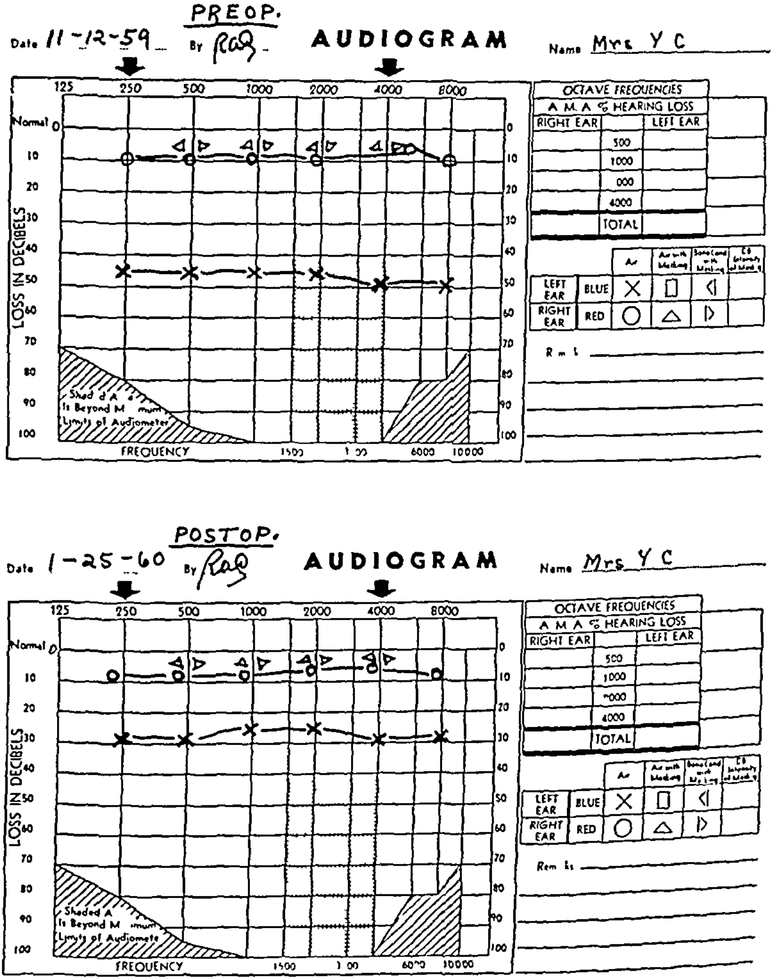

Fig 11 Preoperative and postoperative audiograms in Type III tympanoplasty with post-auricular pedicle graft

3 This flap carries with it a rich blood supply in the mastoid cavity, preventing, therefore, postoperative suppuration and promoting faster epithelization and healing

4 There is no deformity of the auricle if the technique described is followed

5 All the incisions are extra-cartilaginous to lower the risk of perichondritis and chondritis

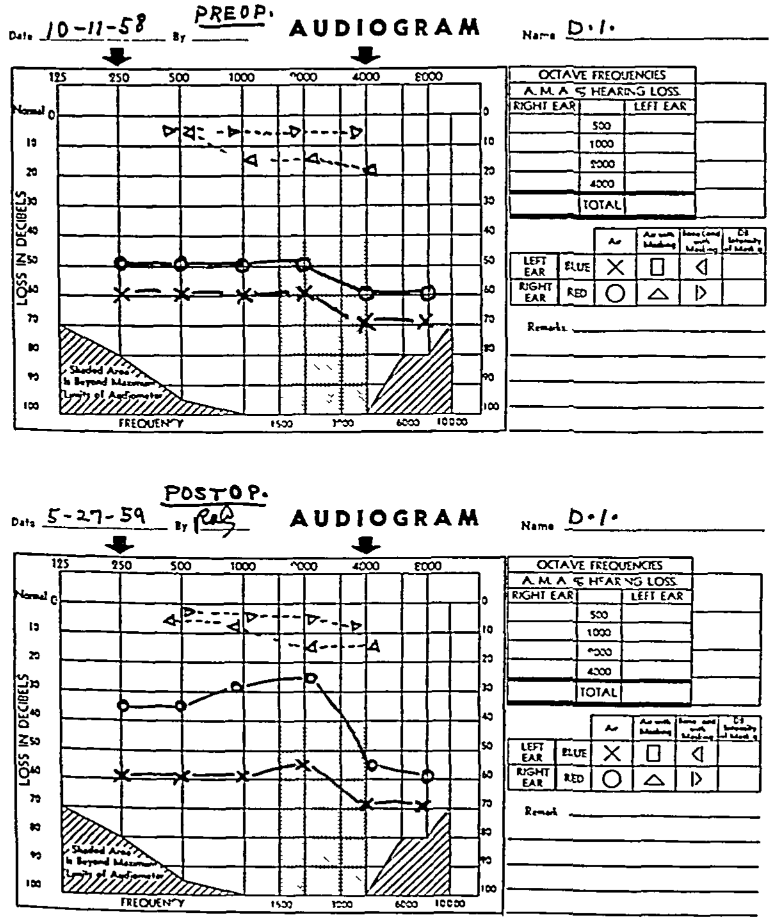

Fig 12 Preoperative and postoperative audiograms in Type V tympanoplasty with post-auricular pedicle graft

6 The name "Middle Ear Reconstruction" is suggested to describe tympanoplasty, whenever the pedicled post-auricular flap is used

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